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A TREATISE

ON THE

PRINCIPLES AND PRACTICE OF MEDICINE

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BY

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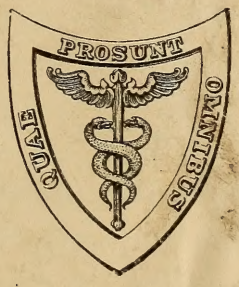
THIRD EDITION, THOROUGHLY REVISED, AND REWRITTEN

ILLUSTRATED WITH 80 ENGRAVINGS AND 23 PLATES



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PREFACE TO THE THIRD EDITION.

The demand for a new edition of a work in so highly developed a field of medical literature is both a commendation and a summons to improvement. The author has spared no effort in complying. The real advances throughout this enormous and active domain have been incorporated, and the whole work has moreover been virtually rewritten to secure increased brevity and clearness. The result is indicated by the fact that a vastly greater amount of information is furnished in a space decreased by some two hundred pages.

A few of the additions and changes may be mentioned. Particular attention has been given to therapeutic details in accordance with the recent awakening of the profession to the importance of logical treatment; numerous new preparations, and modified names and dosages, particularly for children, are explicitly specified. There are practically new chapters on ictero-anemia, the ductless glands, *x*-ray findings, erythremia, sepsis (infection, toxemia, bacteriemias), high calory-feeding in typhoid with a table of food values, sporotrichosis, blastomycosis, trichinosis, hook-worm disease, pellagra, gas poisoning, the arrhythmias and other cardiac neuroses, tropical splenomegaly and various other tropical affections. Due consideration has been given to the meningitis serum of Flexner and Jobling, Strong's work on amebic dysentery, Brill's disease, anaphylaxis, paratyphoid, blood cultures in typhoid and other bacteriemias, the "carriers of infection," the recent epidemics of meningitis and poliomyelitis, vaccines, serotherapy, the spirochete as the cause of syphilis and the recent status of tuberculin in its diagnostic and therapeutic application. The diagnostics and therapeusis of cardiac failure, hypertension, diabetes, gastric and duodenal ulcer, constipation, drug addictions, neuralgias, etc., have been elaborated fully. New plates are introduced, illustrating the diphtheria bacillus and its cultural appearance, the Spirochetæ pallida and refringens, and *x*-ray plates in gastric subjects.

The chapters on tuberculosis and syphilis are designedly amplified, as these diseases touch every organ, enter every specialty, and attain as great sociological as medical importance; indeed, an understanding

of these maladies is almost an understanding of medicine. The Wassermann and luetin tests, congenital lues, and new matter on mercury and salvarsan are treated with much detail, because of their colossal importance. Attention has been given to numerous criticisms and many minor alterations have been made in the interest of logic, clarity and conciseness.

So wide is the range of Modern Practice that an author who would cover it in a single volume, and a reader who would grasp it intelligently, must approach their respective tasks with the aid of *thorough system*. An effort has been made to deal with the subject in this manner, from its main divisions, which follow the most rational classification, down to the subordinate paragraphs. Careful use has been made of types of various prominence to facilitate the finding of a topic and the appreciation of its importance. With the vast mass of material, an attempt has been made to so arrange the facts that the reader may grasp the process of reasoning. Causative pathology has been blended with the consecutive clinical features of disease, reasons have been given for facts, exceptions have been subordinated to what is usually found at the bedside, and the allurements of typical clinical pictures and dogmatic generalizations have been avoided because they hold neither in practice nor at the postmortem table.

The author also conceives that a book on Practice should be *well directed*, which implies that it should deal adequately with scientific theories and principles, but that it should recognize that the final object of its existence is the *application* of knowledge *to the cure or alleviation of disease*. Accordingly, an unusual amount of space has been devoted to treatment, to the detailed consideration of drugs and to numerous formulæ and prescriptions ready for the student to use or improve upon. The physiological action of drugs has been dwelt upon carefully because, in the writer's experience, the symptoms of disease may be confounded with those of the remedies exhibited for its cure. The index embraces the chief references to the remedies and the formulæ for their exhibition, and the table of contents sketches the general scheme of arrangement.

The reader will also find a large number of tables giving the differential diagnosis of diseases likely to be confused, and in many instances, of entire subjects, such as those of the liver, brain and kidney, the chief eruptive diseases and those of the typhoid group. They are practical, though necessarily schematic. Nowadays the border-lines of surgery and medicine overlap, and the practitioner, internist and surgeon must be familiar with the province of each other. For this reason,

as far as the scope of the treatise admits, surgical indications and results are introduced.

Names and dates are employed to give credit, where it is possible, to the great workers in the history of Medicine.

The writer wishes to acknowledge the extremely careful attention bestowed by the Publishers upon every literary and typographical detail, and the assistance of Dr. Milton Mandel in the proof reading.

A. R. E.

Chicago, 1916.

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SECTION I.

THE SPECIFIC INFECTIONS.

BACTERIAL DISEASES.

SEPTIC INFECTIONS.

Synonyms.—Sapremia, septicemia (sepsis), pyemia, septicopyemia.

Definition.—To understand sepsis is to understand all infections. Sepsis is no longer a purely surgical or obstetrical term; today it is more important in medicine than surgery. *Intoxications* are the result of the absorption of substances elaborated by microörganisms. *Infections* are always due to microörganisms, which enter the body and multiply there. Tetanus is an infection, yet it is non-contagious. *Contagion* refers to infection by direct contact, as in scarlatina, or by indirect means, as water infected by typhoid dejecta. *Sapremia* is the absorption of putrescent substances, but not of the germs which develop them. *Toxemia*, a broader term, includes intoxication and sapremia; *toxins in the blood* are largely the result of bacterial metabolism. Probably all pathogenic bacteria produce toxins; the latter are divisible into *intracellular* toxins, found in the bodies of microörganisms—the case in most toxemias—and the *extracellular* toxins, thrown free into the circulation. *Septicemia* (sepsis) is the presence in the blood of microorganisms (bacteriemia)—usually pyogenic—without hematogenous suppuration or metastasis. *Pyemia* is infection, caused by microörganisms, which leads to hematogenous suppuration or metastasis. Septicopyemia is septicemia (bacteriemia) plus pyemia (metastatic suppuration).

General Groups.—1. Some infections—such as carbuncles, puerperal parametritis, diphtheria and tetanus—are *local* so far as the causal microbe is concerned, and their symptoms are due to absorbed toxins (toxinemia, toxemia). Local reactive inflammation seeks to protect the body against generalization of the microbes, by means of (i) mechanical measures, as pouring out of the leukocytes, deposition of fibrin or development of connective tissue; (ii) phagocytosis; and (iii) bactericidal and antitoxic action of the lymph and blood serum.

2. Other infections—such as carbuncles, puerperal sepsis, anthrax, and gonorrhea—*at first local*, may *become general* when their germs enter the blood (*bacteriemia* or *septicemia*). Some infections, especially typhoid and pneumonia, once considered local, are always general.

3. Still other infections, often resulting from the same microörganisms, cause *metastatic suppuration* (*pyemia*). Among these are multiple

abscesses from carbuncles, puerperal fever, typhoid osteomyelitis, pneumococcic endocarditis, and gonorrheal arthritis. It is frequently clinically difficult to preserve these types which so often blend; what is seemingly toxemia may prove bacteriemia if blood cultures are made; apparently pure bacteriemia may prove pyemia at necropsy; all three grades may occur successively; the primary infection—*e. g.*, with scarlatina, tuberculosis, and diphtheria—whether local or general, may be complicated by a later secondary or a simultaneous mixed infection, especially by the streptococcus; again the symptoms vary, first with the variety of microbe and its virulence; second, with its localization (in the throat, lung, pelvis, etc.); and third, with the physiological resistance or susceptibility of the patient. Subjects of malignancy, cardiac, vascular, nephritic, cirrhotic and other diseases, readily succumb to an ultimate sepsis to which the name *terminal infection* is applied. For these reasons we will discuss septic infections as a whole, and indicate such special etiological and clinical variations as seem necessary.

Etiology and History of Septicopyemia.—Of the numerous causal organisms, the most common are the pyogenic cocci, the pneumococcus and colon bacillus; less frequent are the gonococcus, anthrax bacillus, typhoid or diphtheria bacillus, Friedländer's pneumobacillus, meningococcus, Bacillus pyocyaneus, B. influenzæ, B. phlegmones emphysematosæ, B. aërogenes encapsulatus, and least often, the Micrococcus tetragenus, the blastomycetes, the spirilli and bacilli of Vincent, the Bacillus fusiformis, and the proteus group. Wunderlich (1847) spoke of spontaneous pyemia, and Leube (1878) of cryptogenetic (occult) septicopyemia, yet careful clinical and necropsy investigations reveal the *atrium of infection* in 94 per cent. of cases; (1) *skin* lesions, such as furuncles, felons, navel infection in infants, and invisible, as well as visible wounds (staphylococcus); (2) *throat* (streptococcus), as in tonsillitis, scarlatina or diphtheria; (3) *nose* (not frequent in sepsis, though in meningitis the atrium for the meningococcus and pneumococcus); (4) *ear* (pneumococcus and streptococcus); (5) *lungs* (pneumococcus); (6) *intestines* and *bile tracts*, gall-stone infection, appendicitis, dysenteric ulcers (colon bacillus, typhoid bacillus); (7) *urinary* tract (pyelitis from colon bacillus; vesical and urethral infections, chiefly by the staphylococcus, far less by the streptococcus and gonococcus); and (8) *vagina* and *uterus* (chiefly streptococcus), causing puerperal infection.

Puerperal fever, primary wound fever and severe late pyemic infection were known to Hippocrates.

Boerhaave (1720) declared that secondary abscesses were due to pus in wounds. John Hunter (1774) associated purulent phlebitis and pyemia. D'Arcet (1842) drew a sharp distinction between toxemia and pyemia. In 1846 Virchow discovered that the "pus in the veins" was softened thrombi and that the "pus in the blood" was merely leukocytosis; he used the terms septicemia and pyemia in their modern construction. Following his work, the subject was clarified by bacteriology.

General Pathology and Symptomatology.—(*A*) General Toxemic Stage.—1. *Fever.*—The *virulence* of the germ and its toxin (rather

than its variety) *governs the intensity of the toxemic symptoms.* Fever is associated with cellular reactions which form specific antibodies (antitoxins, lysins, opsonins, and agglutinins). It is difficult to state how far fever is benign and how far injurious. By toxemic disturbance of the cerebral centres regulating temperature equilibrium, more heat is produced and less eliminated. Fever is usually the first clinical symptom and in general the fever tends to be remittent or intermittent; (*a*) streptococcus infections usually produce irregular intermittent fever with moderate variations; (*b*) the staphylococcus and pneumococcus produce remittent or continuous fever; (*c*) the colon bacillus and gonococcus cause intermittent fever with great variations. Fever may be absent in very severe forms, especially before death, when the cells of the body are overwhelmed by an excessive production of toxins.

2. *Chills* occur in septicemic and especially in pyemic forms and are usually irregular. They mark the discharge of bacteria or toxins into the circulation.

3. *Nervous toxemia* may appear as unrest, delirium, stupor, dry tongue, and the status typhosus, or it may resemble meningitis. The sensorium may be clear.

4. *Vasomotor and cardiac weakness* may be present with increased, irregular or hopping pulse; the cardiovascular system may suffer the same damage as in typhoid or pneumonia toxemia.

5. *Increased respiration,* from augmented gaseous exchange, and bronchitis may be noted.

6. Albuminuria and acute nephritis are common from microbic or toxemic injury.

7. *Septic diarrhea,* in which the dark, perhaps bloody, movements number six to ten daily, may be observed. Tympanites is frequent from the toxins lowering the tone of the intestinal muscle.

8. Acute *splenic tumor* is present.

9. *Anatomically* we find cloudy swelling and fatty degeneration of the heart, liver and kidneys; a vascular, soft, moderately swollen spleen, with indistinct markings and wrinkled capsule; acute nephritis; hemorrhages into the skin, retina, and mucous and serous membranes, with or without bacteria; lung edema, and, where there is marked anemia, there is fatty heart, increased iron deposit in the liver, hemorrhages by injury to the vessel walls or necrosis in the parenchymatous organs or lymph glands.

(*B*) Blood Findings.—1. *Bacteriemia* is found in 50 per cent. of cases clinically, and in 95 per cent. at autopsy; 10 to 40 c.c. of blood are removed antiseptically from the median cephalic vein by an aspirating needle and mixed freely with bouillon. The *importance of blood cultures,* made daily, in severe or doubtful cases, cannot be overestimated. Some circulating bacteria are killed by the blood. In most streptococcic and staphylococcic infections, these organisms are found in the blood.

2. *Polymorphonuclear leukocytosis* and iodophilia are very common, though not invariable, particularly in sepsis due to pyogenic organisms. The lymphocytes are generally decreased, *e. g.*, in contrast to the typhoid formula (page 32). However, Cabot and others describe, in wound

sepsis, boils and widespread streptococcic adenitis, an extreme lymphocytosis, resembling acute leukemia.

3. *Anemia* is usual, and may resemble the pernicious form.

(*C*) METASTASES.—They are apparent clinically, or first found at necropsy. *Thrombophlebitis* develops at the seat of primary infection, and disintegrated thrombi, containing bacteria, escape into the venous circulation. They lodge in the lungs or, passing them, reach the left heart, and arterial system. Purulent lymphangitis may, in the same way, cause metastatic suppuration. If *"end arteries" are plugged*, infarcts result; if the arteries are not terminal, small suppurative foci develop, especially where the circulation is slow, as in the liver, kidney and spleen. Not all infarcts suppurate, either because the germs are less virulent, or the embolic material is free of microbes. (1) *Lung* localization may cause multiple or single abscesses, and often serofibrinous or *purulent pleurisy.* (2) *Cardiac* metastasis develops in 21 (even 63) per cent. of the metastatic group; *endocarditis* is localized chiefly on the mitral (62 per cent.) and aortic (22 per cent.) valves; it is attended by a heart murmur in only 60 per cent., and often by *pericarditis;* the *heart muscle* is sometimes the seat of abscesses. (3) *Cerebral metastasis* may cause *meningitis, brain abscess* or softening (encephalomalacia with aphasia and hemiplegia). (4) *Renal* localization may incite nephritis or suppurating infarcts, with albuminuria, cylindruria, pyuria and bacteriuria. (5) *Splenic* localization usually results in enlargement, frequently in pain, and rarely in perisplenic friction. (6) Localization in the *bones* may cause early fugitive bone pain, or later, fixed osteomyelitic tenderness over one or more bones, with inflammatory signs; in the *joints* (as in the postfebrile arthritides) it may cause simple pain, or serous or purulent synovitis; in the muscles it may produce abscesses, purulent edema, or Wagner's polymyositis. (7) In the *skin ecchymoses* develop in 50 per cent. of fatal cases, usually late, often symmetrical, and sometimes on the nose, fingers, toes or ears. Ecchymoses may be peculiarly significant of sepsis in the absence of other symptoms. *Pustules* and vesicles are less frequent; herpes occasionally develops, particularly in genito-urinary colon infections. Scarlatiniform, morbilliform, roseolous or urticarial eruptions are only toxemic. (8) In 33 per cent. retinal ecchymoses occur, first described by Litten as round, irregular spots or in streaks, with small white centres and appearing late or just before death. Least frequent is panophthalmitis, of which 33 per cent. is due to ulcerative endocarditis. (9) The *peritoneum* is chiefly involved in puerperal forms. (10) The slightly enlarged *liver* is more often due to toxic parenchymatous degeneration and cardiac weakness than to abscess, which occurs in only 15 per cent. of cases of septicopyemia. Intestinal infarction is rare. In one type icterus is associated with meningeal symptoms.

Some metastases develop, maybe years later than the original infection, as typhoid osteomyelitis. This *bacterial latency* is also noted in diphtheria in which virulent bacilli persist possibly for months in the throat; in tuberculosis where apparently healthy lymph glands may contain tubercle bacilli and perhaps also in diseases which recur, as erysipelas

and articular rheumatism. *Recurrence* may also be due to fastness or mutation of the infecting organism, resulting from enduring subinfection.

Special Symptomatology.—This may be considered etiologically or topographically. Only the leading characteristics are presented.

(*A*) Etiological.—1. *Streptococcic infection*, the *most frequent primary* and *secondary septic infection*, constitutes 66 per cent. of all forms, the coccus entering the blood in 33 per cent. of all fatal diseases. Its atrium is most often *the throat* and the female genitalia; it enters the veins in puerperal pyemia more often than the lymphatics (erysipelas and puerperal sepsis); it more often produces septicemia (65 per cent.) than pyemia (35 per cent.); the lungs are usually unaffected; endocarditis occurs in 7 per cent.; and its course is chiefly acute, sometimes subacute (ulcerative endocarditis).[1]

2. *Staphylococcic infection* usually *enters* by *the skin;* the furuncle is the typical primary lesion. On the eyelids, nose or lips, this dangerous infection may reach the facial vein, then the ophthalmic vein and cavernous sinus. Furuncle of the neck may reach the transverse sinus. Infections of the skin are the most frequent cause of *osteomyelitis*. Meningitis is known to have followed a felon. The coccus passes the throat with difficulty, but then with increased malignancy. It travels by the veins or lymphatics; it produces metastasis in 95 per cent. and endocarditis in 6 per cent. of cases. Abscesses of the lung and kidneys and pustular skin eruptions are very frequent; the coccus is found very often in the blood and urine. Staphylococcic infection is usually acute.

3. *Pneumococcic Infections.*—In all cases of pneumonia (*q. v.*) the pneumococcus appears in the blood (pneumococcemia). In actual sepsis following pneumonia, it enters the blood through the pulmonary veins. Infection through the ear or bile tracts is less frequent, but in the latter type, is particularly lethal; metastases occur in 25 per cent. and endocarditis in 6 per cent. The pneumococcus alone may produce pus in the joints, skin, thyroid and serous membranes. Netter states that pneumococcic sepsis is less severe than other types, but it is often malignant. It may be associated with the pyogenic cocci. It is yet unknown why lung infections so frequently cause brain metastases.

4. *Colon sepsis*, which is less frequent, may follow wounds, suppuration in cholelithiasis, cystitis, pyelitis, or intestinal obstruction. Endocarditis is exceptional, and sepsis (78 per cent.) is more common than metastasis (22 per cent.). Infection was hematogenous in the majority of Rovsing's 285 cases of colon infections of the urinary organs; the trouble began as acute nephritis or pyelitis in 180 cases, and fever, hematuria and later, pyuria were common.

5. *Gonococcic infection* is rather more benign than the other types. There may be the more common gonorrheal arthritis or gonorrheal

[1] Schottmueller differentiates three strains of streptococci by cultures on blood-agar: (i) S. pathogenes seu erysipelatos; fine colonies, with round, clear, hemolytic areola. (ii) S. mitior seu viridans; very fine green colonies. (iii) S. mucosus, possibly identical with the pneumococcus; the green colonies after twenty-four hours present a glistening, mucoid appearance. (The S. saprophyticus (anhæmolyticus) produces no change in blood-agar plates.)

endocarditis, pericarditis and pleuritis. Thayer found the gonococcus in the blood (*v.* Gonorrheal Infection).

Other forms are less frequent, as typhoid suppuration in the pleura, bones, meninges, etc., sepsis from the pneumobacillus, meningococcus, etc. Pyocyaneus sepsis is attended by rapid pulse, vomiting, diarrhea, and a hemorrhagic diathesis.

(*B*) Topographical.—The clinical varieties of sepsis are legion and might be extended to the chronic infections as the syphilitic spirochete sepsis, to the zoönoses, malaria, trypanosomiasis or certain intoxications as meat poisoning. (1) *Ulcerative endocarditis;* (2) *cholangitic sepsis;* (3) *septic sore throat;* and (4) *otogenous sepsis* (*v.* Sinus Thrombosis, Meningitis, Brain Abscess) will be considered elsewhere. The two following localizations are of importance to the practitioner and internalist:

(5) *Osteomyelitis* is either a solitary metastasis or part of a generalized sepsis. Even when a single bone is involved, the blood usually contains the causal microörganism. It is due to the staphylococcus chiefly; then to mixed infection with the streptococcus, which is found in all fatal cases, or with the pneumococcus; and but rarely to the typhoid organism. In most typhoid and pneumonia autopsies, the causal bacteria are present in the bone-marrow. Acute osteomyelitis is most frequent in the growing bones of children. Trauma is a promoting element and the onset occurs with *chill,* fever, vomiting, diarrhea, headache, and *bone pain.* It is usually first seen and often mistaken by the practitioner. The rare chronic cases, which last one year or even thirty, may simulate sarcoma or syphilis.

(6) *Puerperal fever* is caused chiefly by the streptococcus (95 per cent.). Its varieties are (*a*) *toxemia* or *sapremia,* caused by toxins or putrid decomposition products respectively. Even in cases apparently of this group, bacteriemia may be found (colon bacillus, saprophytes or streptococcus). The rare puerperal tetanus is wholly toxemic. (*b*) *Lymphangitic form:* lymphangitis, from infected vaginal or cervical wounds, may cause (i) moderate, benign, localized *parametritis.* (ii) General *septicemia,* from vaginal or cervical wounds, infection following the lymph vessels through the pelvic cellular tissue to the general circulation. The symptoms appear within a day or so after delivery. Death may result in two to fourteen days, or encapsulation and massive cellulitis may develop. (iii) Lymphangitic *peritonitis* is more frequent (20 per cent. of fatal cases). It begins three or four days after delivery and is usually generalized and suppurative. Infection may travel directly to the peritoneum or, more often, by retrograde lymphatic routes (Virchow's "erysipelas grave internum puerperale"). It is more common in labor at term than in abortion. The symptoms are chill, fever, tense pulse, vomiting, abdominal pain and distention, and often effusion and diarrhea. In many apparently purely peritonitic cases bacteriemia is found. Early death is the rule, commonly in five to six days, but encapsulation is possible. (*c*) The *thrombophlebitic* (pyemic) type occurs in 50 per cent. of fatal cases. It commonly begins in ulcerative or ichorous endometritis, in contradistinction to the lymphangitic form of vaginocervical origin. Infective thrombophlebitis travels (i) from the upper

veins by way of the spermatic veins, to the cava and left renal vein; the phlebitis may actually extend into the vena cava; (ii) from the lower plexus, by way of the uterine veins into the hypogastric veins, and thence to the inferior cava. Bilateral venous thrombosis is common (45 per cent.). Metastases are common in the lungs, kidneys, joints, muscles, and spleen. Ulcerative endocarditis, meningitis and peritonitis may be present. The course may be stormy from predominating toxemia or slower with metastases. The fever is irregular, the pulse rapid, the chills severe, the anemia marked, and streptococcemia is present in over 90 per cent.

Diagnosis.—The direct diagnosis rests upon the detection of an atrium, the symptoms of toxemia and metastatic suppuration and the blood cultures. The *differentiation* from typhoid, malaria, and miliary tuberculosis is fully considered on pages 46 and 47. Solitary local or visceral symptoms, as osteomyelitis, arthritis, endocarditis, etc., may cause confusion if not viewed in their broad relations to a possible sepsis.

Prognosis.—The prognosis depends upon the organism, *its virulence* and *dissemination*, the patient's *physiological resistance* (whence the obviously poor outlook in terminal infections), and finally on the *surgical accessibility* of the primary lesion or metastatic foci. Only 17 per cent. of Lenhartz's cases with bacteriemia recovered, yet recoveries are recorded in cases of gonococcemia and streptococcemia. A stormy onset and chills are ominous. A case of Lenhartz with empyema, lung abscess, panophthalmitis, diffuse venous thrombosis and multiple osteomyelitis recovered. Puerperal infections, with the large wound surface and frequent hemorrhages, are severe, the mortality of all forms being 55 per cent., and of severe forms 65 per cent. (Curschmann). The streptococcus has been regarded with particular dread; however, Bertelsmann states that the prognosis is twice as favorable as in staphylococcic invasion.

Treatment.—Surgical *prophylaxis*, even in apparently insignificant wounds and obstetrical asepsis is important, especially in subjects reduced by general diseases. Treatment of existing sepsis consists in giving the largest possible amount of food which can be digested; in free administration of *salt solution* by rectum or transfusion, to flush the toxins through the kidneys; and in the free use of *alcohol* The treatment of hypostasis, diarrhea, vomiting and chills is symptomatic. The temperature is seldom influenced by drugs safely. The internal use of germicides is justly abandoned. We have not observed the slightest benefit from Credé's silver ointment. Credé's collargol has been used with benefit, intravenously, in doses of one to two and a half drams. Nuclein and ʒj-ij of fresh brewers' yeast are also advised. *Surgical intervention* is indicated whenever definite accessible foci are localized.

Vaccine and Serum Therapy.—Nature heals and the progress to recovery is usual, if infection is not too rapidly fatal. The so-called immunity principles reside in the phagocytes and soluble, probably protein bodies, preformed in the body and increasing under the stimulus of infection. The various portals of entrance of the infective agent into the body are detailed above; whereas the surfaces of the body are

inhabited by myriads of bacteria, they resist infection usually, the epithelium being protective, the hydrochloric acid of the stomach, the overgrowth of the infective virus by the normal flora of the intestine and the acid reaction of the vagina combating localization, etc. Gaining access to the body, the microörganisms produce toxins, which in turn excite the production of antitoxins. The antitoxin is, then, a reaction product of the organism against the toxin. *Immunity* is *active* when the body, in response to infectious germs (antigens), protects by producing its own antibodies; it is *passive* when protection is achieved, *e. g.*, by the use of antidiphtheric serum in which the antibodies formed in the blood-serum of another animal are employed. The immunity or protective forces of the body are the bacteriolysins which destroy the bacteria, the agglutinins which clump the virus, the antitoxins which neutralize the toxin and the opsonins which so alter the bacteria that they fall an easy prey to the phagocytes.

Guided by the opsonic index, bacteria, dead or alive, may be injected to stimulate the lagging reactive powers of the body; the bacteria are the antigen. Vaccine therapy is indicated, theoretically, only in subacute and chronic infections; in acute general infections, particularly of the fulminating type, vaccines are contra-indicated, since they expose the patient to greater danger, by imposing the negative phase (in which the opsonic index is lowered). In septicemia, pyemia and grave sapremia, vaccines should be avoided or employed only with the utmost caution. In surface infections, in furunculosis, in lingering infections from mild types of parasites, and in infections with slow invasion, vaccines sometimes may be useful. The average interval of inoculation is five to ten days. The range of dosage is 100 to 1000 millions in staphylococcic infections; 5 to 200, in streptococcic; 10 to 200 in pneumococcic; 5 to 500 in gonococcic; and 10 to 200 in colon infections. Vaccines should be autogenous. In commercial stock vaccines dangers linger beyond those inherent in vaccine therapy in general; the uncertainty of the source of these cultures, of their age, potency, dosage and purity are most important.

Antistreptococcic and other sera are most uncertain.

TYPHOID FEVER.

The name (*typhos*, *stupor*) refers to the clouded mentality, and dates from Hippocrates. Bright, Louis and others recognized that ulceration of the bowels occurred in certain continuous fevers, but typhoid was first described as a disease separate from typhus by Gerhard and Pennock (1837).

Definition.—Typhoid fever is a *general infection*, characterized—

1. *Etiologically* by the Bacillus typhosus.

2. *Anatomically* by hyperplasia and ulceration of the lymphatic structures of the intestine, hyperplasia of the spleen and mesenteric glands, and by parenchymatous degeneration in other organs.

3. *Clinically* by a characteristic fever, roseolous eruption, enlargement of the spleen, slow pulse, a peculiar serum reaction, typhoid bacillemia, and often by intestinal symptoms.

Etiology.—The *typhoid bacillus* was first detected by Eberth and Koch (1880), and first cultivated by Graffky (1884).

1. Characters.—It has thick, rounded ends, is three times as long as wide, in length is one-third the diameter of a red blood corpuscle, and its numbers are usually proportionate to the degree of infection. It is polymorphous, sometimes thread-like, contains no spores, is flagellated and actively motile. It is a facultative anaërobe growing on various media, especially on agar, gelatin and, characteristically, on potato. It does not produce acid, indol, ferment lactose or coagulate milk. It stains with aniline dyes but not by Gram's method; it produces an *endotoxin* only by the disintegration of the bacillus itself.

Typical lesions have been produced in chimpanzees and goats.

Its tenacity of life is great, the germ persisting for years in bone lesions and from three weeks to three months after death. It resists drying for months but in water seldom lives longer than three weeks and its multiplication is prevented by saprophytes. The germ may live for months in ice, sour milk, feces or in the upper layers of the soil. It may survive a year in soiled clothes, which explains contagion in washer-women. It is killed in a few hours by direct sunlight.

2. Location.—It is found in the *typhoid ulcers*, the lymphatics of the mesentery, the *spleen*, the *feces*, *blood*, *gall-bladder*, *urine*, roseolæ, less frequently in the lungs, esophagus and mouth, and in various secondary foci, as in pleurisy, pneumonia (sputum), endocarditis, meningitis, parotitis, osteomyelitis and abscesses. It has been isolated from the blood of the fetus in maternal typhoid infections.

3. Entrance.—Its chief and probably sole *atrium* is the *digestive tract*, the infection being carried in water, milk, butter, ice, vegetables or oysters. Water or milk infection is the usual explanation for "explosive" epidemics. As shown in the Spanish-American and Boer wars, dried feces containing the bacilli may be carried by sand, flies, cockroaches and other insects.

4. Exit.—The germ leaves the body chiefly in the *feces* and *urine*, which are dangerous far into convalescence; they are the chief means of dissemination, the disease being less often conveyed directly from one individual to another, although direct infection occurs in hospitals and camps. Lentz found that after a typhoid attack 4 per cent. of persons become "chronic carriers of typhoid bacilli," the microbes remaining in their dejecta for one, fifteen or even fifty-four years. Soper describes a cook who, though perfectly well herself, was a "bacillen-träger" and caused 26 cases of typhoid fever in five years, and 54 typhoid infections are attributed to a dairy hand. The bacilli-carriers are twofold: the primary, who carry bacilli in the blood or stools, but resist infection or reaction and the secondary carriers, who have suffered infection—the more common class.

Alimentary infection is the most common, occurring by water in 71 per cent. of typhoid infections, by milk in 17 per cent., by flies in 4 per cent. and by clothes, dust, etc., in 8 per cent. (direct contact, 3 per cent.), in 35,674 cases (Schüder). Kayser holds that milk causes 27 per cent., water 15 per cent., and typhoid-carriers 10 per cent.

Predisposing Etiology.—1. Reduced Physiological Resistance.—Reduced resistance, as from overwork, mental depression; decreased hydrochloric acid formation or dilated stomach, is a frequent predisposing cause; yet typhoid very often occurs in the robust. It is claimed that the weak or emaciated are immune—*e. g.*, those with syphilis, tuberculosis, cancer, endocarditis or anemia. Most cases occur in time of war among the poorer classes, or among recent residents in typhoid localities (Trousseau).

2. Season.—For unknown reasons most infections develop in the late summer and fall ("autumnal fever").

3. Age.—The years from fifteen to twenty-five include 56 per cent. of cases; a few cases occur in the first year of life. Their number increases from the first to the fifth year. Between the fifth and fifteenth years as many are affected as after thirty-five. The disease is relatively rare after fifty.

4. Geographical Distribution.—Although it is the most common continued fever in temperate climates, it is a world-wide disease and sustains a most intimate relation to the *water supply*, the disposal of *sewage*, the density of population and personal hygiene.

Immunity.—Immunity bears no relation to the severity of the disease; usually conferred after one attack, it is not life-long nor as frequent as, *e. g.*, in scarlatina. In the Hamburg epidemic (1887) 2.4 per cent. of the cases were second attacks. Second attacks may equal or exceed the original in severity.

Symptoms.—The general clinical picture offers more variability than any other infection. *Typhoid is not an intestinal disease, but a general infection,* with *bacillemia.* Its forms are:

1. The typical enteric, frequently with diarrhea, tympany, hemorrhage and perforation.

2. The septicemic, without any or with very slight intestinal lesions; the Widal reaction and bacillemia are present.

3. Other localizations than enteric, *e. g.*, in the lung, bone, etc.

4. Mixed infections—malaria, streptococcus, colon bacillus, etc.

5. Paratyphoid, with close clinical resemblance to typhoid but with organisms differing culturally and otherwise (p. 47).

The *incubation* lasts two or three weeks, with symptoms more vague than in other infections, *e. g.*, depression, pains in the head, back or limbs, disturbed sleep, chilliness, anorexia, epigastric oppression, constipation or diarrhea. The incubation was three days in a girl who drank a typhoid culture with suicidal intent.

The *first week* corresponds anatomically to the intestinal catarrh and the beginning of medullary infiltration of Peyer's plaques. The disease dates from the fever, which each evening is from 1° to 1.5° higher than on the previous day, until a temperature of 103° to 104° is reached. The pulse is full, dicrotic and rapid, but slow in proportion to the fever. The tongue is coated white. Thirst, anorexia, dry cough, enlarged spleen, eruption of rose spots and typhoid bacillemia are found. The abdomen shows slight distention and pain or tenderness over the ileocecal region or epigastrium. Headache, constipation, and apathy are usually present.

The *second week* corresponds to the end of medullary infiltration and the beginning of eschar formation. The fever is higher and usually continuous; the morning remission is less. The pulse is full, faster and less dicrotic. The Widal test appears. The tongue is dry and glazed, as are also the lips, pharynx and mouth, to which the mucus adheres. The face is apathetic, its lines obliterated, the mouth half-open and the upper lip retracted, showing sordes. The voice is weak. The bronchitis increases, and the urine is febrile. Meteorism appears and yellow pea-soup dejecta, involuntary in severe cases. Roseolæ develop during this week. Nervous symptoms increase with higher fever; delirium follows; the patient becomes less querulous; and euphoria from narcosis by the toxins is the rule, while pain suggests complications. The toxemia is due to endotoxins liberated from typhoid bacilli, killed by the tissues. The patient may begin to recover through the opsonins, bacteriolysins and agglutinins, overcoming the toxins, or may die at this stage from hemorrhage, nervous toxemia, etc.

The *third week* is the stage of ulceration. The fever gradually declines with marked morning remissions and unaccountable variations. The pulse is smaller and faster (100 to 120), and loses its dicrotism. The tongue clears and the roseolæ are replaced by miliaria from sweating. Diarrhea may be seen for the first time in this week. When the fever falls the emaciated patient complains of hunger, weakness and pain. Death may occur in the typhoid state from heart weakness, pulmonary inflammation, paralysis of the nerve centres, hemorrhage or perforation.

The *fourth week* (cicatrization) usually marks convalescence, as the average typhoid runs three to six weeks; in severe cases the symptoms of the third week may continue. Convalescence may be uneventful, or marred by slight fever, rapid pulse, weakness, etc.

Symptoms in Detail.—To avoid repetition, and to explain anatomically the clinical symptoms, the typhoid pathology will be incorporated under the appropriate organs, and complications and sequels classified with the usual typhoid manifestations.

1. Fever.—Fever is a cardinal symptom of typhoid; it is toxemic, the toxins so affecting the heat centre (in the corpus striatum), that heat production is increased and its elimination decreased. It is subacute, averages three or four weeks in duration, usually comes on without a chill, and resolves by lysis. Wunderlich diagnosticated typhoid from the fever chart alone. In the *first week,* corresponding to the catarrh and medullary infiltration, the evening temperature is 0.6° to 1° more than in the morning, and *the ascension is ladder-like.* In the *second week,* corresponding to the end of infiltration and to the formation of the slough, the *fever is continuous from one-half to three weeks.* In the *third week* (ulceration) the fever is remittent (the amphibolic stage or *stadium hecticum*). The *fourth week* (cicatrization) is the stage of "steep curves," lasting from three to eight days. When a sudden drop occurs, recurrences and relapses are not infrequent.

Convalescence.—At first the typical curve is subnormal, with a later gradual rise to normal.

Atypical Fever Course.—As to *onset*, two variations may be observed. (1) There is a sudden explosive onset in 10 per cent. of typhoids. The writer saw a sudden rise to 106° on the second day. (2) When there is an initial chill every other disease should first be excluded, yet Osler observed chills in 22 per cent. of his cases (*a*) at the onset, especially in children, and in light forms; (*b*) throughout the course, and accompanied by sweats, the *sudoral type;* (*c*) in complications—pneumonia, otitis media; (*d*) after antipyretics, tubbing, or (*e*) in defervescence from secondary sepsis.

In the *second week* especially high temperature suggests complications; if the fever becomes very high, the course is apt to be severe. In a fatal case, the writer observed fever ranging from 106° to 108° for one week. Sometimes a pseudocollapse may occur, particularly in subjects between twenty-five and thirty years of age. In the *third or fourth week* the

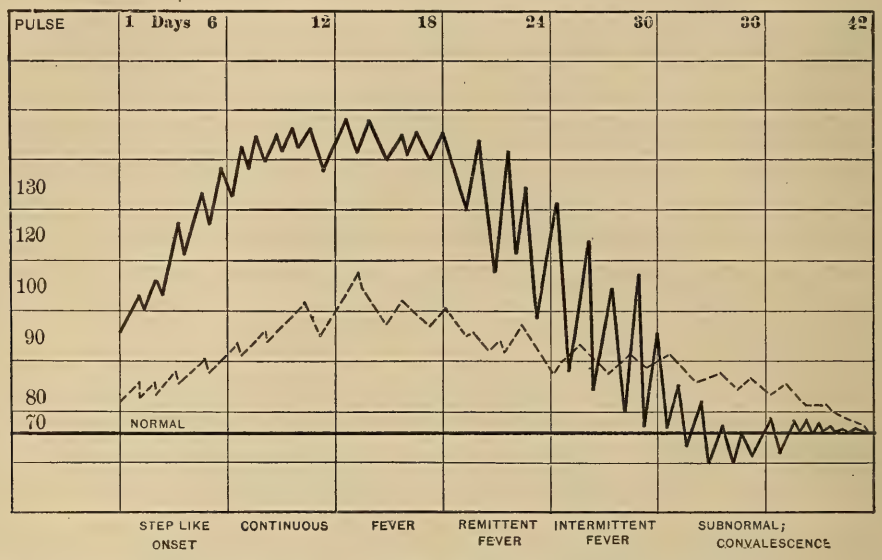


Fig. 1.—Typical typhoid-fever curve. The heavy line marks the temperature curve, and the broken line the pulse curve.

fever exceptionally may decline by crisis; a brusque defervescence occurs in 29 per cent. of cases (Jaccoud).

The fever may be intermittent or remittent throughout the disease, especially in children, the aged and in severe cases; protracted irregularity may suggest miliary tuberculosis or sepsis.

In the *typus inversus*, seen chiefly in children and the aged, the morning temperature is higher than the evening record. Afebrile typhoid is exceptional. Modification of temperature may be caused by excitement, entrance to the hospital, relapse, hemorrhage, abortion, peritonitis and other complications.

2. The Splenic Tumor is a cardinal symptom, occurring early and persisting in exacerbations and relapses. In importance and size, it ranks third to the splenic enlargement of malaria and sepsis. Its cause is the bacillus or its toxin which produces splenic hyperemia and hyperplasia—changes analogous to those in the intestinal lymph structures—and endothelial proliferation in the splenic vessels (Mallory). In the

first week the spleen is hyperemic and swollen to twice its normal size. Its markings are indistinct. In the second week it is darker and more pulpy. By the fourth week it usually has disappeared, but *as lony as splenic tumor persists the disease persists.*

Frequency.—(*a*) *Anatomically* the splenic tumor is present in 98.4 per cent. of the cases. Its rare absence is explained by capsular thickening or induration or infarction of the organ. (*b*) *Clinically*, it is present in 75 to 90 per cent. The percentage varies with the skill and the method of examination (the sign being often absent in children, in adults older than forty-five years, or after hemorrhage). It should be palpated with the patient in the right diagonal position; the examiner should sit on the right side of the patient palpating with the right hand pressing evenly on the abdomen, and the left hand hooked beneath and lifting up the lower left ribs. If the palpating *fingers* are *not pressed too deeply* into the splenic region, the spleen is almost invariably detected. Tympany

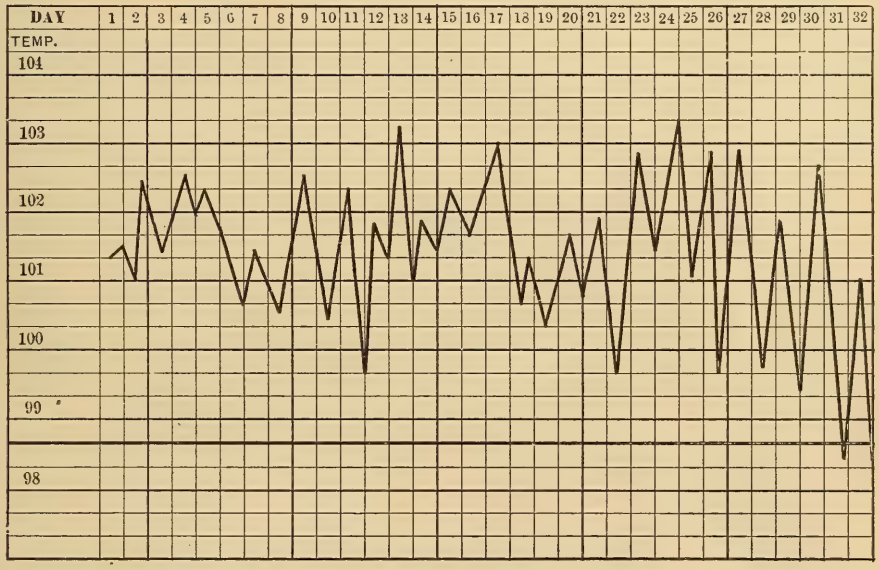


FIG. 2.—Typhoid fever, irregular fever curve throughout the course.

may obscure the splenic tumor. Percussion of the spleen gives utterly unreliable results. The author has twice seen the spleen ruptured by rough palpation. Rare complications are abscess, infarct and spontaneous rupture (38 instances, Bryan, 1909).

3. SKIN.—(*a*) *Rose spots* (roseola typhosa, *tâche rosée lenticulaire* of Louis) constitute the third cardinal symptom. They are *round spots*, varying in size from a pin-head to a lentil (lenticular), *rose-colored*, slightly elevated, purely hyperemic and therefore *disappearing on pressure* and *reappearing on its release.* Pustulation is rare (5 cases, Eggleston), as also are fusion, vesiculation and hemorrhage—the term petechia being inappropriate. They *usually appear from the seventh to the tenth day.* They occur *in crops*, during two weeks, each crop lasting from three to five days. Occasionally they outlive the fever. They appear on the lower chest and abdomen, though sometimes twenty-four hours earlier on the back, because of its warmth. They rarely occur on the neck or

face, and are more frequent on the proximal parts of the extremities than on the distal. (If many spots appear here, they are probably not roseolæ.) They number five to ten, or even thirty. They are more abundant in women, less so in children and the aged, and seldom so numerous as to resemble measles closely. Eichhorst found them constantly in 2044 cases, Osler in 93 and Murchison in 33 per cent. Typhoid bacilli in the roseolæ (in 100 per cent.) may establish a doubtful diagnosis, but rarely an early one; in miliary tuberculosis, meningitis and pneumonia, roseolæ are very rarely encountered.

(*b*) *Other Cutaneous Manifestations.*—The typhoid odor is musty or semicadaveric. Furunculosis is the most common cutaneous complication. Miliaria are frequent, though less than in sepsis; usually appearing in the third or fourth week, they indicate convalescence. The author has observed herpes only ten times in upward of 2000 cases; it is far less frequent than in pneumonia, malaria, typhus, meningitis and influenza. Decubitus occurs in 1 per cent., mostly in severe and hospital cases. (See THERAPY.) Noma is a rare complication. In 204 cases of gangrene collected by Keen, 50 per cent. occurred in the leg and 25 per cent. in the face, neck and trunk. Sweats and chills occur in the "typhus sudoralis," especially in the latter half of the fever course, described in Naples by Borelli and in Paris by Jaccoud. Edema is due most frequently to phlebitis, sometimes to anemia, and rarely to nephritis. The skin sometimes shows atrophic striæ cutis distensæ like those of pregnancy. Other cutaneous manifestations are: Urticaria; desquamation (in 7 per cent.); erythema, even of the nodose or exudative forms; morbilliform rashes; purpura; pelioma typhosum or the *tâche bleuâtre*—pale blue spots, measuring 4 to 10 millimeters—due to pediculosis and therefore seen in parts nearest the pubic or axillary hair (Piednagel, 1837); erysipelas or phlegmon; and alopecia after the typhoid attack, rarely followed by permanent baldness.

4. CIRCULATION.—(*a*) *The slow pulse is the fourth cardinal symptom.* It is lower than the fever would justify, especially in men under forty years, *e. g.*, pulse 90 with the fever 103°. Its slowness is due to *an inhibitory action of the toxins* on the medulla or the heart. The pulse may range from 50 to 60, with the fever above 103° and the respirations above 40. Slowness is of good import. *Dicrotism*, evidenced by a double shock, occurs in 80 per cent. of cases, which, with slowness, suggests typhoid. It is usual in adults, but is absent in children and often in adults at the fastigium in very severe cases. Since it indicates lack of arterial tone, it is also absent in arteriosclerosis, or in vasomotor paralysis. The systolic *blood-pressure* is 115 to 125 mm. A fast pulse early in the typhoid course is a poor prognostic, especially in men (heart or lung inflammation, perforative peritonitis, hemorrhage and severe intoxication). It may also run up on visiting days, in women, children, emaciated and nervous individuals, or in the stage of great temperature variations. Irregularity or crossing of the pulse and temperature curves is ominous. Early in convalescence, when there is subnormal temperature, the pulse is usually normal in rate or slow, from depressed conductivity or myocarditis. Later it is usually above

normal, especially after getting out of bed, or after a bowel movement. On the whole, bradycardia in convalescence is more common than tachycardia.

(*b*) *Heart Muscle.—Parenchymatous degeneration,* evidenced by albuminous granules, nuclear swelling and pigmentation, makes the heart flabby, soft, pale and even waxy. Fatty and hyaline degeneration, vacuole formation, periarteritis, endarteritis and segmentary myocarditis are the usual findings in fatal cases. *Interstitial myocarditis,* like that of diphtheria or scarlatina, occurs in over 50 per cent. of typhoid cases. Its clinical signs appear at the end of the second week, lasting into the fourth week, or develop first in convalescence. The symptoms are *gradual dilatation* of the left ventricle, weak tones (especially the first tone at the apex), accentuation of the second pulmonic, a systolic bruit, and a fast, irregular pulse. Tachycardia, without myocarditis, may result from the toxins acting on the medulla oblongata. It usually regresses completely, and chronic myocarditis rarely develops.

Dilatation of the right heart may be observed in severe cases. Less frequent are the following complications: Cardiac thrombosis and embolism; pericarditis, once in 1000 typhoid autopsies, mostly fibrinous

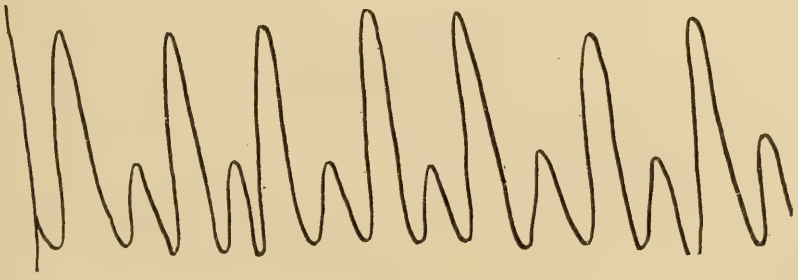


Fig. 3.—Pulse tracing in typhoid, showing dicrotism.

and in complicating sepsis; and endocarditis, once in 1000, mostly mural over myocarditic patches, sometimes due to Eberth's bacillus. *Collapse,* with its usual signs, may cause death from heart degeneration, or *from toxic vasomotor paralysis* (as in diphtheria or pneumonia); myocarditis is the most frequent cause, while pulmonary embolism, uremia, cerebral hemorrhage, etc., are far less common. Sometimes no clear cause is discovered at autopsy. Sudden death is recorded in 80 instances (Dieulafoy). Collapse develops most often during convalescence and mostly in strong men (80 per cent. of Dewèvre's 140 cases) (see Prognosis).

(*c*) *Vessels.—Phlebitis typhosa* occurs in 1 per cent. of cases, being marantic, or infective due to Eberth's bacillus or pus cocci, when pain and fever are present; it occurs chiefly in males and is usually left-sided, since the left iliac vein has a slower current, being crossed by the right iliac artery. The symptoms are pain, coldness, edema and sometimes a tender, palpable cord, manipulation of which may precipitate embolism. Thrombosis usually occurs in the saphenous vein where it empties into the femoral, sometimes in the popliteal vein, and rarely in the arms. It may extend from the leg to the cava, when the other leg may be

involved. There is some danger of detachment, pulmonary embolism, and sudden death; but relative recovery is usual after two or three months. L. A. Connor believes that thrombophlebitis develops in 10 to 15 per cent. of typhoids and causes embolism (33 per cent.), post-typhoid chills, and "tender toes" (usually considered as a plantar neuritis). Arteritis typhosa, most often femoral, is rare, W. W. Keen collecting only 44 cases, and it is specific, caused by the typhoid bacillus. Spontaneous gangrene was noted in 44 typhoid patients, among 180 cases of gangrene in youth after various infections (Barraud, 1904). Arteriosclerosis may be initiated by typhoid.

(*d*) *The Blood.*—The *red blood cells* average 4,000,000. Anemia is most frequent during the third week or in relapses. An apparent increase of the red disks may result from loss of fluids, as from diarrhea. The hemoglobin decreases slightly in excess of the oligocythemia. The *leukocytes* decrease from 8000 to 1700, averaging 5000 per cubic millimeter. *Leukopenia* is more common in typhoid than in any other febrile condition and helps to differentiate it from pneumonia, sepsis, meningitis, etc. Severe infections always exhibit a low count; the converse is not true. The polymorphonuclear neutrophiles are absolutely and relatively decreased to below 60 or 50 per cent., while they are increased in most other infections. The large mononuclear and transitional forms are relatively increased. The eosinophiles are greatly decreased; their return is a good prognostic. *Leukocytosis* occurs only in complicating inflammations.

Typhoid is *a primary bacillemia or septicemia* (Fränkel and Simmonds, 1885). The bacilli reach the blood from the lymphopoietic organs, and from the blood reach the feces secondarily by way of the bile or by ulceration of Peyer's patches. A *positive blood culture* is the safest and surest means of diagnosis *in the first week of typhoid.* Cultures, repeated day after day, are positive in 100 per cent. in the first week, in 71 per cent. in the second, and in 36 per cent. in the third week. Convalescence is rapidly established as soon as the bacillemia ceases.

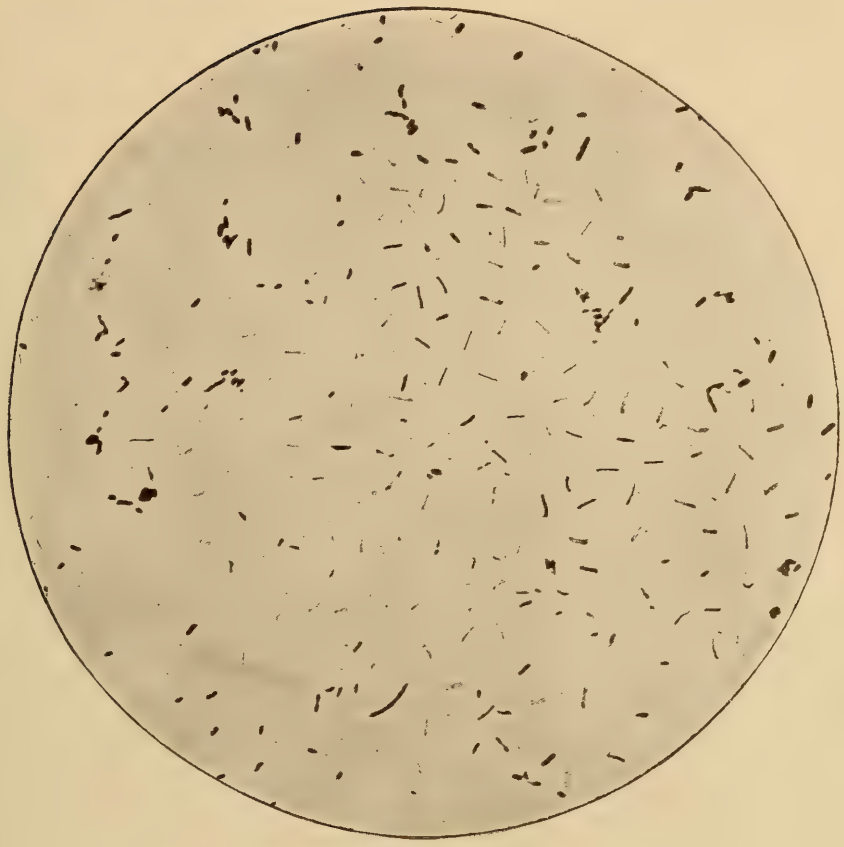
The Gruber-Widal Test.—The agglutination and immobilization of typhoid bacilli occur when they are brought in contact with a typhoid serum. The agglutinins are produced by the tissues reacting to the bacilli. Gruber pointed out the value of the test in proving the existence of a previous typhoid, while Widal applied the laboratory facts at the bedside.

Technique.—A bouillon culture, not more than eighteen hours old, is used, taking forty parts of the bouillon and one of the blood (dilution of 1 to 40), since the test is quantitative rather than qualitative. The test may be made macroscopically in the test-tube, or on the microscopic slide, where immobilization, clumping and agglutination of the bacilli may be observed, sometimes in a very short time—an hour is the usual time-limit for the test. Ficker employs dead cultures with equally accurate results, and this method is more convenient and safe than the use of living bacilli (Plate I).

Significance.—The reaction may be found thirty years after a typhoid attack. It is sometimes absent in typhoid, particularly in severe cases,

PLATE I

FIG. 1



Bouillon Culture of Typhoid Bacilli before the Addition of Diluted Typhoid Serum. × 500. (After Cabot.) Serum diagnosis.

FIG. 2

The Same, Five Minutes after the Addition of Typhoid Serum (dilution 1 to 10), showing Typical Clump Reaction. × 400. (Cabot.)

and it often develops late, *e. g.*, not until the seventeenth or sixty-seventh day. Cabot found the reaction in 97 per cent. of 5978 typhoid cases. In Osler's series it appeared in 93 per cent. before the eighth day. When it appears in the first week, it is due to a "silently developing" infection (Widal), *i. e.*, the typhoid is more advanced than we appreciate. The test is most significant when it is absent early and develops later. Typhoid bacillemia decreases as the Widal develops. The Widal reaction is specific; its occasional occurrence in icterus is probably due to a previous attack of typhoid. Some maintain that the reaction is not one of infection, but one of immunity, agglutination becoming most marked toward the end of the disease.[1]

5. Nervous Symptoms.—Typhoid was once called "nervous fever" (*febris nervosa, nervenfieber*). The nervous system may be conspicuously involved even in the incubation stage.

(*a*) *Headache.*—Toxemic headache is almost invariable, being most often frontal or occipital, constricting or throbbing. It disappears in the second week, but if it lasts or develops late, it may indicate meningitis. Insomnia may be both early and lasting. The early excitation passes into the later depressive euphoria of the typhoid state.

(*b*) *The typhoid state* begins in the second week (*v. s.*) with apathy and delirium, which is usually mild. The patient may attempt to get out of bed, and, in alcoholics, delirium tremens may be simulated or actually develop. In mild cases delirium lasts into the third week. In irritable subjects or in overwhelming intoxication, we may have photophobia, stupor, coma, loud breathing with an open mouth, involuntaries, waving of the hands (*floccitation*), leaping of the tendons (*subsultus tendinum*), picking of the bedclothes (*carphologia*), trismus, contractures, grinding of the teeth and rigid neck, which symptoms are due to toxemia. In coma-vigil the patient is semicomatose subjectively, but the open, unseeing eyes give a "watchful" appearance. Anatomically a "wet-brain" is found. Convulsions (0.2 per cent.) may develop in children, in severe intoxication, or from cerebral complications as meningitis, encephalitis, embolism, etc.

(*c*) *Nervous Complications.*—The author has seen five typhoids taken to the detention hospital in the third week under a diagnosis of mania. *Psychoses* are due to the exhausting inanition of protracted toxemia, or sometimes an hereditary predisposition. They are generally *post-febrile* and of the depressive variety such as melancholia; alienists assert that one-third to one-fifth do not recover. Two-thirds of juvenile cases recover (Edsall). Typhoid may cure a previous insanity.

Meningitis occurs in 0.5 per cent. of cases, generally as a late complication. Meningitis may be due to the pneumococcus, pyogenic cocci, tubercle bacillus (4 cases recorded) or Eberth's bacillus, which has been found on lumbar puncture in over twenty-six cases (Cole), of which half were serous and half suppurative. Typhoid meningitis may develop without typhoid lesions in the bowel (Henry). In a patient recovering

[1] Chantemesse reports an ophthalmoreaction, similar to the tuberculin reaction of Calmette. Gay has devised a skin test; and a hemolytic test is suggested, similar in principle to the Wassermann test.

from typhoid and exhibiting no meningeal symptoms, bacilli were found in the cerebrospinal fluid (Stühmer). Meningitis must not be confounded with pseudomeningitis or "meningismus," in which toxemia produces irritative meningeal symptoms as described under typhoid state (*v. s.*). The writer observed hemorrhagic pachymeningitis.

In the *brain*, edema, degenerated and pigmented ganglionic cells, and nuclear multiplication are the most common findings at the autopsy. Hemorrhage, embolism, arterial and sinus thrombosis, and abscess (sepsis) are rare pathological complications. Aphasia, monoplegia, hemiplegia and paralysis of the cerebral nerves are rare clinical complications. The author twice noted hemiplegia developing suddenly in convalescence, probably embolic from detached heart-clots, the patients being allowed to leave the bed too soon. Forty cases of hemiplegia in typhoid are on record.

In the *cord*, myelitis, multiple sclerosis, infantile paralysis and Landry's paralysis are rare complications. The knee-jerks are sometimes increased, especially in emaciated subjects, and ankle-clonus may appear.

Neuroses may complicate convalescence, such as chorea, hysteria, neurasthenia, Basedow's disease, tetany, paralysis agitans, etc.

Neuritis in the single or multiple form (*q. v.*) may follow typhoid. It constitutes the "tender toes" of Hanford and always results in recovery (see PHLEBITIS, p. 32).

6. DIGESTIVE TRACT.—The upper lip is retracted and bleeds easily. Sordes of dried saliva, food and epithelium are observed on the teeth and gums. Though the tongue shows nothing characteristic, early it is white on the dorsum, moist, flabby, and therefore indented by the teeth and its edges are red. It may be moist throughout the disease. The coating is explained partly by the milk diet and lack of mastication. In the second week it becomes dry (from the fever, lack of saliva and mouth-breathing), small and fissured; it bleeds, and a brown coating replaces the white. In the third week or later, a triangular patch clears at its tip. Typhoid bacilli are said to occur in the mouth in 50 per cent. of cases. Acute glossitis is a severe, yet rare complication.

The *throat* is swollen, granular and eroded. *Angina* may usher in the disease and lead to a wrong diagnosis; usually of the ordinary type, it may be due to the typhoid bacillus. The phlegmonous type is very dangerous. Letulle observed yellowish or pinkish-gray ulcers on the uvula or anterior pillars in one out of every three cases.

Parotitis, occurring in 1 per cent. of cases, became less frequent since special care of the mouth has lessened infection ascending Steno's duct. It is usually one-sided, is due to the typhoid bacillus or pyogenic cocci, and is usually seen on the fifteenth day. The prognosis is poor, especially when due to metastasis. Two personal cases, caused by the typhoid bacillus, recovered. Ulceration of the esophagus is very rare; the cicatrized ulcer may produce later stenosis (12 cases, J. E. Thompson, 1904).

In the *stomach* specific typhoid lesions are rarely observed; hematemesis may occur. Early and severe epigastric pain rather contra-

indicates typhoid. Incipient nausea and vomiting occur mostly in nervous women or children; at the height of the disease they suggest meningitis, overfeeding or perforation, and in convalescence dietetic errors. *Anorexia* is observed during the onset and fastigium; great hunger denotes convalescence.

Specific changes in the intestines: (*a*) *Hyperemia, catarrh* and beginning medullary infiltration in the lymphoid structures during the first week, most changes being observed in the lower ileum, whence the name ileotyphoid. (*b*) *Medullary infiltration* or *hyperplasia*, during the second week, occurring first in Peyer's patches and then in the follicles. The patches are oval and parallel with the long axis of the gut; they lie opposite to the mesenteric attachment, are 3 to 5 millimeters high, and are at first red, then paler from compression of the bloodvessels. The solitary follicles, even to the sigmoid flexure (colotyphoid), may be principally enlarged. These changes are not distinctive of typhoid, but are more significant in adults than in children, in whom they are frequent in digestive diseases, the exanthemata and diphtheria. The lymphoid swelling is explained by proliferation of the endothelial cells of the vessels to act as phagocytes; when they degenerate, fibrinous thrombi develop (Mallory). Medullary infiltration need not advance to ulceration; regression may occur, and the lymphatic structures then assume a "shaven beard" appearance. (*c*) *Ulceration* occurs in the third week from anemic necrosis, slough formation and exfoliation of small particles. The typhoid ulcers are usually seen without opening the gut. They have an irregular oval outline, sharp, steep, often undermined edges, with a red floor generally composed of the muscular coat. The lower ileum is most affected and in extreme cases may present almost no normal surface. The solitary follicles, both in the large and small gut, ulcerate at their apices. As to localization of the typhoid ulcer, Baer states that it may occur in any part of the digestive tract (*v. s.* mouth). The pharynx is affected in 2 per cent. of the cases, the esophagus in 4 per cent., the stomach in 2 per cent.; 97 per cent. of the ulcers develop in the small intestine, 89 per cent. in the ileum, 2 per cent. in the appendix, and 33 per cent. in the large intestine. (*d*) *Cicatrization* occurs by granulation tissue and growth inward of the epithelium. The cicatrix is depressed, slightly pigmented and does not narrow the intestinal lumen. Slight bowel lesions are usually present in most cases reported "without intestinal lesions."

The *mesenteric glands* are swollen in the second week, contain typhoid bacilli and are altered like the intestinal lymphatics. The swollen glands usually regress, but may soften, sometimes causing peritonitis or simulating perforation. Six cases of suppuration are recorded; Le Conte reports a recovery after operation.

No parallelism exists between the frequency, constancy or severity of the clinical symptoms, and the degree of anatomical involvement, *e. g.*, in ambulant cases there may be perforation or hemorrhage, or lethal cases may occur with little intestinal change.

Meteorism is less common than usually described. It occurs mostly in the duodenum or jejunum above the ulceration, but sometimes involves

the colon. If much tympany develops the prognosis is poor, since *meteorism is due to toxemic paresis of the intestinal musculature* rather than to local ulceration; tympanites develops in four-fifths of the fatal cases. The heart and lungs may be crowded upward. It may occur with hemorrhage or perforation.

Abdominal *tenderness and pain* occur in half the cases; they are due to: (*a*) Local bowel conditions, as gas, constipation, inflammation in the ileocecal region, or perforative and peritonitic complications; (*b*) to splenic or hepatic tumefaction; (*c*) distended bladder; (*d*) muscle changes or cutaneous hyperesthesia, so frequently observed in fever of any variety; (*e*) pleurisy or phlebitis, etc.; (*f*) typhoid *appendicitis* may simulate ordinary appendicitis which is differentiated only by the blood count, seroreaction or clinical evolution (the fever, rose spots, etc., preceding local pain and tenderness). Gurgling has no diagnostic value in typhoid. A thickened ileum may be palpated.

The *stools* are not pathognomonic save that the Bacillus typhosus, present in 30 per cent. of cases, reaches the bowel by the bile or through the ulcers. The dejecta are thin, offensive, pea-soup-like and alkaline. Because the feces are poor in mucus, two layers form on standing, an upper, cloudy, and a lower, yellowish-gray, granular and flocculent layer. The stools contain red corpuscles and triple phosphates, the latter more abundantly than in any other disease. Shreds or sloughs may also be found.

Diarrhea in the febrile stage occurs in 25 per cent. of cases. Three to six stools daily are the average; if this is exceeded, the type of the disease is severe; prodromal diarrhea rarely occurs. Diarrhea is a *measure of the degree of toxemia* or of *colonic involvement*, and is not a result of ulceration of the small intestine.

Hemorrhage is seen in 5 per cent. of cases. It usually occurs after the first two weeks, and is less frequent in relapses and in children (1 per cent.). *Ulceration into the muscularis* is its chief cause. Oozing, from simple hyperemia and bleeding in the hemorrhagic forms are exceptional. Bleeding is more common in ambulatory and delirious patients. There are no symptoms if the hemorrhage is small (occult hemorrhage); if it be large, *acute anemia* develops, with *collapse*, evidenced by a fast pulse, lowered temperature and pallid skin. The intellect often clears, the spleen becomes smaller and fresh blood, or, later, *black, tar-like movements* are voided (manifest hemorrhage). The abdomen is often distended. There may be dulness on percussion from blood retained in the bowel, if the peristalsis is weak and the hemorrhage is very profuse. In two rapidly fatal cases one and two pints of blood were vomited. Confusion with bleeding piles and bismuth stools is possible. The average number of hemorrhages is four.

Some patients improve at once after hemorrhage, as was remarked by Graves and Trousseau; yet 30 to 50 per cent. die. Large blood clots are ominous, indicating erosion of a large artery. Hemorrhage may cause death within an hour (Trousseau, Leymaire).

Perforation occurs mostly at the time of eschar formation, at the end of the second or in the third week; but it may also occur later—

in the third to fifth week (Leube). Scott found that 92 per cent. occurred between the second and fifth weeks. It develops in 10 per cent. of fatal cases and *in 3 per cent. of all cases,* but it very rarely occurs in children under five years; after this year perforation is half as frequent as in adults. Scott's figures, in 9713 cases, show perforation in one-third of all fatal cases. Perforation is usually low in the ileum, 81 per cent.; or in the colon or sigmoid, 12 per cent.; in the appendix, 3 per cent.; in Meckel's diverticulum, 2.4 per cent.; in the jejunum, 1.2 per cent.; according to Fitz's figures. In 83 per cent. but one perforation is found; several may occur, twenty-five being observed in one patient. It is promoted primarily by *deep ulceration,* and secondarily by movement, straining, coughing, constipation or tympanites; in 75 per cent. of cases perforation occurs in severe infections. Three types are encountered: (1) Least frequently, undiscovered latent perforation; the typhoid state masks the perforation, which affords an unpleasant postmortem surprise. (2)

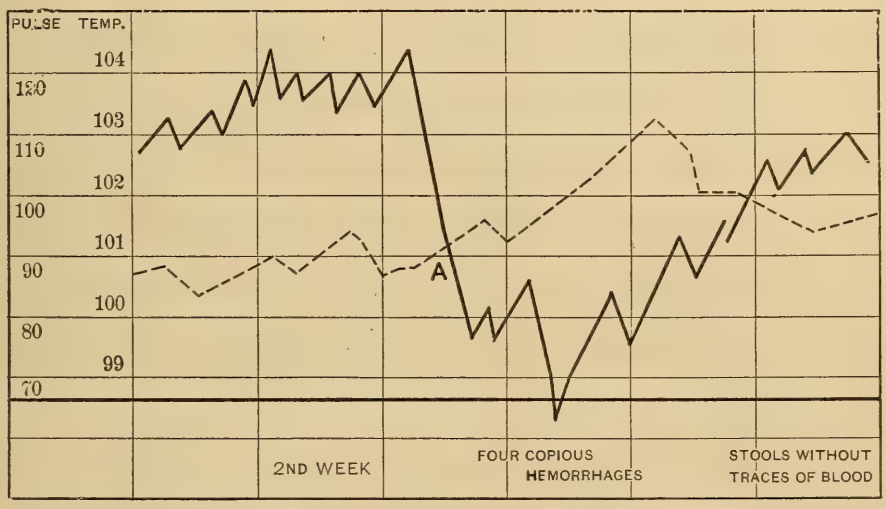


FIG. 4.—Typhoid-fever curve, with fall of temperature (solid line) after hemorrhage and rise of pulse (broken line). Note crossing of curves at *A*.

More frequently, cases with gradual onset, and ambiguous symptoms. (3) Most frequently, cases with sudden symptoms and more or less clear diagnosis. The *symptoms of perforation* are: (*a*) *Pain* due to the perforation itself (in 75 per cent.), usually localized in the hypogastrium or right lower quadrant of the abdomen and followed later by the pains of peritonitis. In 70 per cent. the pain is sudden and severe; in 20 per cent., gradual in onset; and in 10 per cent., absent. (*b*) *Tenderness* on palpation (75 per cent.), evidenced by the board-like rigidity of the abdominal muscles. (*c*) *Limitation of the respiratory movements* of the abdomen, due to pain and tenderness. (*d*) *Vomiting* of gastric contents or fecal matter. (*e*) *Intestinal obstruction.* (*f*) *Abdominal distention,* appearing possibly for the first time—in which event its significance is enhanced—or increasing, if already present; nevertheless the abdomen may be flat; in one typhoid patient, seen first in a dying condition, the scaphoid abdomen contained three pints of pus. Abdominal dis-

tention crowds upward the diaphragm, heart and lungs, and causes hurried breathing. (*g*) Disappearance of the hepatic flatness by reason of free gas in the peritoneum. This is more significant if the abdomen was not previously distended. Perihepatic adhesions prevent obliteration of the liver dulness; a gas-distended colon may overlie the liver, and in some cases the colon normally overlaps the liver. W. W. Herrick suggests the detection of pneumoperitoneum by introducing a blunt needle attached to a wash bottle. (*h*) Other occasional abdominal findings are flatness in the flanks, caused by fluid; the Beatty-Bright friction-rub; absence of peristalsis, rectal or vesical pain or tenderness. (*i*) Marked leukocytosis is found in but half the cases and special emphasis on its absence is most misleading. (*j*) Sudden fall of temperature, rapid rise in pulse-rate and other signs of shock may occur at the time of perforation, though less frequently than the usual descriptions indicate. The writer has seen the pulse-rate remain normal. Dieulafoy's case, successfully operated upon for perforation, resulted fatally from two later perforations. The sudden clearing of the patient's intellect may deceive the inexperienced physician. In the advancing generalized peritonitis, the fever, if it has fallen, rises; the pulse becomes rapid, small and thready; the skin clammy and dusky, and the features pinched (facies Hippocratica). Hiccough is common. The peritonitis rarely remains local; in six of the author's cases, the local abscess ruptured later. Ninety-eight per cent. of unoperated perforations die. Hemorrhage and perforation may occur in association or in succession.

Peritonitis occurs, frequently in ambulatory cases, from rupture of the bowel, gall-bladder, spleen or lymph glands; occasionally from post-typhoid appendicitis; or without rupture of any organ or tissue (in 2 per cent. of fatal cases).

The *liver* in the early stages is anatomically hyperemic, large and firm; later its lobular markings become indistinct from cloudy swelling and fatty change. The organ is softer, and Wagner's lymphomata and some focal necrosis occur. Clinically, some tenderness is not unusual and at times may lead to an incorrect diagnosis of liver abscess or cholecystitis.

Icterus is very rare (0.5 per cent.), and its absence is of diagnostic value. Icterus may occur from duodenal catarrh, gall-stones, cholecystitis or abscess. The author has seen icterus but once in typhoid fever. Single liver abscess is rare, as are suppurative pylephlebitis and suppurative cholangitis. Thirty cases of liver abscess are on record (von Eberts, 1911) and six instances of abscess with pure typhoid cultures (Venema). Suppuration generally follows mild infections; icterus is present in 15 per cent., and in 50 per cent. the abscess is seated in the right lobe.

Typhoid favors the *formation of gall-stones*, which may develop months, years or decades later.

Cholecystitis has been reported by Chiari and others; thirty-nine perforations are on record, among 154 cases (Thomas, 1907). Tenderness, pain, muscular rigidity and enlarged gall-bladder are found in most cases. Typhoid bacilli from hematogenous infection are found in the

gall-bladder, free or in its walls, in nearly all autopsies, although seldom having produced symptoms.

7. The Respiratory Tract.—This is the seat of frequent and manifold symptoms and complications.

(*a*) *The Nose.*—Epistaxis occurs in 20 per cent. of cases, usually early and in young subjects; it is often profuse, sometimes dangerous, but rarely lethal. Croup and diphtheria are rare. The author has seen but two cases begin with profuse coryza.

(*b*) *The Larynx.*—Ulceration of the swollen hyperplastic lymph follicles (like the intestinal changes), occurs in the posterior wall, followed often by edema glottidis. These "bed-sores" are found in 12 per cent. of lethal cases. *Perichondritis* and *necrosis* are attended by pain, aphonia, stenosis, mediastinitis, cervical cellulitis and diffuse emphysema, indicating tracheotomy; the author has seen but three cases. Dupuy collected 255 cases of perichondritis and submucous laryngitis, and believes they explain 3 to 10 per cent. of typhoid fatalities. Without operation the mortality is 98 per cent.

(*c*) *Air Tubes.—A dry catarrh* of the trachea and bronchi is very frequent. Rales, retrosternal rawness and pain are present in the *bronchitis* of typhoid. *Bronchiolitis* is regular and specific. It is found especially in the lower lobes. Coughing in early typhoid is of value in differentiation from simple intestinal catarrh. The outlook is less favorable when severe and early coughing occurs.

(*d*) *Lungs.—Hypostasis,* from a dorsal decubitus and weak heart, is often relieved by change of posture. It is very common in weak adults and in severe infections. The percussion note is tympanitic or dull; the fremitus is increased if the patient is not too stupid to talk, or decreased from stagnation of secretion; the breathing, especially the expiration, is distant or sometimes bronchial. Rales, moist, crepitant and subcrepitant, are heard.

Lobar pneumonia may, in rare instances, dominate the early clinical picture (pneumotyphoid) and is of two types: The more frequent type, (*a*) in which at the height of the disease, the pneumonia is a true complication, due to the pneumococcus or sometimes to the colon bacillus and streptococcus. The pneumonic symptoms are atypical, as in most secondary pneumonias, often without chill, with a slower course, and often ending by lysis or, less often, with such complications as abscess or gangrene. More than one-half of these cases die. The less frequent second type (*b*) is due to Eberth's bacillus. The course is that of an initial pneumonia, which does not end typically, but shows later a typhoid temperature, rose spots, Widal reaction, etc. These cases are confusing in their incipiency, and the typhoid is apt to be overlooked. *Lobular pneumonia,* from inhalation, pus cocci or Eberth's bacillus, is a complication. *Abscess* follows pneumonia or sepsis; it occurs in 0.7 per cent. of fatal cases. *Infarct* occurs in 6 per cent. of typhoid fatalities, thrombi from the leg reaching the lungs by way of the right heart and accompanied by hemoptysis, chill, temperature, pain, etc. *Gangrene* (1 per cent. of typhoid autopsies) is usually metapneumonic, but may be caused by perichondritis or aspiration of food.

(*e*) *Pleura.—Serofibrinous pleurisy* (1.6 per cent.) is rare unless the lungs are diseased. Postmortem statistics show it in 6 per cent. Empyema (0.2 per cent.) is due oftenest to Eberth's bacillus. In 1903, 55 cases of pleurisy were collected in which the typhoid bacillus was found.

(*f*) *Tuberculosis.—Acute caseous pneumonia, subacute tuberculous peribronchitis* with a few bacilli, fever and aggravation of the bronchial symptoms, or occasionally miliary tuberculosis, may occur. The sooner tuberculosis begins after typhoid the more unfavorable is its course.

8. Genito-urinary Tract.—(*a*) The *urine is febrile, i. e.*, decreased, acid, concentrated, with a high specific gravity, but the amount of urine depends upon the amount of water ingested. Water is retained in the tissues in protracted fevers. Typhoid bacilli are present in 30 per cent. of cases and may give the urine a glistening appearance. Bacilluria is hematogenous and occurs in severe infections; it is favored by retention and albuminuria; the bacilli multiply in urine of low acidity and usually disappear spontaneously, but may persist months or years; cystitis is due to other organisms (Connell). In convalescence, the urine is increased, is neutral in reaction, and has a lower specific gravity. (*b*) Serum albumin, globulin and rarely peptone are found in *febrile albuminuria.* Serum albumin is found in 50 per cent. of the cases; cylindruria is approximately half as frequent. The prognosis is three times as unfavorable when albumin is found. Albuminuria appears from the seventh to the tenth day, and its average duration is twelve days. It corresponds anatomically to parenchymatous and fatty degeneration of the kidneys. (*c*) *Nephritis* in typhoid is of the acute parenchymatous type. It is often hemorrhagic, and occurs usually in the first three weeks but occasionally in convalescence. It is found in 1 per cent. of cases, mostly in men, rarely in children. Nephritis indicates a severe infection, from which 50 per cent. of the cases die. Uremia and suppression of urine are rare. If not fatal, nephritis usually regresses entirely and rarely becomes chronic. Early marked renal symptoms constitute the nephrotyphoid of French writers. Miliary typhoid abscesses in the kidney may keep up the bacilluria. (*d*) The urine of typhoid exhibits the *diazo reaction* of Ehrlich, due to incomplete oxidation of the proteid metabolism of the aromatic sulphates. The author found it in 98 per cent. of his cases. It occurs in the early stages of the disease, at the acme and during relapses. It closely parallels the bacillemia. It is a valuable sign, though not of differential value. Its absence somewhat contra-indicates a florid typhoid. (*e*) *Cystitis* and *pyelitis* occur, due chiefly to pus organisms or colon bacilli. Simple retention of urine is common in the typhoid state. Hemoglobinuria is rare. Typhoid bacilli may form the nuclei of renal stones. (*f*) The *genitalia* are more often implicated in women than in men. Menstruation often appears early at the onset of typhoid and is suppressed in 60 per cent. of the cases. Hematocele, endometritis, vaginal erosions, ulcers, inflammation, gangrene and mastitis are rare complications. *Pregnancy* is of grave import, especially in its later stages. At the acme of the fever, abortion and premature delivery intervene in 65 per cent. The maternal mortality is increased twofold at least. Blumer and Dobbin report puerperal infection from the typhoid

bacillus. *Orchitis* usually develops late or in convalescence, with chills and fever. Velpeau described the first instance. J. G. Beardsley collected 102 cases, chiefly due to the typhoid bacillus. The patient generally recovers in two weeks. It is frequently associated with urethritis. Emissions are common in convalescence.

Adrenal Function.—According to Sergent, adrenal insufficiency may ensue in typhoid or other infections; peritonitis and hemorrhage may be simulated at the height of the disease, and anemia, prostration and psychasthenia may be evident in convalescence, exhibiting prostration, lowered temperature, the white dermographic line, low arterial tension and collapse, and relieved by adrenal therapy.

9. Special Senses.—Ocular muscle paralysis (neuritis) and inflammation are very uncommon. In convalescence, mydriasis and paralysis of accommodation often result from exhaustion. Uhtoft, in 253 cases of optic neuritis, found 17 cases following typhoid. Involvement of the ear includes functional toxemic disturbance of hearing, abscess and otitis media (2 per cent.). Mastoid disease and meningitis are very exceptional.

10. Muscles, Thyroid Gland and Bones.—The *muscles* anatomically are dry, of a smoked-meat appearance, and may be the seat of hemorrhage, rupture, granulofatty or waxy degeneration, and abscess, due to Eberth's bacilli and pus cocci. Myositis may explain some instances of muscular tenderness with cramps.

In the *thyroid gland* a strumitis typhosa, with suppuration or regression, is due to the typhoid bacillus. Recovery is the rule.

Bone disease is more frequent than the 237 cases of Keen indicate. *Periostitis* is the more common. These post-typhoid complications are due to the typhoid bacillus (75 per cent.), or pus cocci (25 per cent.). They attack the tibia (38 per cent.), the ribs (13 per cent.), femur, ulna, temporal bones, etc., occur chiefly in young individuals and are characterized by indolence and recurrence. *Osteomyelitis* may resemble syphilitic or tuberculous osteopathies. Typhoid bacilli have been found fourteen years after typhoid in osteomyelitic fistulæ; this typhoid septicopyemia is a striking instance of bacterial latency. *Arthritis* may be typhoid, gonorrheal or septic. Keen noted eighty-four cases. It may be poly- or mono-articular, serous or purulent. The hip is most frequently affected. The "typhoid spine" of Gibney, of which Elkin and Halfpenny collected 94 cases (1914), is characterized by pain, often referred to the belly, tenderness and stiffness in the back; these seem to be root symptoms. It is clearly an embolic spondylitis.

Anomalous Courses.—1. Malignant.—The *malignant* or hyperpyretic form is rare. It is severe even in the earliest stages. The temperature rises rapidly and there may be hyperpyrexia. The pulse is weak and rapid. Meteorism, diarrhea and albuminuria are present, and death usually follows the deep intoxication.

2. The slow severe form lasts four weeks to four months. The fever is often remittent with frequent exacerbations. Defervescence may occur or death intervene, marasmus alone being found at autopsy. The severe types of typhoid have decreased during the last two decades.

3. Typhus Abortivus.—*Typhus levissimus, typhoidette,* exhibits a short, mild-course temperature with a short initial stage, or often a crisis, lasting one to three weeks. Bronchitis is frequent, splenic enlargement is constant and meteorism, diarrhea, roseolæ, hemorrhage and perforation are rare. Relapses are more frequent.

4. Typhus Ambulatorius.—*Typhus ambulatorius* (latent or walking typhoid) occurs especially in men, who often endure it by the help of alcohol. There are two types: (*a*) the lighter and atypical, and (*b*) the usual course, very often ending in hemorrhage, perforation, etc. The temperature often is not high, but is usually less at night than during the diurnal activity. The pulse is faster because of exertion. The spleen is often enlarged and the roseolæ are frequent.

5. Typhus Afebrilis.—The afebrile form is most often encountered in family practice. It may be ambulatory and is diagnosticated by the pulse, spleen, roseolæ, Widal test, etc. Atypical cases have increased in recent years.

6. Hemorrhagic.—The hemorrhagic form is characterized by nasal, subcutaneous and intestinal hemorrhages—the blood dissolution of the older writers. It occurs especially in children and alcoholics (0.1 per cent. of typhoid cases). The prognosis is usually bad. The four cases which the author has seen occurred in 1900, in St. Luke's Hospital, where a severe house epidemic prevailed.

7. Visceral Forms.—The so-called *visceral forms* may cause confusion. They include the gastric or bilious types, nervous fever, meningotyphoid, cerebrotyphoid (mania, psychoses), laryngo-, tonsillo-, nephro-, and pneumotyphoid.

8. In children the course is shorter and milder. The younger the patient, the less is the fever. Epistaxis is less common. The pulse is faster than in adults, though after the twelfth year it is equally slow. Roseolæ occur as often in children as in adults, and in some cases are abundant and confluent (between the fifth and tenth years). Bed-sores are very rare, but noma is more frequent than in adults. The nervous system is generally less involved in children, though if the infection is severe, convulsions, irregular pupils, rigidity of the neck, etc., may occur (see Typhoid State). Neuralgias and psychoses are rare. The frequency of aphasia has not yet been explained. Atelectasis, hypostasis and lobular pneumonia are more frequent. Children exhibit less meteorism, hemorrhage (1 per cent.) and perforation. There are fewer anatomical changes. Splenic enlargement is palpable in 50 per cent. Initial vomiting, pain and perhaps diarrhea, are more frequent. Albuminuria and nephritis are relatively rare. *In infants:* Griffith and Ostheimer assembled 325 cases in infants, of which 139 occurred under one year, 187 in the second, and 68 between two and two and one-half years. The mortality under one year (*v. i.*) was 73 per cent., and of the entire group, 50 per cent. Typhoid is rare in the first two years of life, and is sometimes overlooked, being very atypical. The course is short. Bronchitis, splenic tumor and roseolæ are infrequent. If the disease is severe, a very high temperature, meningeal symptoms, and initial vomiting occur, and also a 50 per cent. mortality caused by toxemia, pneumonia, perforation or ulceration of the larynx.

9. In the Aged.—Typhoid is infrequent after the fortieth year. The fever is lower, atypical and often absent. The pulse is faster, irregular and without dicrotism. There is greater lung congestion, owing to weakness of the right heart, and lung complications are more frequent. The nervous symptoms are dominant, often occurring with great depression. Roseolæ are fewer and bed-sores are more frequent. The spleen is less often enlarged because of old infarcts, induration and a thicker capsule. Tympany and hemorrhage occur more often. Convalescence is naturally slower. In 1903, the author had under his care, in Wesley Hospital, a woman, aged fifty-seven and her mother, aged seventy-two, both with typhoid, ending in recovery.

Relapses and Exacerbations.—Relapses and exacerbations differ in degree only. Their explanation is probably involved in the immunity problem, an exhaustion of the antibacterial forces of the body. In the *relapse*, the temperature falls to normal or nearly normal, for five days on the average, and then fever and other signs of typhoid reappear. It occurs in 9 per cent. and varies with the epidemic, sometimes occur-

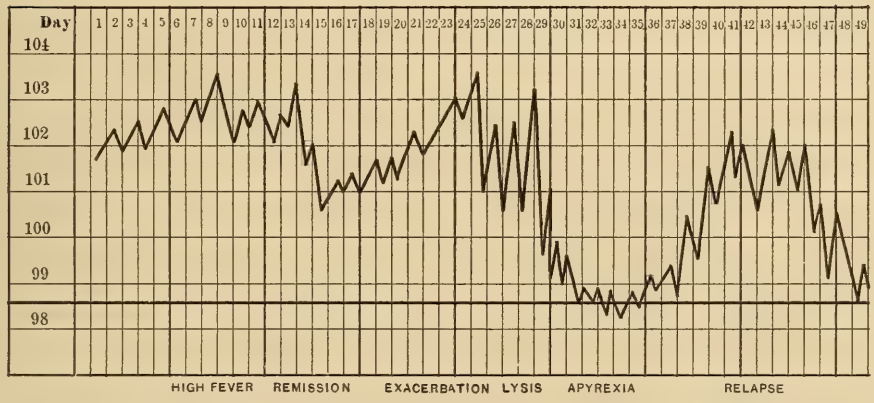


Fig. 5.—Showing remission, exacerbation, lysis, and relapse.

ring in as much as 35 per cent. Relapse is more likely to occur (*a*) in light or moderately severe cases (75 per cent.) than in severe ones (25 per cent.); (*b*) when the splenic tumor does not disappear (but the spleen may have been enlarged from some previous disease, as malaria); (*c*) when the temperature in convalescence does not become subnormal; (*d*) when the pulse is variable and rapid; and (*e*) more commonly in young than in old subjects.

The fever runs the same course as in the first attack, and averages ten days. The pulse is faster and less dicrotic. There are less roseolæ, nervous and respiratory symptoms, hemorrhage (4 per cent.) and perforation (0.7 per cent.), whence, as a rule, the prognosis is relatively good. There are seldom more than two relapses—three or four being very rare. The author has seen five relapses, covering, with the original attack, as many months. They may be afebrile, with a return of the roseolæ, spleen, etc. Relapses should not be confused with Biermer's "after fever," due to inanition or possibly, sepsis. *Recrudescence* is renewal of the fever after it has lessened, but not entirely subsided, and in 50 per cent. is followed by more severe symptoms.

Convalescence.—Convalescence requires two or three weeks and is not certainly established until subnormal temperature is observed. The pulse tends to run high on exertion or excitement. Anemia, emaciation, phlebitis, tuberculosis, psychoses, bone, muscle and other complications may intervene. If the appetite is poor in convalescence, complications are to be expected.

Diagnosis.—General Rules.—1. Observe long and carefully. Say that a diagnosis is impossible in the first few days.

2. No single sign is absolutely diagnostic, except bacilli in the blood and the Widal reaction.

3. Diagnosticate from positive findings and careful exclusion.

4. The typhoid state only indicates toxemia, not typhoid.

5. In doubtful cases remember the great statistical frequency of typhoid.

6. Remember its manifold forms, *e. g.*, pneumotyphoid, etc.

7. Be on the safe side in light, ambulatory or abortive cases as to the diagnosis and treatment.

The Cardinal Signs are: (*a*) Bacillemia is found in practically all early cases.

(*b*) The serotest is successful in 97 per cent. of cases; its absence does not exclude typhoid.

(*c*) The eruption has a diagnostic value in its location, occurrence in crops, time of appearance and short duration.

(*d*) The slow and dicrotic pulse is suggestive.

(*e*) The fever curve shows a gradual, step-like onset, continuous fastigium, steep curves in the breaking stage and subnormal register in convalescence.

(*f*) Splenic tumor is *per se* suggestive after the first week. Splenic puncture, to detect bacilli, is never permissible.

Secondary Signs.—These are, in order of importance, the typhoid tongue, the stools, bronchitis, typhoid state, leukopenia, intestinal symptoms and the diazo reaction.

Symptoms contra-indicating typhoid are coryza, early vomiting and sweating, herpes, endocarditis, retraction of the abdomen, negative diazo test, leukocytosis (unless some complication has occurred), icterus and rigors. Any of the above symptoms may, exceptionally, complicate typhoid.

Differential Diagnosis.—Differentiation may be most difficult from acute miliary tuberculosis which may develop in typhoid. Typhoid may be complicated with septicemia, very rarely with malaria in this climate, or with meningitis (*v.* table on pages 46 and 47).

Exanthematous typhus (*q. v.*) and recurrent fever (*q. v.*) are easily distinguished. *Exanthematous diseases* are rarely confused, except, perhaps, in the initial stage. In 75 instances typhoid and scarlatina coexisted (Fournier). An extensive eruption of roseolæ resembling measles is easily distinguished by the catarrhal symptoms, temperature curve and course.

Fibrinous pneumonia rarely resembles typhoid, and still more rarely occurs with typhoid in its earliest stages by the localization of typhoid bacilli in the lungs. Though typhoid may begin with severe bronchitis or pneumonic symptoms, the intestinal and cardinal findings of typhoid

usually appear after a few days. Sudden onset with chill, pain in the side, abrupt rise of pulse and temperature, rapid respiration, bloody, viscid sputum, herpes, leukocytosis, and a tympanitic note and a few crepitant rales over one lobe, followed later by typical pneumonic infiltration as consolidation advances from within toward the periphery of the lung, establish the diagnosis of pneumonia on the second or third day.

In *intestinal anthrax* fever, splenic tumor and nervous symptoms are accompanied by hemorrhagic vesicles in the mouth, vomiting, colic, hemorrhage from the intestine and kidney, cyanosis and dyspnea, anthrax bacilli in the blood, urine, stools or sputum, and inoculations establish the diagnosis.

Febrile gastritis rarely simulates typhoid; the rare fever is low or irregular, and early gastric symptoms are not common in typhoid. Without blood cultures, paratyphoid may not be distinguished from acute gastro-enteritis. *Febrile enteritis* is characterized by much mucus in the stools, whereas the typhoid stool seldom contains much mucus. Splenic tumor is most uncommon in enteritis. The fever in enteritis is lower or more irregular and the bronchitis, slow pulse and other cardinal signs of typhoid, are absent. Cases of *typhoid developing suddenly* may be confused at first, with grippe, diphtheria or other diseases. *Febrile syphilis* seldom causes diagnostic trouble but must be considered in dubious cases.

Paratyphoid.—The paratyphoid bacillus resembles the typhoid bacillus morphologically but differs biologically; in cultures it forms no indol, therein resembling the Bacillus typhosus, but like the colon bacillus it ferments grape-sugar. In Bacillus A, the growth on potato is very thin; in Bacillus B, very thick; it may very closely resemble Gärtner's bacillus enteritidis. Infection may occur from milk, water and tainted meat. The bacilli are found in the stools, blood, roseolæ and urine, and agglutinate in higher dilutions (1 to 1000 or 2000) than the typhoid bacilli. The first clear cases were reported by Schottmüller, in 1900 and Gwynn, though the term paratyphoid was first employed by Achard and Bensaude in 1896. Paratyphoid constitutes 4.5 per cent. of cases with the general clinical picture of typhoid fever. In paratyphoid the temperature is more brusque in onset, shows greater remissions, and may end by crisis. The pulse is slow and dicrotic. Roseolæ are present in 80 per cent., the spleen is less often enlarged and no leukocytosis is observed. Prodromal catarrhs, reddening of the throat, diarrhea, cervical adenitis, edema of the lids, vomiting, rigors, epigastric pain, herpes, myositis, arthritis, pleurisy, endocarditis and meningitis, are more common than in typhoid and may enable the practitioner to differentiate paratyphoid, clinically. The diazo reaction is positive in 45 per cent. The clinical course covers two to four weeks. Relapses are common. The infection is usually mild and rarely fatal (3.6 per cent.). Ulceration of the intestinal lymph structures is rare in the fatal cases, though other necroses occur, thus explaining the hemorrhage (5 per cent.). Pneumonia and perforation are unknown. Nervous symptoms are not prominent. The blood seldom responds to the Widal test. Grunberg and Rolly hold that the Widal reaction is less a specific than a group reaction, as they found agglutination in the paratyphoid group by typhoid

Pathognomonic signs.	Pulse and circulation.	Fever.	Splenic enlargement.	Blood.
TYPHOID: Typhoid bacilli in the blood, urine, roseolæ or stools. Widal test.	Relatively slow dicrotic pulse early in the disease, especially in men, and in not too severe infections. Endocarditis and pericarditis very rare.	Typical rise, acme, defervescence, and subnormal in convalescence. Remissions and intermissions following continuous fever speak for typhoid. Responds to hydrotherapy as a rule. Sometimes chills at onset or in course.	Very frequent; larger than in miliary tuberculosis; appears in first week.	Bacilli in blood, early; Widal test later. No leukocytosis unless complications, but leukopenia. Polymorphonuclears decreased. Eosinophilia rare. Late anemia.
MILIARY TUBERCULOSIS: Rarely tubercle bacilli in the blood or splenic pulp; tubercles in the choroid.	Rapid, 120 or more, high even during remissions; slowed in tuberculous meningitis and in the aged. Dicrotism uncommon. Sometimes pericardial rub.	No cycle; irregular or remittent. Seldom long continued, and if so, longer than in typhoid. Exacerbations with renewed dissemination of tubercle bacilli. Brain involvement may lower fever or make it variable, as inversed type.	Less frequent; smaller and usually appears later.	Sometimes tubercle bacilli in blood. No Widal. Leukocytosis not often but more than in typhoid. Eosinophilia more frequent.
MALARIA: Plasmodium in the blood.	Pulse moderate in rate; sometimes dicrotic; very rarely endocarditis.	Intermittent, beginning very often with chills; type varies (quotidian, tertian, etc.); often rises high at once; if continuous (subcontinuous), at least made irregular by quinine or usually wholly abolished (for a time at least).	Much enlarged; more than in typhoid; and persists frequently as ague-cake.	Plasmodium, melanemia, no leukocytosis save in pernicious forms. Great and early anemia. No Widal.
SEPSIS (INCLUDING ENDOCARDITIS): Cultivation of pyogenic organisms, pneumococcus, etc., from the blood or various foci.	Usually fast and irregular; cardiac localizations very frequent in sepsis, puerperal especially, and in endocarditis (a cardiac form of sepsis). Cardiac enlargement, thrill, bruit, accentuated pulmonic tone, etc.	Chills, sweats, steep elevations, and sudden depressions almost always at some time, and usually repeated with each discharge into the blood of infective material.	Especially enlarged from sepsis and infarcts. Also often the lymph nodes.	Various organisms. Marked polymorphonuclear leukocytosis very frequent; sometimes absent in endocarditis. Seldom eosinophilia. No Widal.
MENINGITIS: Lumbar puncture (*a*) meningococci, tubercle bacilli, pyogenic organisms, etc.; (*b*) increased turbid fluid, etc.	Rate differs with type; variable.	Differs with variety, but seldom as high as typhoid; no cycle.	Rare.	Leukocytosis in epidemic and purulent types; rarer in tuberculous. No Widal.

serum. A mixed infection with typhoid may occur. In about half the cases due to the paratyphoid B, the symptoms are choleriform. Bacilli-carriers are as important as in typhoid; a wide-spread house epidemic occurred from kitchen infection among the patients and physicians in the Allgemeines Krankenhaus in Vienna.

Trichinosis is differentiated from typhoid by the history, muscular

Skin.	Lungs.	Nervous system.	Sputum, urine.	Alimentary tract.
Roseolæ, typical in location, crops, appearance, containing typhoid bacilli. Sweating and miliaria less often and later. Icterus rare. Herpes rare.	Breathing somewhat increased from toxemia (cerebral breathing), hypostasis or other complications. Rales below and behind. Tympanitic note never heard in typhoid.	Early headache, replaced later by delirium, typhoid state; actual meningitis very rare; intoxication may closely simulate meningitis. Very rarely disk changes.	Typhoid bacilli in urine. Diazo test in nearly all cases.	Mouse-like breath, semi-characteristic tongue, ochre yellow stools, tympany, diarrhea, bowel hemorrhage, perforation, bacilli in stools. Retracted abdomen or early vomiting rare.
Roseolæ absent or extremely rare, and then not in crops. Drenching sweats common. Herpes more than in typhoid. Skin anemic and very often cyanotic.	Breathing increased even 40 to 60; cyanosis and dyspnea without adequate physical findings. Fine and localized rales heard above and in front. Tympanitic percussion note from lung relaxation. Evidences of pleural adhesions, apical changes, fresh pleural rub.	Meningitis tuberculosa very frequent; optic neuritis; choroidal tubercles.	Tubercle bacilli sometimes found in sputum, urine and feces, but indicate old tuberculous lesions, not of miliary tuberculosis. Diazo test less frequent.	Diarrhea not common, but may rarely occur, as indeed may hemorrhages; both result from tuberculous ulceration of bowels.
In severe, eyes subicteric or marked, very frequent and urticaria the next most frequent exanthem.	If lung congestion (severe forms), it is variable, recurrent, shifting from lobe to lobe.	Headache at onset is rare; later is often pulsating or neuralgic. Delirium from beginning in estivo-autumnal forms is frequent. Restlessness, anxiousness. In pernicious forms may have convulsions, coma, etc.	Hemoglobinuria in pernicious malaria. Diazo test fairly frequent, though much less than in typhoid.	Nauseating breath. Sometimes abdominal pain even in mild types; may rarely have diarrhea, even hemorrhage in alimentary pernicious types. Liver congestion frequent, also icterus.
Frequent sweating, miliaria. Septic polymorphous rashes. Subicterus, frequently purpuric skin eruptions, herpes not uncommon.	Bronchitis frequent. Pulmonary infarcts and pleurisy.	Dependent on cause. Septic meningitis (with arthritis, pleurisy, etc.), cerebral embolism; retinal hemorrhages or embolism.	Acute embolic nephritis. Peptonuria.	Septic diarrhea.
Herpes in epidemic cerebrospinal form. Tache cérébrale.	Variable breathing; Biot's breathing.	Early triad, headache, retracted neck and hyperesthesia. Headache violent throughout the disease, convulsions, paralysis, Kernig's sign, etc. Optic neuritis, retinal hemorrhage, tubercles in choroid.		Early cerebral vomiting. Retracted, scaphoid abdomen.

pain and edema in the striped muscles, trichinæ in the muscles, and by eosinophilia. Three cases of double infection are reported.

Prognosis.—The death-rate was 20 per cent. in 50,000 cases collected by Jaccoud. At present it is 5 to 10 per cent. because of more accurate diagnosis (thus including lighter cases), decreasing virulence and better hygiene and treatment.

The *prognostic factors* are:

1. Age: The mortality is least between the second and tenth years, higher after the twenty-fifth, and highest in babes.

2. Constitution: Meagre and muscular patients are more likely to recover than the plethoric or adipose.

3. Sex is immaterial, aside from pregnancy.

4. Acute diseases modify the prognosis, *e. g.*, scarlatina, measles, variola, diphtheria, malaria, erysipelas, etc.

5. Chronic diseases, such as gout, cardiac, pulmonary and renal disease darken the outlook; in alcoholism, 34 per cent. mortality results from weak heart or hemorrhagic diathesis; convulsive affections, such as epilepsy or chorea, may cease; in diabetics the fever is low and the glycosuria may disappear.

The prognosis is better in relapses and worse in recrudescences. In war times the prognosis is usually less favorable; the Spanish-American war was an exception, the death-rate being but 7 per cent., as against 21 per cent. in the Boer war.

Death usually occurs between the eighteenth and twenty-eighth days, and is caused by—

1. *Toxemia*, the most frequent cause of death (40 per cent.), including hyperpyrexia, hemorrhagic types, heart weakness, vasomotor paralysis and cerebral toxemia.

2. *Local typhoid lesions*, as meteorism, diarrhea, peritonitis (in 16 per cent. of fatal cases), or hemorrhage (in 20 per cent.).

3. *Complications*, as lung inflammations (in 15 per cent.), nephritis (4.5 per cent.), etc. In septicemia (*"forme septicémique généralisée"*), the symptoms are high fever, chills, sweats, enlarged glands and spleen. Pyemia may result from acute osteomyelitis, suppurative parotitis, adenitis, phlebitis, etc.

4. *Collapse.* Dieulafoy estimates that sudden death occurs in 2 per cent. of typhoid cases (see page 31). The pulse is always more important than the fever, and early rapidity is unfavorable. Bradycardia and marked agglutination are favorable prognostics.

5. We have already considered the ominous significance of pseudo-collapse, the typhoid state, early delirium, diarrhea and meteorism; of the various severe complications, such as noma, gangrene, laryngeal perichondritis and pneumonia; and of the more severe types, such as the hyperpyretic and hemorrhagic and of the ambulatory variety. Typhoid beginning suddenly entails a high mortality.

Treatment.—Dujardin-Beaumetz said: "The best treatment for typhoid is a good physician." Treatment concerns (*A*) prophylaxis; (*B*) hygiene; (*C*) intestinal antisepsis; (*D*) antipyretic measures; (*E*) diet; (*F*) symptomatic treatment; and (*G*) convalescence.

(*A*) Prophylaxis.—1. Prophylaxis has as wide a scope in medicine as asepsis and antisepsis in surgery. In Chicago, during the years 1889 to 1892, inclusive, there were 4747 deaths from typhoid. In the four years after the drainage-canal was opened there were 2235 cases, that is, the mortality was reduced 75 per cent. In 1905 typhoid was rare.

General prevention is difficult, because of the ubiquity of the germ and the carelessness of individuals. Systematic coöperation regarding *sewage* regulation, *water supply* and typhoid-carriers could efface typhoid. Koch particularly emphasized this point and abolished certain typhoid foci. The difficulties are the infrequent recognition of the particularly dangerous light cases, light forms notably in children (Koch), carelessness and the bacilli-carriers. It is easy to boil water for home use, and yet it is done in but few families, even in those of physicians. The traveller should avoid unboiled water and milk. In communities where sewers exist, sewer connections should be compulsory; if privies are inevitable, they should be water- and fly-tight. Private and public cisterns should be periodically tested. Work camps, country resorts and fair grounds demand careful inspection. The dairies and depots for milk distribution require closest scrutiny.

2. In the *treatment of a developed case* everything with which the germ may come in contact must be sterilized. Next to the feces, the urine is the most important conveyer of the bacillus; these are the usual means of dissemination, and their proper disinfection in every case would obliterate typhoid. The bacilli may persist for years in the feces, five to seventeen years in the bile and five years in the urine. Chloride of lime should be placed in the bottom of the urinal or bed-pan before and after use. Acidulated sublimate (1 to 500) may also be used for the urine and 10 per cent. crude carbolic solution for the stools; but neither disinfects at once, for which reason the dejections should stand an hour in a quart of the disinfectant. Both the pans and urinals must then be sterilized. The same disinfection is indicated for thermometers and rectal tubes. The perineum, after each movement, should be sponged with a 1 to 2000 bichloride solution, and also the back and thighs, when there are involuntary evacuations. The rubber draw-sheets and linen should be sterilized regularly with carbolic acid or bichloride of mercury, and the blankets, pillows and mattresses, steamed when taken off the bed. The bath-tub should be carefully cleaned with carbolic solution, and one-half pound of chloride of lime added to the water after bathing, as the patient's soiled skin and the urine voided during the bath are obvious sources of danger. Cleansing the hands after examination or tubbing prevents direct transmission of the disease to nurses, orderlies and physicians; 2 per cent. of hospital typhoid is acquired by direct contact in the care of the disease. Rubber gloves are the safest protection. In the Spanish-American war 20 per cent. of our soldiers were directly infected by flies, and in the Boer war more died of typhoid than were killed by bullets, infection being carried by flies and sand. The few cases among the Japanese is one of the many lessons they taught us. Flies should be carefully excluded from the house by screens. Quarantine should be enforced.

3. *Typhoid Bacillus-carriers.*—Since 2 to 4 per cent. of typhoid convalescents become chronic carriers, and one person in every 500 well people becomes a carrier (Park), special care in convalescence is indicated. Hexamethylenamine (urotropin) and salol, given in the disease, are helpful, but they are useless later. Segregation, draining the gall-bladder and

washing the urinary bladder are ineffectual. Vaccination (*v. i.*) offers the greatest promise.

4. *Typhoid Vaccination* (Pfeiffer, Kolle and Wright, 1896).—In the Spanish-American war 10,729 troops were quartered at Jacksonville, Florida; among them 2693 probable cases (1729 certain cases) of typhoid developed, with 248 deaths and in contrast with these figures: in our army of 12,659 men during the 1911 maneuvers in Texas, not one typhoid death and but one mild case developed (here vaccination had not been completed). The vaccines are given as follows: the first inoculation of 500, and the second and third each of 1000 million bacilli at ten-day intervals, into the subcutaneous tissue. Local reactive erythema develops with some pain and a small percentage suffer a rigor, generalized erythema, herpes, fever (101° to 102°), vertigo or diarrhea; reaction is less after the second and third injections. An immunity of from under one year to two and a half years is conferred. The agglutinative power of the blood is increased, the opsonic index rises and the white cells increase. Vaccination is indicated in "carriers," soldiers and hospital attendants, as internes, nurses or laundry-women; it is also advisable in children, who leave home for schooling, vacation or travel. Against paratyphoid infections, a similar vaccine is employed. Typhoid vaccines are clearly indicated in prophylaxis, but their value in the developed disease is not established.

(*B*) Hygienic Treatment.—Absolute physical and psychical rest is imperative, and the best results are obtained in cases which have been put to bed early. Ambulatory cases never fare as well. The invariable use of the bed-pan and urinal is insisted upon. The furniture, carpets and hangings should be removed from the room, which should be freely ventilated—fever patients do not catch cold. The nurse should have written instructions. The baths should be supervised by the physician, especially at the onset. The single bed should be low, should have a firm mattress, rubber draw sheet and perfectly smooth cotton or linen sheets. A second bed is desirable for change and convenience.

(*C*) Antiseptic Therapy.—Antiseptic therapy is largely of historical interest, as typhoid is not a local enteric disease but a bacillemia. Calomel given early in typhoid was thought to reduce the virulence of the attack and lessen bowel complications; it is harmful. Salol (phenylis salicylas) in doses of grs. x, t. i. d., is the least objectionable, since it is mild. The urine should be closely watched for signs of renal irritation. Dilute hydrochloric acid, ʒ ss after each feeding, prevents diarrhea and tympany. No one of this group of agents essentially influences the clinical course, and at the most, only decreases fermentation.

(*D*) Antipyresis.—1. *Drugs* are seldom indicated, for they merely reduce fever and do not eliminate its cause, the toxins. Quinine in one or two doses of fifteen to twenty grains has little influence on the fever. Coal-tar products, in full repeated doses, depress the brain, circulation, respiration and metabolism; cause vomiting and eruptions; increase the danger of collapse; and are not indicated in typhoid as they are in the fugitive fevers. In all cases drug antipyresis is far inferior to—

2. *Hydrotherapy,* the only logical febrifuge, since it alone eliminates the fever-exciting toxins.[1] Nature's method of reducing temperature by sweating, evaporation from the moist skin, should be helped by removal of the bedclothing. (*a*) The *full cold bath* of Currie (1787) and Brand is given with water at 64° to 68° F.; it lasts fifteen to twenty minutes and is repeated every three hours for temperature of over 102° or for toxemia. Chilling is averted by vigorous friction, which must not abrade the skin; by cold affusions to the head; or by administration of alcohol. The apparent brutality of the Brand method excited popular and professional prejudice, but it is a most valuable measure. Early resort to hydrotherapy gives the best results. When the portable bath-tub is not available, A. H. Burr uses a wooden frame, shaped like a large box; the base fits over any bed; its upper part sustains a heavy rubber sheet, which passes under the patient and constitutes the sides

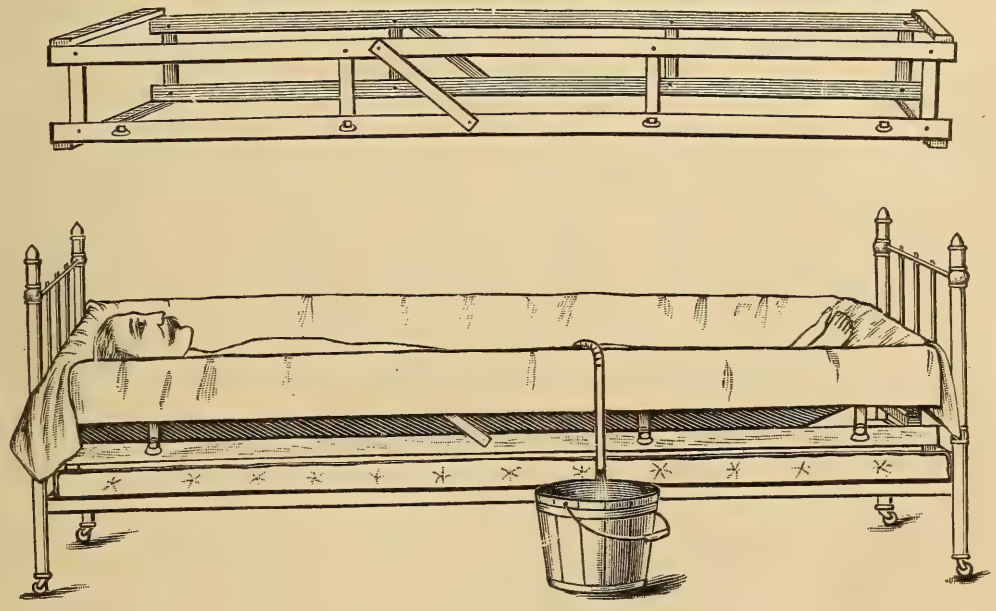


Fig. 6.—Burr's bed-tub.

and bottom of the tub (Fig. 6). Its use obviates the necessity of lifting the patient and saves excitement. Water is poured over the patient from pails and is removed with a siphon. The *advantages* of Brand's method are: (i) The mortality is lowered 6 to 8 per cent.; (ii) toxemia is lessened, and therefore nervous and digestive symptoms, heart weakness, and pulmonary complications are relieved; fever is successfully reduced and there is less visceral degeneration; (iii) renal secretion is stimulated, thereby washing the toxins out of the blood; (iv) the skin becomes more active and bed-sores less frequent; (v) the clinical course is shorter and fewer complications occur. *Contra-indications* to Brand's method are hemorrhage, perforation, great prostration, alcoholism, phlebitis and advanced arteriosclerosis. Pregnancy, nephritis and pneumonia

[1] Toxemia disturbs the heat centre, located in the corpus striatum and connected with various parts of the brain, notably the vasomotor and other vital centres in the medulla. Though views differ, it seems that in fever there is a great increase in the production of heat, without a corresponding increase in its elimination.

are no contra-indications. Various *substitutes* may be indicated for special reasons: (*b*) In the *gradually cooled bath* the temperature of the water is gradually reduced from 90° to 75° or 65°. It is adapted to cases coming under treatment late and to those with tendency to collapse. (*c*) The *protracted tepid bath.* (*d*) *Cold sponging,* first with warm water to dilate the skin vessels and then with cold water; equal parts of alcohol aid evaporation. One part of the body after another is exposed and sponged. It is a refreshing and quieting placebo, but inferior as an antipyretic. (*e*) The *cold pack* is also much less successful. In sthenic cases, ice applications to the head, axilla, spine, etc., may be used, or the body may even be ironed with ice. Ice-water enemas are never used in typhoid.

(*E*) DIET.—The *mouth* and pharynx should be carefully cleansed of sordes and food particles lest infection travel to the ears, salivary or lymphatic glands. Vaseline keeps the lips soft. A boric acid solution, with tincture of myrrh or tincture of hydrastis, meets every requirement. The liquor antisepticus, U. S. P., contains 2 per cent. boric acid and antiseptic oils. The cleansing must be gentle lest fresh abrasions be inflicted. Fifty ounces of *water* should be given daily at definite intervals. It flushes the kidneys, lowers temperature by increasing the sweat production, and lessens parenchymatous degeneration of the viscera. Pure water is superior to barley- or albumin-water, lemonade, etc., which dull the appetite.

In the *diet* no reference is made to individual symptoms, as there is no correspondence between the clinical symptoms and the anatomical ulceration of the intestines. Graves was the first to feed fever patients, who up to that time had been starved, but at the present time there is some risk of overfeeding. *Milk* is the standard diet. Six to eight ounces should be sipped at intervals of three hours, day and night, as the patient, after being wakened, readily falls to sleep again. It may be diluted with half an ounce of lime-water or a little carbonated water, which should not be cold, since the gas expands at body heat. The physician himself should inspect the stools for curds. As in infant feeding, curds indicate reduction of the cream. Very few patients cannot take milk. Recalcitrant patients may be given koumyss, matzoon, buttermilk, whey or certain baby foods. If digestion is weak, *peptonized milk* may be given with thin, strained gruels or egg. Carbohydrates are indicated to lessen nitrogenous waste (Kendall), and well-boiled cornstarch, strained vegetable soups and tapioca are allowed if no tympany results; taka-diastase aids their assimilation. Meat-juice is obtained from freshly slaughtered beef, by a meat-press. It may also be given frozen (meat-juice, ℥ij; sugar, ℥ij; lemon-juice, ℥j; yolk of one egg; cognac, ℥j). Yolk of egg in soup or wine, and white of egg with twice its weight of water afford some variety. Gelatin conserves metabolism but may irritate the bowels. Beef-tea contains only stimulating substances, less than 1 per cent. albumin, and no fat or carbohydrates and may irritate the kidneys and intestines. In stupid patients with anesthetic throats, food may enter the larynx and cause aspiration pneumonia. In these cases the nasal catheter is indicated. To its outer end a funnel is attached through which food, drink, and medicines are given. A safety-pin across its outer end prevents its escape downward. The catheter should be long enough to pass into the esophagus beyond the larynx. Rectal feeding is sometimes indicated.

High Calory Feeding.—Years ago Shattuck, Hare, von Müller and others advocated more generous feeding, and lately Coleman and Schaffer, especially, have popularized high calorie feeding. At the height of the infection, the pyrexia (fever and toxemia), the alimentary disturbance and the starvation increase metabolism about 34 per cent., as regards the protein and fat of the body, somewhat as in simple starvation, although more extensively. Magnus-Levy demonstrated that metabolism is qualitatively the same in the febrile as in the well individual. To compensate the increased nitrogenous waste, a diet more ample than is usually given, is desirable. Coleman and Schaffer hold that theoretically 5000 calories are demanded; 3000 are accepted as a compromise. The approximate values of available foods in calories are as follows: One quart milk, 700 (plus 6 per cent. milk-sugar, 900); 1 pint cream, 900; 5 ounces oatmeal or hominy, with 2 ounces cream and 3 teaspoonfuls sugar, 300; eggs, each 80; cup of custard, 100; 1 slice of well-buttered bread, 200; 8 ounces milk-sugar, 1000. Absorption in fever is 90 per cent. of normal. The protein and fat loss is about 6 per cent. each, whereas carbohydrates are perfectly assimilated. Two quarts of milk give but 1400 calories of the 3000 or more needed. Coleman believes that the ampler diet decreases indigestion. Eggs supply *protein* (6 eggs=7 gm. N.=480 calories) and are of higher food value in a mixed diet; Coleman discards meat, except for its extractives in soup to stimulate appetite, though v. Müller advocates brains, hash, etc.; 100 gm. protein (=16 gm. N.) is the upper limit. *Carbohydrates* should constitute half the calories and are supplied by bread, toast, crackers, mashed potatoes, milk-sugar, and gruels (oatmeal and cream of wheat); cellulose is shunned (in vegetables and fruits). *Fats* include cream, butter and cocoa; in excess they disturb digestion, but as much as 200 to 250 gm. are sometimes tolerated. If the milk-sugar is too sweet in milk, it may be taken in lemonade, ice-cream or custard. In high feeding, two to four bowel movements a day are usual. In every instance this diet must be individualized, regarding the weight, appetite and digestion, and increasing slowly. It is difficult to give 3000 calories in the severest types, who most need the diet. Appended are caloric values, borrowed from White.

DAILY FOOD DEMANDS.

Adult.	Body weight.	Calories per pound.	Total calories.	Total grams proteid.
At rest in bed	150 lbs.	12	1800	72
Slight activity	"	15	2200	88
Light work	"	17	2600	115
Moderately hard work	"	20	3000	120
Very hard work	"	23–30	3500–4500	140–180
Child.				
Age 0–6 months	7–15 lbs.	42–40	300–600	1 gram per lb.
6–12 "	15–20 "	40	500–800	35–40
2 years	25 "	36	900	42
4 "	35 "	34	1200	55
8 "	50 "	28	1400	60
12 "	75 "	22	1600	75

	Calories.
1 gram proteid	= 4.1
1 " carbohydrate	= 4.1
1 " fat	= 9.3
1 " alcohol	= 7.0

FOOD VALUES IN HOUSEHOLD MEASURES.

Foods as eaten.	Actual amount.	Household measure.	Approximate.			
			Calories.	Proteid, grams.	Fat, grams.	Carbohydrate, grams.
DAIRY:						
Milk	8 ozs.	A glass	160	7.5	9.5	12.0
Skimmed milk and buttermilk	8 ozs.	A glass	80	7.5	1.0	11.5
Cream {thin (20 per cent.)	16 gms.	A tablespoon	30	0.5	3.0	0.5
Cream {thick (40 per cent.)		A tablespoon	60	0.5	6.0	0.5
Condensed milk {sweetened	20 gms.	A heaping teaspoon	70	2.0	2.0	11.5
Condensed milk {unsweetened		A heaping teaspoon	35	2.0	2.0	2.5
Butter	10 gms.	A pat or ball	80	....	8.5	
Cheese {Cream		One-inch cube	65	4.0	5.0	0.5
Cheese {Skim milk	15 gms.	One-inch cube	45	4.5	2.5	0.5
Cheese {American		One-inch cube	70	4.0	5.5	
Eggs, whole	50 gms.	One	75	6.5	5.0	
Eggs, yolk	15 gms.	One	55	2.5	5.0	
MEAT AND FISH (cooked):						
Beef-tea, clear soups	5 ozs.	A teacup	5–20	1–4.5	....	0.5
Fish {lean (cod, flounder)	50 gms.	A heaping tablespoon	35	8.5		
Fish {fat (shad, salmon)		A heaping tablespoon	105	11.0	6.5	
Meat {lean		A medium slice	70	11.5	2.5	
Meat {medium fat	50 gms.	5 x 3 x ¼ inch	150	11.5	9.0	
Meat {fat			200	8.5	18.0	
Oysters, medium size (raw)	16 gms.	One	8	1.0	0.2	0.5
CEREALS AND VEGETABLES (cooked)						
Bread, white or Graham	25 gms.	One slice, 4 x 4 x ½ inch	70	2.3	0.5	13.0
Vienna roll	40 gms.	One	115	3.5	1.0	22.5
Crackers (Uneeda)	7 gms.	One	30	0.5	0.5	5.0
Cereals, cooked, moist	40 gms.	A heaping tablespoon	35	1.0	....	7.0
Cereals, eaten dry	5 gms.	A heaping tablespoon	20	0.3	....	4.0
Shredded wheat	30 gms.	One	110	3.0	0.5	23.0
Gruels (cereal)	8 ozs.	A soup plate	75	2.5	1.0	14.0
Thickened or cream soups	8 ozs.	A soup plate	160	5.5	4.5	24.0
Macaroni	25 gms.	A heaping tablespoon	25	1.0	0.5	4.0
Potato, boiled or baked	95 gms.	One medium size	90	2.0	....	20.0
Potato, mashed	35 gms.	A heaping tablespoon	40	1.0	1.0	6.0
Rice, boiled	30 gms.	A heaping tablespoon	35	1.0	....	7.0
Corn, canned	35 gms.	A heaping tablespoon	35	1.0	0.5	6.5
Peas, fresh	35 gms.	A heaping tablespoon	40	2.5	1.0	5.0
Lima beans, canned	25 gms.	A heaping tablespoon	20	1.0	....	3.5
Squash	35 gms.	A heaping tablespoon	20	0.5	....	3.5
FRUITS:						
Apple, pear	120 gms.	One medium size	75	0.5	0.5	17.0
Apple sauce	45 gms.	A heaping tablespoon	70	....	0.5	16.5
Banana	100 gms.	One medium size	100	1.5	0.5	22.0
Orange	130 gms.	One medium size	70	1.0	....	15.0
Strawberries	100 gms.	A medium saucerful	40	1.0	0.5	7.5
Dried figs, dates, raisins	100 gms.	A medium saucerful	350	2.5	3.0	76.0
Fruit jelly, sweetened	50 gms.	A heaping tablespoon	160	0.5	....	38.5
DESSERTS:						
Custard	40 gms.	A heaping tablespoon	55	2.5	0.5	9.0
Ice-cream	40 gms.	A heaping tablespoon	135	1.5	9.0	11.0
Sponge cake	20 gms.	A slice 2 x 4 x ½ inch	75	1.5	2.0	13.0
Pudding (rice, tapioca, bread)	45 gms.	A heaping tablespoon	80	2.0	2.0	13.0
ALCOHOL	12 gms.	A tablespoon	85			
Whisky, brandy, etc. (50 p. c.)	1 oz.	A small wineglass	85			
Wines (8–45 per cent.)	1 oz.	A small wineglass	15–50			
MISCELLANEOUS:						
Sugar	8 gms.	A heaping teaspoon	33	....	....	8.0
Honey	10 gms.	A heaping teaspoon	33	....	....	8.0
Olive oil	4 gms.	A teaspoon	37	....	4.0	
Olives	7 gms.	One medium size	15	....	1.5	0.5
Almonds, shelled	25 gms.	A heaping tablespoon	165	5.0	13.5	4.5
Cocoa powders	10 gms.	A heaping teaspoon	50	2.0	3.0	3.5

(*F*) SYMPTOMATIC OR EXPECTANT TREATMENT.—1. *Digestive System.* (*a*) *Vomiting.*—It must be ascertained that the patient has received no solid food from friends and that perforation has not taken place. Rectal feeding, warm carbonated water, cocaine gr. ¼, or tincture iodine ♏½, for several doses, sinapisms or an ice-bag to the epigastrium, are indicated.

℞—Phenolis . . . . . gr. iv
Bismuthi subnitratis . . . . . ʒiss
Sacchari albi } . . . . . āā q. s.
Acaciæ }
Aquæ menthæ piperitæ . . . . . q. s. ad. ʒij
M. et fac emulsum.
S.—One teaspoonful, undiluted, every fifteen to thirty minutes.

(*b*) *Constipation* indicates enemas of water or oil every other day. Typhoid cases with constipation fare better than those with diarrhea, and cathartics cause meteorism.

(*c*) *Diarrhea* is most frequently due to toxemia and is therefore most efficaciously treated with the cold bath; curds (prevented by diluting or skimming the milk), beef-tea or impure milk, may be its cause. Treatment is indicated by more than four movements daily, colic, tenesmus, unrest or weakness. Cold tea with whisky and pulv. aromaticus, gr. xv; or paregoric and bismuth, each ʒj after each movement; extr. opii., gr. i; or the starch-and-laudanum enema usually give relief at once.

℞—Bismuthi subnitratis . . . . . ʒiij
Extracti opii . . . . . gr. ij
M. et div. in pulv. xij.
S.—One to four a day. (Since absorption from the alimentary tract is slow and uncertain, the effects of powerful drugs given by mouth should be watched carefully.)

℞—Acidi tannici . . . . . ʒj
Tincturæ opii . . . . . ♏xl
Emulsi terebinthinæ . . . . . q. s. ad. ʒj
M. et S.—One teaspoonful every three hours for 3 or 4 doses.

℞—Plumbi acetatis . . . . . gr. iss ad iij
Opii pulveris . . . . . gr. i ad iiss
Sacchari albi . . . . . gr. lx
M. et divide in pulv. vj.
S.—A powder every three hours (note caution under first formula).

(*d*) *Tympany.*—In severe cases albumin-water must be substituted for milk. The cold bath often gives relief, since meteorism is generally toxemic. Hydrochloric acid after feeding may lessen the tympany. The turpentine stupe of Jenner should be applied to the abdomen, flannel cloths being dipped in warm water and ʒss ol. terebinthinæ and slapped hard together to spread the oil evenly and thus avoid blistering. The ice-coil is also beneficial. The old turpentine emulsion (emulsum olei terebinthinæ, ʒj) is excellent, though the urine must be watched for renal irritation. Whisky, spices, and perhaps eserine sulphate or salicylate gr. $\frac{1}{50}$ are effective. Emulsum asafetidæ, Oj; spiritus chloroformi, ʒij; and ol. terebinthinæ, ʒss, as a warm enema, often give relief.

(*e*) *Hemorrhage* necessitates absolute quiet, mental, physical and alimentary. The patient should not be moved for any reason. In severe cases he should lie in the voided blood, with cotton tucked around the hips and between the legs to catch the urine and feces, which should pass into it without his being lifted on the bed-pan. No food, no water, no enema, no bath, should be given. Morphine quiets the excitement attending hemorrhage, lessens peristalsis and enforces bodily quiet; gr. $\frac{1}{4}$ is injected every hour for three doses, or enough to produce its

full physiological effect. The sole objection to morphine is that it may mask coincident perforation, which occurs once in every five cases of hemorrhage; close circumspection invalidates this objection. Opium or styptics by the mouth are dangerous and uncertain. The lead- or iron-salts probably never reach the bowel. An ice-coil on the abdomen may be beneficial. Gelatin may be injected as in aneurysm, but 10 c.c. of horse serum or diphtheria antitoxin work better results; the latter should be fresh and not of the concentrated type, which largely contains globulins. Schreiber employs an intravenous infusion of 5 to 10 per cent. grape-sugar solution. The question of stimulation is perplexing. There is the risk of forcing blood through the leak by stimulation; on the other hand, the volume of blood must not sink too low; but first the heart must be allowed to run down—nature's most certain method of hemostasis. At a later time only, should strychnine or digitalis be administered hypodermically, or saline transfusions be given; delay in their use is better than haste.

(*f*) *Perforation* necessitates an early diagnosis and immediate operation. Most reported recoveries were operated on in the first twenty-four hours. Recovery occurred in 30 per cent. of 358 operations (Cazin). In twenty cases operated on at Johns Hopkins Hospital, 35 per cent. recovered.

(*g*) Quénu tabulates 67 operations on the *gall-bladder;* 82 per cent. of recoveries occurred in the cholecystostomies and 80 per cent. in the cholecystectomies.

2. *Circulatory System.*—Heart weakness is best prevented by absolute rest and hydrotherapy. An ice-bag placed over the heart tends to regulate and calm its action. *Strychnine* is the best vasomotor tonic; gr. $\frac{1}{30}$ may be given three times a day in cases which do not show signs of circulatory failure, but it is best to await definite indications for stimulation. Digitalis is rarely beneficial in fever cases, and may produce signs of cumulative action; the author saw a case in which, with sudden defervescence, the pulse dropped from 140 to 40. Camphor, gr. ij, should be given under the skin in 1 to 10 parts of sterilized olive oil. Other cardiants are considered under Valvular Disease and Pneumonia. In typhoid *alcohol* may be indicated (*a*) as a food, with milk and eggs, thus aiding digestion; ℥j–iv of whisky may be given every two to four hours in hot milk. Stokes's mixture contained cognac, ℥ij—ovi vitellus 1, syr. aurantii ℥v, and water q. s. ad. ℥v. (*b*) As a cardiant (though pharmacologists hold that pure alcohol is not a cardiant); Meltzer states that it equalizes the circulation. Cognac may be given by the mouth, in coffee; or by rectum, in peptonized milk with two drachms of tincture of valerian. (*c*) As a nervine in debility or old age, as whisky, champagne (especially for vomiting), sherry or port. (*d*) To a less degree as an antipyretic, since it increases the sweat production. Its use must be suspended when the pulse becomes bounding, the respiration hurried, the skin or tongue dry, when there is restlessness, or the odor of alcohol appears on the breath. Alcohol has been abused, and the recent reaction against its use is in part sound. With discretion alcohol is invaluable in severe cases and in alcoholics.

Thrombophlebitis is treated by quiet, elevation, warmth and bandaging from the toes upward.

3. *Respiratory System.*—Epistaxis usually ceases spontaneously. If the blood runs into the throat a tampon should be introduced. Laryngeal complications may necessitate early tracheotomy; only 2 per cent. of severe complications recover without operation. Bronchitis is treated by hydrotherapy, rarely by codeine. Expectorants are avoided since they disorder digestion. Hypostasis is best treated by hydrotherapy, cardiants and explicit written orders for change of posture every two hours.

4. *Nervous System.*—The initial headache may be relieved by hydrotherapy, the ice-cap, ergotin and acetphenetidinum (phenacetin), each gr. v (in extreme cases lumbar puncture may be considered). Depression is relieved by valerian and whisky. For delirium and insomnia, hydrotherapy is the most useful measure; veronal, gr. v–vij, produces a natural sleep; alcohol; morphine, gr. $\frac{1}{4}$; codeine, gr. $\frac{1}{2}$; heroine, gr. $\frac{1}{21}$; sodium bromide, ℥ss–j; or hyoscinæ hydrobromidum, gr. $\frac{1}{120}$ may be indicated.

℞—Morphinæ hydrochloridi . . . . . gr. $\frac{1}{4}$
Sodii bromidi . . . . . gr. xxx
Syrupi aurantii . . . . . q. s. ad. ℥iij
M. et S.—To be taken at bed-time in water.

Typhoid patients should never be left alone, lest they leave the bed or leap from the window. Restlessness may suggest a full bladder.

5. *The Skin.*—Massage with alcohol or olive oil is useful. The skin functions are helped by the full bath. In the formation of bed-sores, local heat, pressure, moisture, maceration and filth are factors. These are generally avoided by frequent change of posture, thick pads or rubber rings and cleanliness. The least erythema, papule, abrasion or pustule on dependent parts is a matter of solicitude; skin infections may cause ward epidemics (Edsall). If bed-sores develop, the simplest treatment is the best, since antiseptics and powders may aggravate. Balsam of Peru is excellent. Scarlet-red ointment (8 per cent.) is often curative; it should not be used in deep ulcerations, as poisoning may occur—faintness, collapse, cramps, etc., as in anilin poisoning. Collodion and adhesive straps are always to be avoided.

6. *Bacilluria.*—Bacilluria is met by phenylis salicylas and hexamethylenamina, each gr. x, t. i. d. Urotropin operates best when the urine is acid or made so, by acid sodium phosphate; urotropin may induce alimentary or vesical irritation and possibly albuminuria. If bacilluria persists, irrigations with silver nitrate or bichloride solutions are indicated. Special care is necessary in the use of catheters.

(*G*) Treatment in Convalescence.—The convalescence must be actual and relapse excluded before the patient can have substantial food. It is best to err on the safe side, as embolism and collapse are not unknown in convalescence. The patient should not leave his bed for two weeks. Irregular fever lingers in some cases until solid food is given

and the patient sits up. In other cases, meat causes a higher temperature (*febris carnis*). An excellent tonic is the following:

℞—Strychninæ sulphatis . . . . . gr. j
Acidi nitrohydrochlorici . . . . . ʒj
Tr. gentianæ co. . . . . q. s. ad. ℥v
M. et S.—One teaspoonful after meals in half a glass of water.

ERYSIPELAS.

Synonym.—St. Anthony's Fire.

Definition.—An acute, febrile, contagion caused by Fehleisen's *Streptococcus erysipelatis;* characterized by a circumscribed inflammation of the skin and adjacent mucosæ, which tends to rapid and broad extension; and self-limited in its course.

History.—Erysipelas was known to Hippocrates and Galen. Trousseau (1848), in France, held it was a wound infection; Werner, in England, maintained that it was infectious; and Koch (1880) found the streptococcus which was more fully described by Fehleisen (1881) and bears his name.

Etiology.—Sporadic cases are seen in every centre of population. It occasionally breaks out epidemically. Sixty per cent. of cases occur between January and May. In the pre-antiseptic era it was the dread of hospital physicians, and even at this day it occasionally visits the most sanitary institutions. It is conveyed by a third person, fomites and, infrequently, by unclean instruments and vaccine. Cases of placental transmission to the fetus are known. The virus is not penetrative but adheres to clothing and furniture with great tenacity. Many persons exhibit a natural, perhaps an hereditary, susceptibility. In its recurrence, it resembles rheumatism. It occurs mostly in the female sex (73 per cent. of cases). Alcoholism, debility and chronic nephritis are predisposing factors. Any distinction between "idiopathic" and "*wound*" *erysipelas* is artificial, for in every instance, some abrasion, perhaps too small for the eye to detect, occurs in the skin or mucosæ.

Bacteriology.—The Streptococcus erysipelatos was found in the lymph vessels by Fehleisen, who first drew the distinction between erysipelas and phlegmon. Its absolute differentiation from the Streptococcus pyogenes is impracticable. Inoculations from cases of erysipelas sometimes produce suppuration, pus has caused erysipelas and erysipelas and suppuration have developed simultaneously, which facts give weight to the theory of the bacteriological unity of erysipelas and suppuration.

Symptoms.—1. INCUBATION lasts three to seven days.

2. ERUPTION.—(*a*) Usually preceded by a few hours of general discomfort, this stage begins with *vomiting, fever, chill and the eruption.* The fever rises rapidly to 103°. The chill occurs in 75 per cent. of cases and is chiefly absent in erysipelas secondary to other diseases. (*b*) The *eruption* first appears on the face (95 per cent.), near the inner canthus, nose or cheek. It consists of a hard, bright red, slightly elevated, and painful area which pits but little on pressure, though its color may disappear. Pathologically, erysipelas (literally, red skin) is simple inflammation; in the skin are found edema, hyperemia, white- and red-

cell extravasation, and *streptococci* which advance *in the lymph clefts* and are most abundant in the spreading edge, and in the lymph vessels beyond it. *Its elevated red edge* is sharply marked or "*wall-like.*" As the area spreads, the *skin becomes much swollen, edematous, smooth, tense, red and hot,* causing a shapeless swelling of the face and obliterating its lines and expression. The extent is indicated more accurately by tenderness than with the eye. The *skin blisters* and under the lens small vesicles are always found. The *prong-like advance of the eruption* is similar to that of spilled alcohol or burning paper, *i. e.*, it disappears behind as it advances forward. Its extension is irregular, because the streptococci follow the lines of least resistance. If obstructed by dense tissue, deep attachments or the so-called ligamenta cutis, its course becomes circuitous, asymmetrical and tardy in one area, though rapid in another. It rarely affects the chin, or passes over it to the opposite side. The neck resists its advance, often successfully, but erysipelas may course over the entire body. The sternum, condyles, ilium and Poupart's ligament check or divert its progress. When apparently obstructed, new islets of eruption appear near by, which may be seen to be consecutive by the faint lymphatic involvement between the foci. The scalp is often unaffected, but if invaded it becomes swollen, and pale or blue rather than red, and the hair falls out. The first area pales, and a coarse desquamation results, while the eruption at more remote points advances. The lymph glands are constantly swollen, tender and palpable, unless hidden by edema. They enlarge proportionately to the dermatitis. The eruption presents few variations: *Erysipelas vesiculosum; E. bullosum; E. squamosum,* or, in old or cachectic subjects, *E. hemorrhagicum.* (*c*) The *fever* rises suddenly, and is 103° to 104° by the fourth day and continuous, then remittent, and after seven to nine days resolves by crisis. In attenuated erysipelas there is little fever and few general symptoms, though the rash may be well developed. In general the intensity and progress of the infection are marked by the degree of fever. The pulse is soft and ranges between 100 and 120.

Course.—When limited to the face and head its usual duration is eight to ten days, rarely longer than two weeks. *Erysipelas migrans* may travel from limb to limb, and recur repeatedly. In one of the author's cases it lasted six months.

Complications and Sequels.—1. Nervous.—Toxemic symptoms are infrequent except in aged, debilitated or alcoholic subjects. Delirium is an unfavorable toxemic development and occurs especially when the scalp is invaded. Meningitis, formerly confused with toxemia, infrequently follows orbital cellulitis or extension along the fifth nerve. Postfebrile psychoses, multiple neuritis, degeneration of the cord and eye inflammation are exceptional.

2. Circulatory.—Endocarditis, pericarditis, myocarditis, acute dilatation, and arterial or venous thrombosis are infrequent. High leukocyte counts indicate concomitant suppuration. Streptococci in the blood are usually ominous.

3. Respiratory.—Erysipelatous coryza and otitis are infrequent and difficult of recognition. Erysipelatous angina is characterized by

much pain, sharply marked lividity, a glazed, varnished surface, marked edema of the uvula and epiglottis, the formation of vesicles or bullæ, and sometimes a wall-like margin. Gangrene may develop in severe infections. The cervical glands may be swollen. Laryngeal, secondary to pharyngeal erysipelas, is rare, severe, easily or necessarily confused with glottis edema, and marked by great turgescence and rapid swelling. Without tracheotomy, fatal stenosis and extension to the lower air-passages may occur. The rare so-called "erysipelas pneumonia" is probably a septic localization; in streptococcus pneumonia, the spleen is enlarged more frequently than in ordinary pneumonia. The advance is rapid, the extension wide, the gray hepatization great, and fibrin formation scant or lacking. In pneumococcic infection the same "wandering pneumonia" may be seen. Pleurisy is usually metastatic

4. Alimentary.—Vomiting is toxemic. Exceptional complications are parotitis, esophagitis, gastritis, enteritis, intestinal ulceration, hemorrhage, pain, icterus and pylethrombosis.

5. Genito-urinary.—Albuminuria is the most constant visceral finding (50 per cent.). Nephritis (4 per cent.) is often of the acute glomerular type. Streptococci have been found in the urine and kidneys, especially in septic types. Erysipelas may begin in the external genitalia, especially in *puerperæ* (page 22).

6. Septic.—Subcutaneous *abscesses*, which resemble cold abscesses, appear in 6 per cent. of cases, especially in alcoholic, nephritic, diabetic, aged or debilitated patients. Phlegmons may develop, usually on the legs. Gangrene is fortunately rare ($\frac{1}{7}$ of 1 per cent.); it develops early over the eyelids, genitalia or joints, or later in the extremities. Herpes is not infrequent. Baldness may also result. The joints may be painful; suppurative arthritis or adenitis is metastatic. Generalized fatal sepsis, aside from the puerperal form, is infrequent. It bears the same relation to local erysipelas as does miliary tuberculosis to a local tuberculous focus.

Diagnosis.—Diagnosis is easy, when the area is the size of a dollar, (*a*) because of its very location; (*b*) the early involvement of the lymphatics; (*c*) often the atrium can be found; (*d*) the redness, smoothness, swelling and edema; (*e*) the elevated, wall-like margin; and (*f*) the prong-like, irregular progression.

In *weeping eczema*, the edges are not raised, the skin is not smooth, there is less fever, the development is slower and itching is present. *Malignant pustule* causes redness and swelling, but anthrax bacilli are found under the primary papule and crust. *Dermatitis venenata* or *medicamentosa*, and pemphigus, are unlike erysipelas. Erythema nodosum is discrete and does not spread. Diffuse simple erythema is a febrile affection, and of short duration. *Dermatitis exfoliativa* in the newborn is afebrile, develops first about the mouth and chin, and reaches the body and limbs later. Its diffuseness and redness are characteristic. In *lymphangitis* the swelling of the lymphatics advances centripetally and presents cord-like areas, which are not confluent, vesiculated, uniformly red or evenly edged. In *phlegmon* and deep *cellulitis*, the process is deeper and harder, and the blisters and margin of erysipelas are lacking.

Erysipelatous angina may not be diagnosed without coincident facial erysipelas. The local findings are not pathognomonic.

Erysipeloid, a zoönotic affection described first by Rosenbach (1884), occurs especially in persons who handle meat; the bacillus is identical with that of mouse sepsis and hog cholera. It is recognized by the "butterfly" eruption on the cheeks or finger-ends, and is unaccompanied by fever or general symptoms. The edge of the eruption is clear-cut, its advancement slow, its color a dark livid-red, and its course lasts one to three weeks with spontaneous recovery. Gilchrist reported 329 cases, of which 323 were caused by crab-bites.

Prognosis.—(*a*) In uncomplicated *facial erysipelas*, the mortality is 3 per cent. in private practice, and double that figure in hospitals. (*b*) In *erysipelas migrans* the outlook is less favorable. (*c*) *Erysipelas neonatorum* is fatal within a week. It begins on the navel, and less often on the vulva or circumcision wounds. Vomiting, diarrhea, peritonitis, icterus, pneumonia, necrosis, gangrene, suppuration and sepsis are much more common than in adult forms. Fortunately, like puerperal fever, this form is now infrequent. (*d*) *Erysipelas secondary* to other infections is serious, especially in chronic maladies, such as alcoholism, diabetes, nephritis and tuberculosis, and also in bed-ridden or senile subjects, in whom the asthenic, hemorrhagic and gangrenous types are chiefly observed. In acute diseases such as typhoid, diphtheria and pneumonia, erysipelas is dreaded. (*e*) Puerperal forms or involvement of the larynx are unfavorable (Hippocrates). (*f*) *Recurrence* is more frequent than in any other infection, and no permanent immunity is conferred. Relapses (10 per cent.) are attacks recurring within a few days. Recurrence (35 per cent.) suggests the possibility of a bacterial latency and develops after months or years; in some individuals, erysipelas may be called habitual. It is observed most frequently in the young and in women (75 per cent.), and mostly on the face. Five to fifteen attacks are not uncommon, and Roger saw forty in one person.

Treatment.—1. Prophylaxis.—In this bacteriological era, the disease has decreased by asepsis and isolation, though in the latter regard many physicians are still remiss. Chronic rhinitis, rhagades, decayed teeth and neglected ulcers, must be considered in habitual erysipelas. (See Smallpox; Disinfection.)

2. Mechanical Means.—Mechanical efforts to limit extension of the disease are always futile. These consist of binding with elastic strips, elastic constriction or massive applications of collodion.

3. Local Applications.—Iodine, creolin, bichloride of mercury, magnesium sulphate (saturated solution), phenol, alcohol, iodoform, boric acid and other topical applications, are recommended, but the water they contain is their most potent ingredient, and was recommended by Hippocrates. No application can reach the deeply situated cocci. Cold compresses afford a certain relief for pain and tension. Ichthyol ointment, 10 per cent., is soothing but not curative.

℞—Creolin	1 part
Iodoformi	4 parts
Adipis lanæ hydrosi (lanolin)	10 parts

M. et f. ung. (Koch.)

4. Drugs.—Erysipelas is a self-limited disease. Iron (Hamilton Bell, 1851) is well tolerated in large doses, but neither shortens nor mitigates the disease.

℞—Tincturæ ferri chloridi . . . . . ʒiiss
Quininæ sulphatis . . . . . gr. xxiv
Spiritus chloroformi . . . . . ʒij
Aquæ . . . . . q. s. ad. ℥vj
M. et f. mist.
S.—Two tablespoonfuls every three or four hours. (J. Burney Yeo.)

5. General Treatment.—The symptoms are managed expectantly, *i. e.*, as they arise. Headache, delirium, pain, vomiting and cardiac weakness are treated as in typhoid. *Rest in bed* and as *generous nourishment* as the stomach will tolerate are indicated.

6. Serum.—Chantemesse reduced the death-rate to 2.5 per cent. by serum treatment (a figure often reached by more simple measures). Vaccines have not proven successful.

Erysipelas as a *curative agent* (*erysipelas salutare*): Permanent cure of lupus has followed a complicating erysipelas. Syphilitic manifestations quickly subside after this infection, and its beneficial effects have been observed in leukemia, Hodgkin's disease, carcinoma, sarcoma, chronic arthritis, tuberculous adenitis, diphtheria and tuberculosis.

PNEUMONIA.

Synonyms.—Fibrinous, croupous or lobar pneumonia, pleuropneumonia, pneumonitis, lung fever.

Definition.—An acute, specific, self-limited infection caused by the pneumococcus, characterized by fibrinous inflammation of the lung, toxemia, pneumococcemia, and solution of the fever by crisis.

Frequency.—Pneumonia constitutes 6.5 per cent. of all internal diseases, 4 per cent. of all diseases, 6.6 per cent. of all medical deaths, and 8 per cent. of all deaths.

Bacteriology.—1. The *pneumococcus* or Diplococcus pneumoniæ (Plate II) was recognized by Fränkel (1884), and Weichselbaum (1886) first demonstrated its frequency and importance. It is a lanceolate, encapsulated diplococcus; it is easily seen in cover slips, it readily stains by the Gram method, and presents many cultural variations. Its *atrium* in pneumonia is *the respiratory tract*, although it may enter the blood by other avenues in sepsis (*q. v.*). From the lung it may spread diffusely, chiefly through the bloodvessels. It is found in dust, saliva, and in 30 per cent. of healthy noses and throats, though usually with attenuated virulence. Experimental inoculation has produced pericarditis, endocarditis and empyema. In lobar pneumonia or apart from this acute bacteriemia the pneumococcus has been found in the blood, joints, brain, bone-marrow, etc.; in otitis media, endocarditis, peritonitis, cholangitis and endometritis; in the urine and feces; in the nasal sinuses; and it may pass from the mother to the fetus. It may be a terminal septic infection. Until recently it was thought that typhoid and pneumonia were local infections and that bacteriemia was a serious complication;

PLATE II

The Diplococcus Pneumoniæ, Stained With Methylene Blue and Fuchsin as a Counterstain. Taken from the Sputum of a Case of Acute Croupous Pneumonia. (Simon.)

the typhoid bacillus is found in the blood in 100 per cent. of early typhoids and the pneumococcus in from 90 to 100 per cent. of pneumonics (*v. i.*). It is assumed that a *pneumotoxin* exists producing the toxemia; when an antitoxin develops, the crisis occurs, but the factors of resistance to, and recovery from, pneumonia are not known. *Immunity* does not result from one attack; recurrence is likely in 23 per cent.; ten, even twenty-eight recurrences are known. The pneumococcus is the sole cause of typical pneumonia, but other microörganisms are sometimes found, as the pneumobacillus (Friedländer), and the typhoid, colon, diphtheria, proteus, influenza, plague, and pyogenic organisms.

Indirect Causes.—(*a*) *Age.*—Pneumonia may develop even in the newborn. Predisposition to infection is great up to the sixth year, then much less until the fifteenth year, when its frequency increases with each decade. Over half the cases of pneumonia occur between the twentieth and fortieth years, and over 80 per cent. between the tenth and fiftieth years.

(*b*) *Sex.*—Eighty per cent. of cases are males. The relation to trauma is in doubt.

(*c*) *Cold.*—Three-quarters of all cases of pneumonia occur in cold months (February, March and April) when the weather varies, the temperature is low, the moisture is great and the winds are high. Dust disseminates the dried sputum. Pneumonia is half again as frequent in the cities as in the country. In the winter of 1903–4, when 4000 died of pneumonia in Chicago, practitioners in towns not one hundred miles distant did not see a case.

(*d*) *Individual predisposition* is a *more important factor than in any other infection.* Susceptibility is increased by debility, overwork and alcoholism.

General Clinical Course.—After a *short incubation* of some hours the *disease begins suddenly.* In infants it often begins with convulsions—in the young with vomiting, and in older subjects with a severe chill, often brusquely in persons of robust health. On the *first day* there is a sudden rise of temperature; the face becomes red, even livid; the eyes are injected; the pulse and respiration are accelerated; the patient lies characteristically flat in bed; there is much depression; headache and sudden sharp pain in the side develop, accompanied by cough. On the *second day* the cough, pain, respiration-rate and headache increase; the expiration is short and grunting, with dilatation of the alæ nasi, and speaking is difficult; leukocytosis develops. The bloody sputum appears, which with the above symptoms establishes the diagnosis before consolidation appears. On the second or *third day* there are evidences of lung consolidation, such as tympany, soon followed by dulness, the crepitant rale, and bronchial breathing; there is an increase in the pulse and respiration; herpes, anorexia, coated tongue, constipation, moderate delirium and febrile urine develop. The coughing is frequent but short in duration, voids a viscid red sputum, and causes catching respiration, pain and broken sleep. The clinical course may be unfavorably modified (*a*) especially by collapse due to myocardial or vosomotor paralysis, or to pulmonary embolism; (*b*) by profound toxemia; (*c*)

by delirium tremens; or (*d*) by death, though at this stage it is rare. In favorable cases, when the toxemia is spent, the crisis occurs, usually on the fifth, seventh or ninth day with gradual disappearance of the local findings. A "critical" fall of temperature, pulse- and respiration-rate, sweating and relief of nervous symptoms result. Lysis is less frequent. *Toxemia is the dominant feature* of pneumonia, and the consolidation is secondary. The degree of toxemia has no constant relation to the amount of consolidation, just as the toxemia of typhoid does not depend on the presence or degree of the intestinal changes.

Analysis of Individual Symptoms.—1. ONSET.—In over 75 per cent. *the onset of pneumonia is sudden. Chill,* convulsions, vomiting and *fever,* after a short incubation, *usually mark the brusque onset.* Repeated chilly sensations do not as accurately designate the onset as does an initial rigor. In no other acute disease except malaria is the chill so severe and constant (in 75 per cent.). The *onset is atypical* in senile pneumonia and in pneumonia secondary to other diseases —*e. g.,* delirium tremens, typhoid and psychoses—the chill, cough, dyspnea, pain and sputum are often absent, so that the existence of pneumonia may not be suspected. *Prodromes* occur in 25 per cent. of cases, mostly in men between thirty and forty years, and rarely last over five days; depression, headache, bronchitis, backache and low fever may be noticed. In one of the author's cases a pneumococcus infection began in the throat, then invaded the larynx, and only after three weeks invaded the lung. In four cases, low fever and hemoptysis for several days suggested tuberculosis.

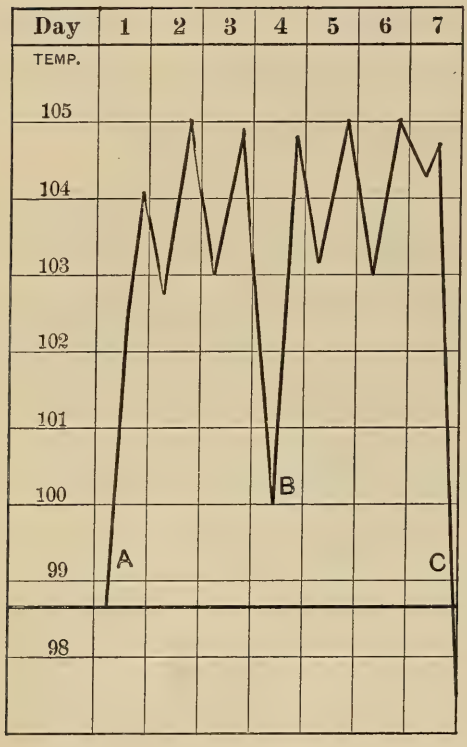


FIG. 7.—Fever curve of typical case of pneumonia: *A*, sudden rise; *B*, pseudocrisis; *C*, crisis.

2. FEVER.—Its usual course is, first, a *very sudden rise;* the fever is then *continuous,* often with not more than 1° or 2° variation; in three-quarters of the cases the maximum fever averages 102° to 105°. It may then remit, which is of good prognostic import and then at the end of a week's illness follows the crisis (*v. i.*). Remittent fever throughout the disease occurs in some cases. Recrudescences occur when consolidation passes from one lobe to another. The fever is lower or even absent in the aged, alcoholics, and in secondary pneumonia; the writer saw a total pneumonia, with normal rectal temperature in a man aged forty-eight. Fever causes a loss of weight of even twenty to thirty pounds. The rise preceding the crisis (*perturbatio critica*) may be mistaken for an antemortem rise; in both conditions there may be delirium, chill or an alarmingly irregular pulse. A pseudocrisis may deceive the physician,

the fever resuming its original elevation. *Crisis* occurs in 80 per cent. of cases, usually on uneven days, as noted by Hippocrates—the fifth seventh, ninth or eleventh day—usually between evening and early morning. It very rarely occurs on or before the third day. It marks the end of the toxemia but not of the consolidation which lasts several days more. In six to ten hours the temperature falls to or below normal,

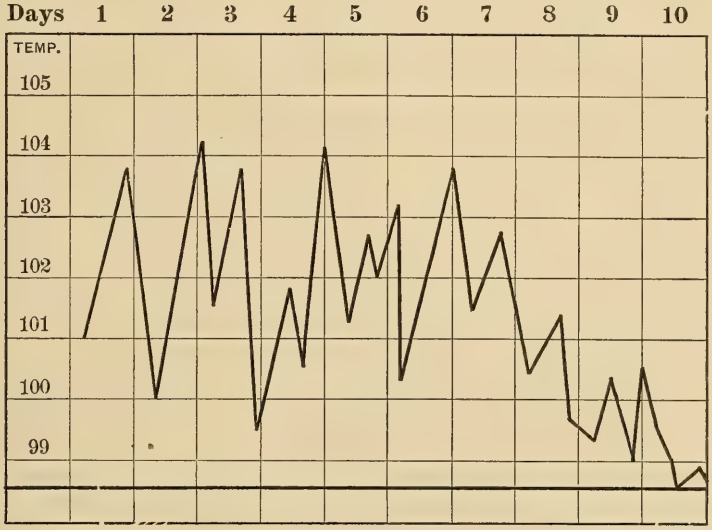


Fig. 8.—Pneumonia; remittent (subcontinuous) fever.

accompanied by sleep, euphoria, lessened pulse and respiration, lateritious deposits, reappearance of the chlorides in the more abundant, limpid urine, and "critical" sweating or even diarrhea. The fall may measure even 10° or 11° and the pulse may reach 30. Lysis occurs in 20 per cent. of cases. It is likely to occur when the fever lasts more than ten days, but lingering fever may indicate progression in other lobes or complications, as empyema or endocarditis.

3. Respiratory Findings.—(1) *Pain* in the side is generally pleural and its presence *in 90 per cent.* of cases gives it a diagnostic value; this

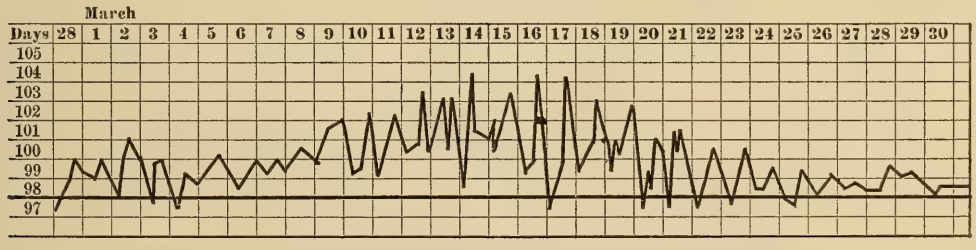


Fig. 9.—Pneumonia fever curve; exceptionally protracted though uncomplicated fever.

most frequent respiratory symptom appears early, is increased by coughing or motion, and is sharp or even agonizing. It is usually localized over the consolidated focus, but in 8 per cent. is referred along the lower intercostal nerves to the abdomen, in children especially, and sometimes in adults. Appendicitis or cholecystitis may be thus simulated. Sometimes the pain is radiated to the opposite side of the chest. Pain is

absent in central, secondary and upper-lobe pneumonias; often in the aged and in children; and in cases with great delirium.

(2) *Dyspnea* is frequent; it is caused especially by *toxemia,* but also by pain, cardiac weakness and bronchitis.

(3) *The Respiration-rate.*—Because of cerebral toxemia, or pleuritic pain, the respiration-rate is *increased* to 24 or 40 (or more in children); and in two-thirds of all cases, it ranges between 30 and 50; it almost never rises to 60 or 80 except in the death agony. The author saw a rate of 96 with recovery; the pulse was 90. Dilatation of the nares is said to be ominous, but to the author seems common in ordinary cases. *Alteration of the pulse and respiration ratio* was first noted by von Jürgensen; the normal ratio of 1 to 4 becomes 1 to 3 or 2. In a personal observation with the entire right lung consolidated, the respiration-rate was 18.

Fig. 10.—Fibrinous coagulum from a case of croupous pneumonia (Bizzozero).

(4) *The Cough.*—The cough always irritates, and seldom relieves the patient. It is present from the onset to the crisis, is due to the bronchitis and almost invariably occurs in lower-lobe pneumonia; it is often absent in upper-lobe, terminal or secondary pneumonia, in very delirious subjects, in the aged and young.

(5) *The Sputum.*—The sputum, at first transparent, mucous and scanty, soon becomes *viscid,* and difficult to raise and to expectorate, as it adheres to the mouth and lips. It is so viscid that the sputum-cup may be inverted without spilling the sputum. In two days it becomes *red and rusty* (80 per cent. of cases). Later it becomes more abundant. Increased fluidity may rarely result from weak heart. Rusty sputum is often absent in children who swallow sputum; in upper-lobe pneumonia, in which there is less hemorrhage, less consolidation and less cough to dislodge exudation; and finally in the senile and secondary pneumonias, because of weakness or toxemia. The cause of unusual color in the sputum, such as lemon- or brick-color, is unknown; it is sometimes observed in pulmonary edema or incipient gangrene and obscures the prognosis. Safranin color (*sputum croceum*) indicates resolution. The rusty sputum is of great diagnostic value—*e. g.*, in the beginning of the disease or in central pneumonia—and generally is of good import. *Fibrin threads* or "casts" of the bronchioles are usually seen between the third and seventh days. On shaking the sputum in water and catching the shreds on a slide, they appear clubbed

at their alveolar ends; they are also found in tuberculosis and fibrinous bronchitis. They consist of fibrin, white cells, fatty cells and epithelium. The sputum contains blood cells, epithelia and *pneumococci*, found first by Wolff in sputum and by Weichselbaum in the lung.

(6) *Lung Pathology.*—Laennec, the first to differentiate pneumonia from pleurisy (1819), described three stages: (*a*) The stage of *hyperemia* is very exceptionally observed unless the subject dies in twenty-four to thirty-six hours, although hyperemia may be seen at the edges of the consolidation where the pneumonia is beginning to extend. The lung crepitates less and floats; it is red, tears readily, since it has lost its elasticity, and on section exudes a serosanguineous fluid. Under the microscope, congestion, serum and diplococci in the alveoli are observed. The alveolar epithelial cells swell and desquamate, which, according to some observers, are the primary changes. (*b*) In the stage of *red hepatization*, the lung is evenly red, friable, airless (not wholly so in penumonia of an upper lobe), and sinks when put into water. Its weight is increased ten to fifty ounces. The lung has marks of the ribs on its fibrin-coated surface and is enlarged to the size of deepest inspiration (Rokitansky, who developed the gross anatomy of pneumonia). On scraping the cut surface, *fibrinous plugs* or casts of the bronchioles are observed. The lung is *granular on section* (Laennec), from fibrinous exudation into the alveoli; these granulations are seen best by oblique illumination. Under the microscope, coagulated blood and exudate, alveolar epithelium, injected bloodvessels, emigration of leukocytes, diplococci and *meshes of fibrin* are noted. (*c*) In the stage of *gray hepatization*, the granulations are grayish-yellow—not red, because the blood is absorbed; they are larger and looser than in the second stage. The connective tissue is grayer than the granulations, and is sharply defined by the lung pigment. It has been claimed that death always occurs if this stage is reached. If the patient lives, the granulations disappear; the lung exudate becomes semifluid (purulent infiltration) and can be washed out; resorption of this fatty emulsion occurs by the lymphatic vessels and not by evacuation through the sputum. Proteolytic ferments, probably derived from the leukocytes, dissolve the exudate; the products of this autolysis are excreted by the kidneys.

Seat of Pneumonia.—The process begins at the hilum and extends to the pleura, as proved by the *x*-rays. If it remains at the hilum it is central pneumonia (1.7 per cent.); if the entire lung is involved, it is pneumonia totalis or, if the bronchi are plugged, massiva; the terms "double pneumonia" and "crossed pneumonia" are self-explanatory. The lobes are involved in the following order of frequency: (*a*) Right lower lobe; (*b*) left lower; (*c*) right middle; (*d*) right upper; (*e*) left upper lobe. Jürgensen's 15,614 cases of pneumonia show that the right lung is affected in 53 per cent., the left in 37, and both lungs in 10 per cent. The upper lobes are involved in 16 per cent. and the lower in 84 per cent.

(7) *Physical Signs.*—The physical signs develop in the lungs, at the earliest, only after twelve to twenty-four hours.

(*a*) *Inspection.*—The decubitus is often dorsal in lower-lobe, and erect in upper-lobe pneumonia; the patient at first lies on the sound side to

avoid pain; on the diseased side later, for better expansion of the sound lung. The spinal column is concave toward the diseased lung for its better immobilization. The *excursion is less on the diseased side* because of pleuritic pain. Litten's sign is absent and the sound lung is vicariously active. The diseased side is larger, by one inch or so. The accessory muscles of respiration are active and dilatation of the nares is seen. Systolic pulsation of the lung occurs when the lingual lobe is consolidated, or, in the first stage, when the heart beats against semifluid exudation.

(*b*) *Palpation.—Lessened excursion* is detected more readily than by inspection; *vocal fremitus is usually increased* because of better sound propagation, fremitus being normally greater on the right side. It is decreased by (i) bronchial occlusion by fibrinous plugs, usually dislodged, however, by having the patient cough; (ii) coincident pleural effusion; and (iii) great consolidation rendering the chest wall tense. Pleural friction may be felt.

(*c*) *Percussion.*—(i) A *tympanitic note* (Skoda's resonance) is obtained in the first stage, caused by decreased elasticity of the lung and therefore decreased amplitude of vibration, due to the swelling of the alveolar epithelium and turgescent capillaries. This condition lasts one or two days, and may cause doubt as to the side involved. Tympany may denote new pneumonic extension (relaxation of the lung tissue contiguous to areas already consolidated). Tympany in the later stages is not uncommon in upper-lobe pneumonia where exudation is usually incomplete or in moderate compression, *e. g.*, of a right upper lobe when the right middle and lower are consolidated. It is rare in lower-lobe pneumonias. Tympany may persist days after the crisis. (ii) *Dulness* usually develops *on the second day* and is the percussion sign of the second stage. When complete it corresponds to the lobe confines. The dulness is not absolute. It is usually first detected in the posterior axillary line in lower-lobe pneumonia, and generally is more complete behind than in front, and more in lower- than in upper-lobe involvement. Dulness is difficult to detect if the area is not at least 1 by 2 inches, or if it is deeper than 2 inches from the surface. It is absent in central pneumonia. With resolution it is replaced by tympany.

(*d*) *Auscultation.— Decreased breathing,* or sudden local puerile breathing is often noticed at first.

First Stage.—The *crepitant rale* of Laennec remains the auscultatory sign of the first stage; but, as he stated, it is not specific, because it also occurs in tuberculosis, lung edema, infarct, hypostasis and bronchiolitis. It originates in the viscid exudate in the finest tubules and alveoli. It is coarser in the aged and in children because the large tubes are more involved. It is heard at the height of inspiration as a succession or shower of delicate crackles; when not heard, a coughing effort may elicit it; it often disappears after a number of deep inspirations, *e. g.*, when demonstrated to a class, only the first students hear it. The crepitant rale disappears during the second, and reappears in the third stage.

Second Stage.—The intense *bronchial breathing* is due to increased transmission of the breath sounds through the solidified lung. It sounds

as though nothing intervened between the lung and the stethoscope and is especially near when the ear is applied directly to the chest. It is absent or partial, especially anteriorly, in central pneumonia, coincident pleurisy, and obstruction of the bronchi by fibrinous casts. In the latter case, several coughs usually loosen the obstruction and restore the bronchial breathing. *Bronchophony*, also explained by increased conduction, is heard when the patient counts or speaks, but especially after each involuntary expiratory grunt. *Egophony* is occasionally heard, as well as Baccelli's phenomenon (auscultatory whispering). The crepitant rale disappears in this stage, except at the edges of the hepatization where extension occurs.

Third Stage.—The crepitant rale reappears—the rale redux.

4. Circulation.—(*a*) The *pulse-rate* is usually 100 to 120, or higher in children, the aged, and severe *toxemia*. In adults, a rate of 130 to 140 or a slow pulse, indicating grave heart or brain complications, irregularity and gallop-rhythm make the prognosis less favorable. Antecritical irregularity frequently occurs. The pulse is deceptive and its fulness is of no value in prognosis. Extensive pneumonias may compress one subclavian artery, making the corresponding radial smaller. (*b*) In the *right heart*, dilatation from toxemia frequently occurs; the second pulmonic tone is accentuated unless the right ventricle becomes greatly weakened. There may be functional bruits. In very rapid cardiac action, the two heart tones sound alike, resembling the fetal tones (embryocardia). (*c*) *Collapse*, usually due to myocardial or to central *vasomotor paralysis*, may occur at any stage. (*d*) *The blood* shows anemia. The fibrin increase is readily noted in fresh blood preparations. A polymorphonuclear *leukocytosis* appears early, and slightly survives the fever. The leukocytosis ran between 15,000 and 30,000 in 75 per cent. of Cabot's counts; it may reach 100,000. The eosinophiles are decreased or absent until the acme whence the favorable significance of their reappearance. The lymphocytes are decreased. During convalescence many myelocytes appear. The white count remains high in delayed resolution or complications. A low count generally is unfavorable. *Pneumococcemia*, until recently considered of bad import, is of no prognostic value, since it occurs in 90 to 100 per cent. of cases, perhaps even before hepatization. The agglutinative power of the serum increases until the crisis occurs.

5. Digestive System.—There is the febrile thirst and anorexia. Vomiting occurs in 25 per cent. of cases, from toxemia or coughing. The distressing tympanites occurs in the severer cases—and invariably in alcoholic subjects—from toxemic paresis of the intestinal muscularis. The occasional diarrhea is toxemic; constipation is the rule. There is often intumescence of the liver and spleen (in 15 per cent. of all cases).

6. Skin.—The red cheek on the affected side was known to Aretæus. The skin is usually dry, but miliaria and sweating may be seen. *Herpes* is found more often than in any other disease (40 per cent.), from the third to fifth day, chiefly on the face; Howard found causal changes in the Gasserian ganglion; herpes may develop on the body, limbs, genitalia, etc. It is of diagnostic and generally of favorable import.

Rarer eruptions are erythema, roseolæ and purpura. The lymph glands are enlarged in 25 per cent. of cases.

7. URINE.—Characteristic fever urine is found, and the urea is increased threefold after the crisis from overproduction and accumulation during the fever; from the resolving exudate, uric acid and xanthin result. The chlorides are decreased (from 225 grains daily to 30 grains) or absent until the crisis, when they reappear in increased quantities (Rottenbacher); no satisfactory explanation for the chloride retention is known; The urine often becomes neutral or alkaline two days after the crisis. *Albuminuria and cylindruria* appear in 50 per cent. of cases; peptonuria and acetonuria are not uncommon; and the diazo reaction appears in 20 per cent., hematuria in 1.6 per cent., nephritis in 1 per cent., and uremia very uncommonly.

8. NERVOUS SYSTEM.—*Headache* is constant. There may be insomnia, restlessness, or stupor which is always ominous. *Delirium* (22 per cent.) occurs with upper-lobe pneumonia, alcoholism or marked toxemia; nervous symptoms resulting from exhaustion may occur even a week after the crisis (2.5 per cent.). The patellar reflexes are often absent and the pupils may be dilated or sluggish. Convulsions, coma and rigid neck, in children, result from toxemia, brain edema or meningitis. Hemiplegia may result from brain edema or anemia, or arteriosclerosis plus a weakened heart.

Mechanism of Death in Pneumonia.—Death is rare in the first three days, except from some antecedent malady, as nephritis. (1) *Toxemia is all-important in its action on the heart, brain and vasomotor nerves.* Death ensues with rapid pulse and respiration, algid extremities and obtunded sensorium. (2) *Pulmonary edema* is cardiac, or inflammatory, from an extending pneumonia. (3) The *extent of pneumonia* is the least important factor. A massive pneumonia in a robust person causes less disturbance than a small patch in a nephritic. There is no parallelism between the area hepatized and the resulting toxemia. (4) *Pneumococci in other tissues* cause pneumococcic meningitis, endocarditis, etc.

Complications.—These are few but severe.

1. PLEURISY.—Dry pleurisy is so constant as to justify the name of pleuropneumonia, the pleura being covered with a thick, tenacious, exudate, usually pneumococcic; if due to the streptococcus, it is a complication of great danger. Fränkel saw *serofibrinous* pleurisy in but 1 per cent. of 1000 cases, yet Maragliano aspirated some fluid in 66 per cent. of his pneumonic patients. The writer has seen three cases of pneumonia on one side and pleurisy on the other. Pneumococcic *empyema* occurs in 2 per cent. of pneumonias and is notable since the pneumococcus seldom provokes suppuration in the lung (*v.* Pleurisy). Recovery is possible even without operation. *Interlobar empyema* is most serious, because it so often escapes detection.

2. ENDOCARDITIS.—Acute endocarditis complicates pneumonia in 1 per cent. of all cases and 5 per cent. of fatal cases. *Endocarditis is malignant in* 80 *per cent.* of cases; 25 per cent. of all ulcerative endocarditides are pneumococcic. The pneumococcus is found in the vegetations. The *valvular exudate* is voluminous, though *ulceration*, valvular

aneurysm and perforation are less common than in staphylo- and streptococcic forms. Endocarditis develops especially on old valvular lesions in the left heart, most frequently on the aortic valves. The right heart is involved three or four times as frequently as in endocarditis due to other bacteria. Sixty per cent. of cases are associated with *meningitis*. Middle-aged adults are most often affected. Endocarditis is probable when a murmur, systolic or particularly diastolic, occurs with irregular protracted fever, chills, sweats, positive blood cultures and embolism (50 per cent.). Symptoms may directly follow the pneumonia or develop after one-half to one week of normal temperature. Fever is sometimes, and leukocytosis frequently, absent. Symptoms may be absent (*v.* Septic Infections and Ulcerative Endocarditis).

3. Pericarditis.—Its frequency is 1 per cent., and the pericardial rub is absent in 60 per cent. of cases. Dry pericarditis occurs more often in children, at the height of the disease and when the left lung is involved. Effusive pericarditis is less common than the fibrinous form.

4. Arteritis and Phlebitis are less frequent than in typhoid. Steiner collected forty-eight cases of venous thrombosis (1903); it develops in convalescence and heals usually (75 per cent.), though embolism or permanent venous obstruction may develop.

5. Nervous Complications.—*Delirium tremens* may develop in an alcoholic from the shock of the pneumonia onset; or, during the course of delirium tremens, pneumonia may develop insidiously and is easily overlooked and remarkably fatal (*v.* Prognosis). *Meningitis* occurs in 5 per cent. of autopsies, and in 0.3 per cent. of clinical cases, in asthenic patients, in upper-lobe pneumonia, and often accompanies endocarditis. One of the author's cases died a week after apparent convalescence, and the autopsy showed a quarter inch of pus over the convexity. The usual meningitic symptoms develop if infection reaches the base of the brain, but this is not usual (see Meningitis). The mortality is 93 per cent.; the author has seen recoveries.

6. Alimentary Complications.—Parotitis and peritonitis occur, each 0.3 per cent. Dieulafoy described an ulcerative gastritis caused by pneumococcic embolism and attended by gastrorrhagia. Fussell collected 12 cases of acute dilatation of the stomach. Icterus averages 1 per cent.; its causes are hepatic stasis, duodenal catarrh, and hematogenous jaundice from toxemic hemolysis. (See Pneumococcic Peritonitis.)

7. Lung Complications.—Suppurative mediastinitis was regarded as the connecting link between pneumonia, meningitis and pericarditis. Pulmonary induration may follow incomplete resolution. Abscess develops in 5 per cent.; pus and elastic fibers in the sputum, less frequently cholesterin and hematoidin crystals may be the sole signs, and continual coughing the chief symptom. *Gangrene* occurs in 1 to 2 per cent. of the cases. Clinically it is recognized by the horribly fetid sputum.

8. Arthritis.—Hematogenous pneumococcic arthritis may be serous or purulent; its incidence is 0.5 per cent. and its mortality 28 per cent. In 62 per cent. it is monarticular, and the knee is involved in 30 per cent.; 158 cases are reported.

Clinical Types and Variations.—These are as follows:

1. Abortive Pneumonia is recognized by the sputum, fever and respiratory disturbance, but produces no hepatization.

2. Ephemeral Pneumonia is characterized by one day of fever, and very rapid resolution. The onset is brusque and severe but the symptoms speedily abate.

3. Relapsing Type.—After the crisis, the entire cycle recurs (in 0.3 per cent.). Recurrent type (*v. s.*).

4. Local Variations.—Apex pneumonia is often more severe, atypical, adynamic and complicated. Migratory pneumonia travels from one part to another (so-called erysipelas-pneumonia). Central, total and massive pneumonia (*v.* page 67).

5. In Children.—An abrupt onset occurs without chill before the seventh year, but with vomiting (50 per cent.), convulsions (7 per cent.), and other cerebral symptoms. Cough is often absent, no sputum appears, and the pain is referred to the abdomen (third to fifth year). *Pain* is more important than increased respiration, which occurs in any fever; the respirations may number eighty under two years of age; in older children, fifty. Crepitant rales are usually absent because of the superficial breathing caused by pain. Latency of physical signs, such as absence of bronchial breathing, bronchophony or dulness is sometimes noted. Localization is more often central, apical or migratory. Lysis is more frequent than in adults.

6. In the Aged.—Pneumonia is frequently latent, insidious and asthenic, with ill-marked signs, without chill, even without fever, and often without cough, pain or sputum. It is remarkable that so severe an infection can exist with so few of its usual symptoms. In this type, pneumonia is declared by the physical findings only.

7. In Alcoholics.—(See page 71).

8. Asthenic Pneumonia.—In asthenic or "typhoid" pneumonia, toxemia is dominant, with high fever, enlarged spleen, albuminuria, icterus, severe complications and high mortality. In contrast to these cases, the author had two patients who worked throughout the disease.

9. Secondary Pneumonia.—Pneumonia may be secondary to emphysema, arteriosclerosis, hepatic cirrhosis, nephritis, diabetes, tuberculosis, typhoid or other conditions. The frequency of ether pneumonia in abdominal operations suggests the possibility of embolism as a factor; ether predisposes to pneumonia by irritation of the respiratory tract. The nose and mouth should be cleansed before anesthetization to remove native germs.

This type is atypical, ill developed, or masked by the primary disease; the hepatization is rarely typical and is more lobular than lobar; the lung section is smooth and moist, and the exudation is more cellular than fibrinous.

10. Delayed Resolution.—Resolution is effected by lymphatic absorption. (*a*) Resolution may require weeks or even months, especially in apical involvement, and more often after a lysis than crisis; the toxemia disappears but dulness and bronchial breathing persist. (*b*) Imperfect resolution may be caused by abscess formation, or inflammatory involve-

ment of the connective tissue and lymphatic vessels. Induration of the lung by organization of the exudate is usually febrile.

Diagnosis.—Mistakes are rare in the diagnosis of frank pneumonia in adults—they occur mostly in aged individuals, alcoholics, children and secondary pneumonia. All subjects with chronic disease must be closely examined when the respiration, pulse and temperature rise, or when the subject is not doing well.

1. Onset Symptoms.—The chill in adults, or convulsions and vomiting in children, suggest the possibility of pneumonia. Chill, pain in the side, sudden high fever and rusty sputum, are almost final, because practically the only other disease in which they occur is *infarct of the lung*—in which the sputum is reddish, not viscid, usually darker, and more abundant. In 25 per cent. of cases the onset of pneumonia is not brusque and the physical findings of pneumonia are present without a typical history; indeed it may rather be suggestive of simple pleurisy, etc. Pneumococcemia without lung involvement may simulate influenza (*q. v.*).

The occasional reference of the initial pain to the abdomen may simulate *appendicitis*. As a rule the abdominal pain exists without tenderness or with superficial tenderness only. Consideration of the toxemia and respiration-rate generally prevents errors in diagnosis; collapse, intense abdominal tenderness and rigidity may simulate perforation.

2. Physical Findings in the typical case appear within two days after the onset: lessened expansion, increased vocal fremitus, dulness, bronchial breathing, the crepitant rale, the grunting, short expiratory "huh," and a bronchial tone to the whispered or louder voice sounds. These findings are modified by many conditions; thus the fremitus is decreased in massive consolidations, in coincident pleural effusions or in fibrinous plugging of the bronchi; usually the plugs are dislodged by repeated coughing. The dulness is preceded and followed by a tympanitic note. The crepitant rale is found only in the first and third stages, but in almost every case can be found in some part of the affected lung. Bronchial breathing is rudimentary or absent in incomplete hepatization, is often so in upper-lobe pneumonias, in complicating effusive pleurisy, and in obstructed bronchi. The physical signs are pathognomonic only in connection with the cyclic course, disturbed pulse and respiration ratio, pneumococcemia, herpes and leukocytosis.

3. Localization.—Massive pneumonia not infrequently simulates pleurisy, because fibrinous plugging of the bronchi abolishes bronchial breathing, bronchophony, the crepitant rale and vocal fremitus; there may be slight luxation of neighboring viscera. Consideration of the onset, cyclic course and the dislodging of the plugs by forcible coughing almost invariably determines the diagnosis. Conservatism is necessary in the diagnosis of central pneumonia; all pneumonias are central in their incipiency; in two personal cases the local findings did not appear for a week.

4. In Children.—Pneumonia may be mistaken for abdominal disease or pleurisy. "Cerebral pneumonia" may be (*a*) convulsive, occurring

under two years, with fever and vomiting, but the evenly high fever is suggestive even if the cough and dyspnea are obscured by the brain symptoms; or (*b*) meningeal, occurring between the second and sixth years; but the pulse is not slow, and there is no other sign of meningitis.

5. DIFFERENTIATION.—Differentiation from *pleurisy* is usually easy (*v. i.*). In *lung tumors*, bronchial breathing, bronchophony, dulness, red or green sputum, are also noted, but the history and course differentiate. The course differentiates *acute tuberculous infiltration* from an upper-lobe pneumonia in which cough, sputum and typical physical findings are absent. Mistakes in both directions are made, by forgetting that in apical pneumonia the incomplete exudation may impart a tympanitic note, and that the breath sounds are not always bronchial, at least anteriorly. Some tuberculous pneumonias may commence like lobar pneumonia (see COURSE OF PULMONARY TUBERCULOSIS).

PNEUMONIA——*vs.*——PLEURITIC EFFUSION.

Onset sudden, with rigor, herpes.	*Gradual;* chilly sensations commoner.
Friction less common; may be present.	*Usual* at some time in its course.
Cyclical short course; high fever, crisis.	*Longer* duration, lower fever, lysis. Often tuberculous foci at apex, etc.
Sputum rusty.	*Mucous*, if any.
Leukocytosis.	Usually *absent*, unless purulent or complicating a pneumonia.
Ectasia absent.	*Present.*
Fremitus increased (coughing usually dislodging bronchial plugs).	*Absent* over effusion (unless adhesions bind the lung to the chest wall; increased above, over compressed lung or in the interscapular region).
Dulness, preceded and succeeded by tympany (tympany or partial dulness in upper lobe pneumonia is not uncommon).	*Flatness*, increased downward; entering Traube's space on the left side. Level seldom shifts with change of position.
Crepitant rales, *bronchial breathing*, and bronchophony (unless bronchus is plugged).	*Decreased or absent respiratory murmur* (bronchial breathing in interscapular region from compression, or even through the fluid; egophony).
No essential luxation of heart, liver or spleen.	*Luxation* of viscera usual in large effusions.
Paracentesis negative.	Tapping nearly always decides doubtful cases; *fluid found* beneath the flat area.

Prognosis.—Pneumonia is the most fatal acute disease and has even a greater death-rate than tuberculosis. In 1903, 4629 persons died of pneumonia in Chicago, and in 1904, 8360 in New York City. From 1851 to 1860 the mortality in Chicago from tuberculosis was 35 per cent. more than from pneumonia; from 1891 to 1901, the pneumonia death-rate was 9 per cent. more.

There are various prognostic factors: 1. The VIRULENCE of the pneumococcus, which varies from year to year (5 to 31 per cent. mortality). The mortality of 85,000 cases in the German army was 3.6 per cent. In 465,400 collected cases, E. F. Wells found the death-rate 20 per cent.

2. AGE.—The mortality is: one to five years, 30 per cent.; five to ten years, 3; ten to twenty years, 5; twenty to thirty years, 8; thirty to forty years, 25; forty to fifty years, 39; fifty to sixty years, 43; sixty to seventy years, 53; seventy to eighty years, 87 per cent. Cruveilhier

spoke of pneumonia as the most formidable scourge of the aged, and Peter characterized pneumonia as the natural end of old people.

3. Sex.—The mortality is over 50 per cent. higher in women.

4. Location.—Right-sided is more fatal than left-sided pneumonia, and upper- is more fatal than lower-lobe involvement.

5. Unfavorable conditions include cirrhosis and nephritis (almost 100 per cent. mortality); delirium tremens and inveterate alcoholism (50 per cent.); cardiovascular disease, etc.

6. Unfavorable symptoms are long prodromata, hyperpyrexia, enteritis, jaundice, albuminuria, severe pain, marked cyanosis, pulmonary edema, diffuse bronchitis, and a white cell count under 10,000. "When the blood-pressure, expressed in millimeters of mercury, does not fall below the pulse-rate expressed in beats per minute, the fact may be taken as an excellent augury; the converse is equally true" (C. A. Gibson). The mortality is 60 per cent. with the respirations constantly over 60 per minute, and 50 per cent. with the pulse over 130. Over 50 per cent. of delirious subjects die.

7. Unfavorable Complications.—Endocarditis and meningitis are almost always fatal; abscess, gangrene and arthritis are always serious; pericarditis is fatal in 75 per cent. of the cases.

Death is due to *cardiac toxemia.* Mechanical overwork of the heart or diminished respiratory space is of small importance.

Treatment.—1. Prophylaxis concerns disinfection of the sputum, cleansing of the room (page 258), isolation and maintenance of the physiological resistance.

2. Serotherapy.—Serotherapy and vaccines have not proved successful and pneumonia remains a self-limited disease.

3. Hygiene is the same as in typhoid; quiet, frequent change of posture, etc.; the patient should be clothed with a light flannel jacket open down the back. The *fresh air treatment* works wonders in pneumonia; the bed should be placed out of doors or the windows widely opened. Aretæus spoke of the pneumonic's desire for cold air. Cold air often raises the blood-pressures 10 to 20 mm. and allays delirium.

4. Diet.—The diet consists of milk, eggs, beef-juice and cereals (*v.* page 53). Plenty of water is indicated; some is retained in connection with the chloride retention and much voided by the increased respiratory effort.

5. Symptomatic Therapy.—(*a*) *The chill,* which is rarely seen by the physician, is managed as in malaria.

(*b*) *Cough.*—Expectorants are unnecessary; codeine gr. $\frac{1}{4}$ to quiet irritation and carbonate of ammonium gr. iij to stimulate secretion, may be given every four hours; morphine may be indicated (*v. i.*).

(*c*) *Pain.*—The chest should be firmly strapped with adhesive plaster, applied in several oblique directions so that the pieces cross each other. Morphine lessens the irritability of the respiratory centre and relieves pain. Ice, blisters, leeches, and the cautery are superfluous; heavy poultices impede respiratory movement. In pneumonia, general is more important than local treatment.

(*d*) *Care of the heart is the main objective* in treatment. Constant watching is imperative lest the patient leave his bed; it is a safe rule to

consider all pneumonics delirious. Sitting up to drink, defecate or urinate is never permissible in a disease of sudden and alarming changes, with constant danger of collapse. No measure which depresses the heart should be employed, and *acetanilide, antimony and aconite are always to be avoided.*

Cardiac Stimulation.—Strychnine is the best vasomotor tonic, and should be given in every case because vasomotor paralysis and collapse develop without warning, attended by accumulation of blood in the abdominal vessels. Strychnine is here indicated, because it constricts the deep abdominal vessels; the peripheral vascular tonus is not increased, but the brain vessels are flushed and the bulbar vasomotor centre is stimulated. The drug stimulates the central nervous system; the digestive tract, its secretions and musculature; the circulation, the tonicity of the heart, but chiefly the vasomotor centre; and the respiratory centre.

The dose is gr. $\frac{1}{30}$ to $\frac{1}{20}$, every two to four hours. (For dosage in children see page 269.) Some strychnine remains in the system over three days, some is destroyed at once by the liver, and some is quickly eliminated by the kidneys. If the kidneys or liver are diseased as in old age, the drug may accumulate. Full doses produce twitching, nervousness, palpitation or renal-vessel spasm. Hypodermic administration gives less chance for the liver to destroy the drug.

Caffeine is of great value, particularly when morphine is indicated, supporting its tonic effect and neutralizing its somnifacient action. Rhomberg ranks caffeine above strychnine and camphor. (*v.* Therapy of Valvular Heart Disease).

Ammonia is of great clinical importance. It stimulates the heart-muscle, increases its frequency, and raises arterial tension. Its superficial effects are flushing, warmth, exhilaration and increased secretion of urine, sweat and mucus. Its prime indication is sudden functional cardiac adynamia, *e. g.*, at the crisis. It is a local irritant to the skin, and respiratory and alimentary mucosæ, and therein lie its therapeutic value and its clinical disadvantages. Its application to the upper respiratory passages or its ingestion by mouth causes a sudden reflex stimulation of the respiratory and circulatory centres before the drug is absorbed. Gastric, intestinal, renal and bronchial irritation from overdosage, and irritation following subcutaneous administration may be observed. The corrosiveness and fugitive action of ammonium carbonate are obviated by small, frequent doses, gr. iij, every two hours.

Digitalis was once exhibited in enormous doses, which simply proves that the drug is not absorbed; Thomas, in 1865, remarked its inaction in fevers. The author considers digitalis far inferior to camphor, and has repeatedly noticed toxic symptoms at the crisis when the repeated doses of digitalis were absorbed together. Digitalis is a last resort (♏ij–v of the fluidextract hypodermically). *Strophanthin* should not be used if digitalis has been administered, as it may cause sudden death. (See Treatment of Valvular Heart Disease.)

Alcohol was first recommended by Todd for fevers. The chief interest centres in its action upon the circulation. Formerly called a heart stimulant, pharmacologists now hold that alcohol does not stimulate the

heart, except by preparations like champagne which contain other ingredients. The heart's frequency is increased, but the drug acts as a vasodilator or as a blood distributor. Some clinicians reject it because it is narcotic to the respiratory, circulatory and vasomotor centres in the lower brain, or because it is a vasodilator. Alcohol is effective in all varieties of sepsis and it protects the tissues from excessive waste. Alcohol is indicated by collapse or cardiac toxemia, in which hot whisky and water unquestionably stimulate by reflex action on the throat and stomach; cognac, or whisky in hot water, and coffee can also be given by rectum. Alcohol may not be used in any routine fashion; within limits, it is valuable.

Opiates.—The cardiovascular value of morphine is not sufficiently appreciated. Many withhold opiates even when the drug is indicated by maniacal outbursts, active delirium, and harrassing coughing or pain, lest it depress respiration and the heart. However, it decreases the irritability of the respiratory centres, lessens cough, dyspnea, headache, and insomnia. Its renal effects are negligible, as little morphine is eliminated by the urine. Morphine of itself helps to sustain life, and in critical cases its hypodermic use strengthens and regulates respiration. It is a *most valuable adjunct* to strychnine, camphor, or caffeine, which latter has an excellent effect, given by rectum, in neutralizing its narcotic action.

Camphor is an excellent diffusive stimulant, grain ij hypodermically in 10 parts of sterilized almond oil, every two to four hours.

Saline infusions are indicated in profound toxemia with weak heart —two drams of salt in one quart of water. They "lavage the blood." Continuous use of the *salt solution by rectum*, by the drop method, is valuable. Although once abused, *venesection*, employed by Hippocrates, has again come more into vogue. It is valuable at the onset in the robust for severe pain, active delirium and urgent dyspnea, and later when great engorgement of the right heart occurs. Heart stimulants must be used with phlebotomy.

Oxygen, said to relieve dyspnea, is often irritating and injurious. The mouth-piece should be held near, not over, the mouth. (An excess, experimentally, produces hepatization.)

Atropine is valuable for collapse, low temperature and clammy skin, in a daily dosage of not over one-fiftieth of a grain.

Adrenalin (1 to 1000), in 5–10 drop doses, is as efficacious given intramuscularly as intravenously; acting on the nerve-endings of the sympathetic, it is a vasoconstrictor; small and repeated doses are better than larger doses which may induce pulmonary edema.

(*e*) *Antipyretics.*—A high temperature is not *per se* injurious; patients with fever between 103° and 105° progress most favorably and those with temperature above or below these limits run a more severe course. All measures which *suddenly reduce* the temperature must be avoided, especially at the crisis, when the heart sometimes staggers under the sudden fall of the fever.

Hydrotherapy is opposed by many clinicans; by others the tonic vasomotor effects are thought to outweigh the shock. It is contra-indicated

in the obese, the weak and the aged. Cool sponging with water and alcohol is the safest method. *Quinine* has no effect except in large doses; so-called specific action is a phantasy.

(*f*) *Nervous symptoms* are toxemic, but may be considered with regard to: (i) fever; the greater the temperature variation, the more marked are the nervous symptoms, which are most efficaciously treated by baths or cool affusions; (ii) deficient oxygenation, in which cardiants and oxygen are indicated; (iii) insomnia, which is treated by morphine, hyoscine hydrobromide, sulphonal, and cool sponging, but rarely by chloral; and (iv) delirium tremens (*q. v.*).

(*g*) *Iodides* are indicated in delayed resolution.

DIPHTHERIA.

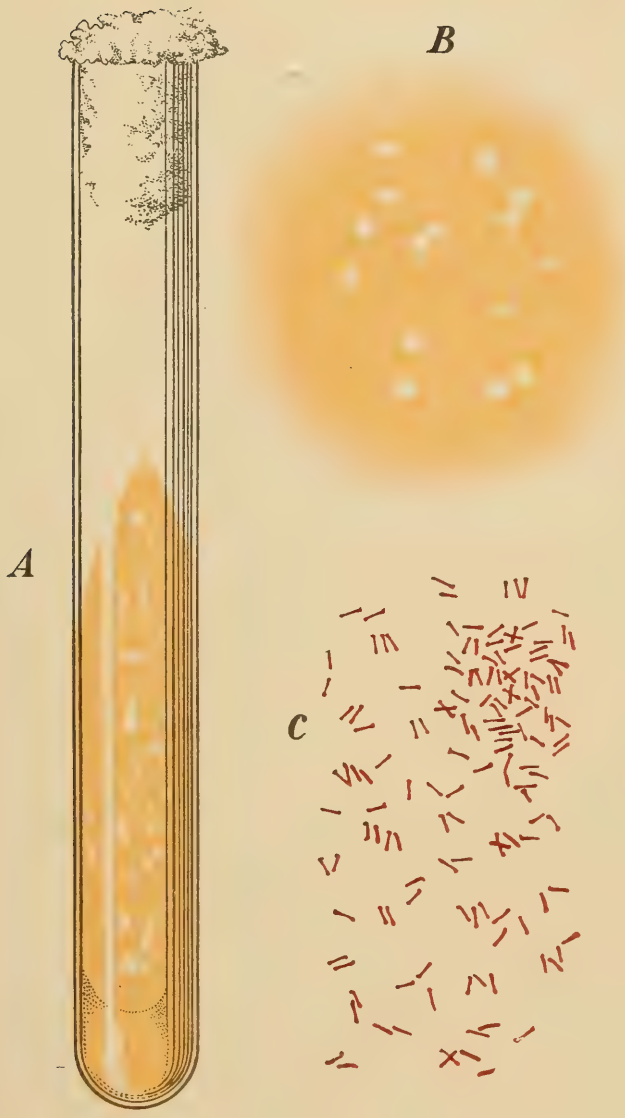
Definition.—An acute specific infective disease, usually of the throat or upper air-passages, and characterized by a membrane formed by the Klebs-Loeffler bacillus, and by toxemia.

History.—Diphtheria was known to Hippocrates. Asclepiades, 100 B.C., performed the first tracheotomy. The first full account was Aretæus', in the first century, A.D. Diphtheria literally means membrane and originated with Bretonneau (1821).

Terms.—True diphtheria implies a membrane formed by the Klebs-Loeffler bacillus whose absorbed toxins produce toxemia. Membrane, however, may be due to the scarlatinal virus, strepto-, pneumo-, staphylococci, etc.; while these are diphtheritic membranes in the old anatomical sense, they are not diphtheritic in the bacteriological sense, and we term them *pseudodiphtheritic*, or *diphtheroid*. Therefore, clinically, there are some discrepancies: (*a*) what seems diphtheria, anatomically, may not be due to the Klebs-Loeffler bacillus; (*b*) what apparently is angina or tonsillitis, may show no membrane or an atypical membrane, but bacteriologically is due to the Klebs-Loeffler bacillus; (*c*) the diphtheria bacillus is found in 1 per cent. of perfectly healthy throats; (*d*) there are bacilli which closely simulate the diphtheria bacillus. The pseudodiphtheritic membrane is most often due to the streptococcus and is seen oftenest in scarlatina, but also in typhoid, measles, Vincent's angina, syphilis and pertussis. It develops wherever the true diphtheritic membrane may occur, *e. g.*, the throat, larynx, bronchi, eyes, skin, etc. Most cases (72 per cent.) clinically resembling diphtheria are proved diphtheria by repeated bacteriological tests.

Bacteriology.—The diphtheria bacillus (described by Klebs, 1883, and cultivated by Loeffler, 1884) is 1.2 to 2μ long and 0.3 to 0.5μ wide, with small round or slightly enlarged ends. It stains readily by Gram's method, Loeffler's methylene blue and carbol fuchsin. There are many variations in form, but the serum culture always reveals the wedge- or mallet-like forms. Their grouping is characteristic, like Chinese letters, in lines set asymmetrically at various irregular or slight angles. They are immotile and sporeless. The bacillus overgrows other associated bacteria on blood serum mixed with glucose bouillon; the culture is visible in ten hours as yellow streaks, whose surface is slightly granulated and

PLATE III



A. Culture of diphtheria bacillus.
B. Same magnified.
C. Diphtheria bacilli.

edges somewhat thick. In twenty-four hours the growths diffuse, with a wave-like surface, undulating edges, and a succulent appearance. The diphtheria bacillus is very resistant, and though it usually disappears three weeks after the patient has shed the membrane, it may live for weeks in the throat (even fifteen months) or for a long period in clothing. It is found chiefly in the membrane, and is less frequently detected in the submucosa, heart (endocarditis), blood (diphtheriemia), lungs (bronchopneumonic foci), spleen, liver, brain, cord, kidney or urine. In one report, it was found in the blood and viscera eleven times in fourteen cases. Symbiosis with the streptococcus, the most common of all secondary infections, increases the virulence of the diphtheria toxin (Roux and Yersin); the streptococcus is found in septic types, bronchopneumonia and glandular suppuration. Less important are the staphylococci—which rarely enter through the throat, but then with increased virulence—the proteus, colon bacillus and pneumococcus.

The *action* of the bacillus is (*a*) *local;* when inoculated in animals, local necrosis develops, whereon fibrin exudes and forms a membrane in which the bacillus is found; and (*b*) *general,* forming toxins, absorbed by the blood and lymph vessels. Roux and Yersin found that the toxins injected alone, after killing the bacillus, produce no membrane or necrosis, but toxemia and paralysis similar to the postdiphtheritic paralysis. Ehrlich distinguishes two toxins: the *toxin* producing the ordinary phenomena of diphtheria, and the *toxon* producing the later anemia and paralysis. The chief receptors of the virus are the nervous and lymphatic tissues.

The *virulence* of the germ varies. This is tested by inoculating a guinea-pig with $\frac{1}{200}$ of its body-weight of a forty-eight-hour bouillon culture, and noting the results; (*a*) death in three days or less results from fully virulent cultures; (*b*) death in three to five days from one of medium virulence; (*c*) death after a longer time, or only local changes, from weak cultures.

Bacilli, known as the pseudodiphtheria bacillus, Bacillus xerosis, etc., resemble the Klebs-Loeffler bacillus, and may become virulent; they are not antagonized by the diphtheria serum but only by the pseudodiphtheria serum.

Dissemination.—Diphtheria is disseminated (1) by patients acutely ill with the disease; the secretion or membrane may be coughed into the mouth of one of the family, nurse or attending physician. Children going to school may impart to others a most virulent diphtheria, though the same bacilli have produced in them little constitutional reaction. (2) By healthy individuals, in 1 per cent. of whom the germ is found without causing diphtheria; these carriers may or may not have been in contact with the disease. (3) By fomites, especially damp cloths, in which the germ may live for five months. (4) The germ probably may be spread by dust, cheese, milk and eating utensils. It is not demonstrated that domestic animals convey the disease. Diphtheria is endemic in all thickly populated districts, from which epidemics develop.

Age.—Most infections occur between the second and fifteenth years, and most deaths occur between the second and fourth years. Jacobi

observed three cases in the newborn, but infants at the breast are less exposed than creeping children who come into close contact with dirt. Adults are frequently affected.

Hypertrophied tonsils, nasopharyngeal and bronchial catarrh, measles, nervous affections and cold weather are predisposing factors. Some individuals are temporarily, and some permanently, immune. One attack confers no immunity, wherein diphtheria resembles erysipelas, pneumonia and rheumatism.

Symptoms.—There are *three major localizations of diphtheria:* the *throat,* the *nose* and the *larynx.*

1. Pharyngeal Diphtheria.—The typical case is pharyngeal.

(*a*) The *simple localized diphtheria* after an incubation of two to seven days begins with fatigue, pallor, coated tongue, anorexia, dysphagia, vomiting, and pain beneath the angle of the jaw. Chilly sensations, fever and thirst and pharyngeal voice ensue.

The *pharynx* is glistening and characteristically purple-red, with, later, areas of gray, yellowish or dirty green color, as small white bands or irregular, gelatinous masses, located on the tonsils, palate, pillars or pharynx. Except in their very incipiency, they are adherent, and on removal leave red, bleeding spots which are rapidly covered again with membrane.

Wagner, Weigert and Oertel showed that the *diphtheritic membrane is composed:* (i) Of necrobiotic tissue; the superficial and, later, the deeper structures are necrosed; bacilli are found in the superficial layers and streptococci and staphylococci may be found more deeply situated; the necrobiosis may be noticed also in the connected lymph glands and internal organs (Weigert's coagulation necrosis). (ii) Somewhat deeper the membrane is composed of fibrin exudation, which is a conservative, walling-off process. (iii) In the deepest part of the membrane emigration of leukocytes occurs. In most adults the membrane is thrown off by the fourth or fifth day; in children by the seventh or eighth day.

The breath is septic in odor. Some nasal discharge is the rule. The glands at the angle of the jaw are tender. *Fever* may be irregular or absent; in 90 per cent. of patients who recover it does not exceed 102°. The patient usually recovers in seven to ten days, and seldom exhibits marked toxemia.

The form described is *the classical type of mild local infection;* the bacilli may be virulent but the tissues and blood resist the infection. *Several deviations from this type* may be misinterpreted: (1) In "catarrhal diphtheria" the membrane is absent or atypical and pultaceous; this type is common in families where the more typical forms are seen. (2) Diphtheria may resemble follicular pharyngitis; between them there is no absolute distinction except bacteriologically, although in diphtheria the red is brighter, the tendency to fuse is greater, and the spots are more yellow, gray or green. (3) Follicular tonsillitis may be very closely simulated, and then is only distinguished bacteriologically. (4) In chronic diphtheria, there is a deposit upon a red, edematous pharynx. The glands may be swollen, but usually there are no toxemic symptoms. (5) The latent diphtheria in marantic subjects may easily escape detection.

(*b*) The *diphtheritic general infection* begins with high fever and *toxemic symptoms* as headache, prostration, increasing anemia, dysphagia and vomiting. The heart movement follows the fever, the tones being dull and often accompanied by a systolic murmur. The pharynx is dark red, swollen and a thin gray membrane is seen on the tonsils or pharynx; it begins as streaks, or a thin veil over the pharynx and spreads rapidly. The secretion is offensive. The hard palate and upper soft palate are usually normal. The neck is tender from glandular intumescence. The nose is often closed by a purulent secretion and diffuse membrane, and oral breathing results, of great moment in nurslings. There is a type peculiar to young children who, with initial nasal symptoms and without throat symptoms, suddenly develop laryngeal stenosis, and in the pre-antitoxin days, almost invariably died. In severe cases the heart is irregular; there are throat paralysis, neck infiltration, dyspnea, albuminous urine, tympany, swollen liver and spleen, antemortem fall of temperature, and death from pneumonia, cardiac insufficiency, toxemia, paralysis or nephritis.

(*c*) In *septic diphtheria* the exudate becomes dirty green or brown, is horribly fetid, and may reach the nose or larynx, causing necrosis and stenosis. It excites lymphadenitis and periadenitis. The respiration is difficult; and nephritis, generalized hemorrhage or local arterial ulceration, failing pulse, low temperature, and intumescence of the abdomen, liver and spleen, usually result fatally. In 1881, 94 per cent. of cases of this type died. According to Baginsky, it is due to the diphtheria toxin, and is seldom observed since the introduction of antitoxin. Others maintain that symbiotic streptococcus infection determines this septic type.

The initial *fever in diphtheria* may be maintained by otitis, pneumonia, adenitis and other complications in the severer types. Septic temperature may prevail. A pre-agonal rise may be observed, or an antemortem collapse. These developments relate chiefly to cases untreated with antitoxin.

Leukocytosis of the polymorphonuclear type develops in 90 per cent. of cases. It does not correspond to the severity of the disease. When absent the infection is either very severe or light.

2. Nasal diphtheria may usher in the disease or accompany the benign, or more usually, *the severe forms*. The serous and often sanious or bloody nasal discharge excoriates the alæ nasi, lips and cheeks (every "coryza" in which this occurs, must excite alarm); membrane often protrudes. Cervical adenitis is suggestively frequent. The prognosis is grave, and convalescence very tardy. In contrast with this severe form there is a *fibrinous rhinitis* usually subacute or chronic, which is due to the Klebs-Loeffler bacillus (82 per cent.), or the streptococcus (18 per cent.); it is obviously dangerous to other children, since the remarkable absence of constitutional reaction, despite the menacing discharge, does not prevent play or school-going.

3. Laryngeal Diphtheria or Croup.—Most cases are diphtheritic (85 per cent.), though some are streptococcic. Diphtheria involves the throat in 84 per cent. and the larynx in 16 per cent. of cases. *Forms:*

(*a*) Catarrhal laryngitis, due to the diphtheria bacillus, produces symptoms of two or three days' duration; they may be severe, even stenotic. (*b*) *Diphtheritic laryngitis* (*croup*) is descending in 97 per cent. of cases, *i. e.*, secondary to pharyngeal or nasal diphtheria; even in seemingly primary cases there is usually some pharyngeal involvement. The child becomes *hoarse* in the night or early morning, coughs the *hoarse, croupy cough,* and after twelve to twenty-four hours is dyspneic. Aphonia is the rule. The *stridor* becomes menacing and then the enduring croup is surely diphtheritic. The *stenosis is due to membrane* in the epiglottis, true cords, trachea or even in the bronchi. The accessory muscles of respiration come into play, with inspiratory retraction of the thorax and epigastrium, especially in rhachitic children, up-and-down excursion of the larynx, extreme restlessness, and temporary improvement after expectorating the membrane. The stenosis is accentuated by the central, immovable position of the vocal cords, swelling in the subchordal tissue and muscular spasm. The cough may disappear because of decreased reflex bronchial excitability. Fever is usually absent. The pulse increases with augmentation of the stenosis. The *respite following the raising of the membrane is usually transient;* exacerbations occur and finally, without intubation or tracheotomy, dyspnea becomes continuous, with stagnation of secretion and membrane in the tubes, absence of the vesicular murmur, stridor, increasing rapidity and weakness of the pulse, cold, clammy skin—in short, the complete picture of carbon dioxide poisoning and total asphyxia develops.

4. Other Localizations.—(*a*) *Diphtheria of skin* is usually associated with pharyngeal diphtheria. The skin is hard and infiltrated, the secretion corrosive, the contiguous lymphatics involved, and cellulitis or phlegmon may result. Most instances of "wound diphtheria" and the membrane which forms in tracheotomy wounds are usually streptococcic.

(*b*) *Diphtheritic vulvovaginitis* (under 1 per cent.) is dangerous, and usually follows severe pharyngeal diphtheria; it consists of diffuse membrane, painful rhagades, ulcers and foul secretion. A streptococcic form is also encountered.

(*c*) *Diphtheritic ophthalmia* is (i) false or diphtheroid (due to pus cocci) or (ii) genuine or diphtheritic, which occurs in 3 per cent. of cases, chiefly in young children. It may be primary in the eye, or secondary to nasopharyngeal diphtheria.

(*d*) *Diphtheritic otitis* occurs in 6 per cent. of cases (as against 33 per cent. of otitis in scarlatina and nearly 100 per cent. in measles). The diphtheria bacillus travels from the throat to the Eustachian tube or middle ear.

Complications and Sequels.—*The most important are heart weakness, nephritis, and paralysis.*

1. Cardiac Complications.—*Anatomically,* the heart muscle is altered early. It is soft, friable, light brown or yellow, and fatty; it is the occasional seat of endocardial and myocardial hemorrhages, acute interstitial myocarditis and fragmentation or myolysis, which Eppinger, in describing eighteen cases of sudden death, considers the usual cause

of death; degeneration may be found in the bundle of His. Pericarditis, with septic or hemorrhagic effusion, or permanent changes due to endocarditis (in 2 per cent. of fatal cases) or myocarditis are uncommon.

Clinically, the first tone is impure or obscured by a systolic murmur in one-half the cases; the second pulmonic is accentuated, the *rhythm is disturbed* (60 per cent.) and sometimes there is gallop-rhythm and embryocardia. The pulse is rapid and soft but it may be ominously slow, even 14. *Heart-failure is more frequent than in any other infection,* and occurs early in severe or gangrenous types, or only after three to six weeks (in 20 per cent.). It is marked by copious vomiting from vagus neuritis, pain in the chest and epigastrium, cardiac dilatation, pallor, dyspnea, cyanosis and convulsions. Heart failure is explained in various ways (*a*) acute interstitial myocarditis; (*b*) myolysis; (*c*) centric vasomotor paralysis; (*d*) neuritis of the vagus; (*e*) degeneration of the bundle of His; 33 per cent. die.

2. Diphtheritic Nephritis.—(*a*) Albuminuria occurs in 33 per cent. of all and in 100 per cent. of severe cases. (*b*) In *light forms* of nephritis there is some albumin, the specific gravity and quantity of urine are about normal, and there are leukocytes or epithelial cells with indistinct nuclei, the cells being highly refractive and coarsely granular. Blood is rarely found. There are some hyaline and granular casts. These toxemic findings correspond in time and degree to the diphtheritic process in the pharynx. (*c*) The *severe forms* of nephritis occur in severe or septic types and are characterized anatomically by diffuse parenchymatous changes, necrosis, hemorrhage and exudation. Albumin, epithelium, casts and cylindroids are abundant. Hematuria is rare; acetonuria occurs in 65 per cent. of cases. Nephritis rarely produces hydrops or uremia as in scarlatina. It rarely becomes chronic.

3. Diphtheritic paralysis occurs clinically (and experimentally) from toxemia in 20 per cent. of cases. Paralysis rarely develops in patients receiving antitoxin on the first day but most often develops in those injected after the third day, or with insufficient doses. Its frequency and intensity is directly proportional to the severity of the diphtheria.

(*a*) *Early paralysis* occurs in septic cases at the acme. Dysphagia is its most common form. There is danger of inhalation pneumonia, especially in stupid cases. Most cases die.

(*b*) *Postdiphtheritic Paralysis.*—This, the typical variety, occurs after two or three weeks and usually follows throat lesions since laryngeal cases are likely to die early; all throat infections leading to paralysis are probably diphtheritic. The most common paralysis is that of the palate, which was described by Ætius in the sixth century, and causes dysphagia, anesthesia of the throat and nasal speech. The pupils widen, and react to light but not to accommodation. The arms frequently escape. Ataxia may be the first symptom. The reflexes disappear early and the muscles react slowly, if at all, to the faradic current, although the reaction of degeneration by no means runs parallel to the degree of loss of voluntary motion. The muscles waste, the palsied limbs are sometimes painful and the muscles and nerve trunks are often tender. The trunk, bladder, and rectum are sometimes involved. The sensory

nerves suffer less, sensation being largely retained above the knees. Sensory disturbance may be noted in the rectal, genital and vesical twigs. The cranial nerves (strabismus, ptosis, diplopia), the phrenic nerve and vagus may be involved; fearful abdominal pains with threatening collapse attributable to sympathetic involvement may be noted. Anatomically, disintegration of the medullary substance, multiplication of nuclei in Schwann's sheath, granular degeneration, and even total loss of the axis-cylinder are present, the changes being almost totally parenchymatous; neuritis (actual nerve inflammation) is seen almost wholly in the palate, due to local invasion from the inflamed throat. The patient recovers in two or three weeks if the pharynx alone is involved, and in three to eight months if the limbs are affected. Death results in 18 per cent. of cases, chiefly from vagus involvement.

(*c*) *Organic Central Changes.*—Accidental findings are cerebral hemorrhage, embolism and acute encephalitis. The literature contains 85 instances of hemiplegia (Rolleston, 1905). The Babinski reflex, found in 20 per cent. of severer cases, is due to toxemic action on the pyramidal tracts. Degenerative cerebrospinal lesions not infrequently occur with multiple neuritis.

4. Respiratory Complications.—The bronchi and trachea exhibit membrane in 50 per cent. of fatal cases. *Bronchopneumonia* is present in 65 per cent. of fatal cases, caused by the Klebs-Loeffler bacillus alone, or by the pus or pneumonia cocci, etc. Anatomically, some cases are due to downward extension of the bronchitis; some are hematogenous; others are due to inhalation, and still others, seen after intubation or tracheotomy, are elevated and granular. Increase of pulse and respiration-rate, local consolidation and accession of fever are observed. Serous, hemorrhagic or purulent pleurisy is seldom encountered.

5. Alimentary Tract.—Fissures, sordes, membrane or ulcers on the lips, suppuration in the tonsils or retropharyngeal space and gangrene sometimes occur. In septic cases the esophagus may be occluded by membrane. Epigastric pain and vomiting are chiefly cardiac; death is usual when vomiting develops late in the disease; the rare hemorrhage and membrane formation escape clinical recognition. Constipation is usual; in septic forms, thick, foul or dysenteric evacuations may be exceptionally observed; there may be membrane formation and swelling of Peyer's patches. The liver is swollen from fatty or cloudy degeneration or from congestion, in which latter case it also becomes painful. Splenic tumor is found in septic types especially.

6. Skin.—The skin is usually normal, except for the febrile hyperemia, cardiac pallor, croup-cyanosis or septic grayness. Herpes, in 4 per cent., petechiæ, symmetrical gangrene (of which 9 cases exist in the literature), and septic dermatomyositis usually occur with adenitis, nephritis or ulcerative endocarditis. Scarlatina or measles may complicate diphtheria.

7. Glands.—The anterior cervical glands are swollen and painful. In the severest forms periadenitis follows, with diffuse cellulitis (angina Ludovici), section of which evacuates serum, seldom pus. Glandular and articular changes are less frequent than in scarlatina. Muscular

degeneration, interstitial infiltration and fragmentation are observed in the heart and diaphragm.

Diagnosis.—*A thick, adherent membrane, which is not removable* without hemorrhage, with subjacent ulceration (Bretonneau) and enlarged glands, is characteristic. *A positive bacteriological diagnosis* is important in cases with membrane, diffuse reddening and tonsillitis, yet four negative examinations are necessary to exclude diphtheria positively. Statistics show positive findings in 92 to 98 per cent. of cases in which the clinical diagnosis of diphtheria has been made. The nose, nasopharynx and pharynx must be examined in every sick child and adult. In suspicious cases antitoxin should be given at once.

The health departments in large cities provide cultures, stations at which cultures and antitoxin may be obtained, and central and subsidiary laboratories for diagnosis. In smaller towns communal laboratories can be maintained easily. The Chicago Health Department has an able corps of physicians for free treatment, consultation and intubation. It is easy to obtain a smear from the suspicious focus, to rub it on the serum without breaking its surface, and to put the tin box containing it under the axilla until the brood-oven can be reached. In half a day characteristic cultures are obtained. Direct smears very often show the bacillus. In laryngeal diphtheria the bacillus is usually found on the pharynx, but not on the tonsils. Repeatedly negative findings are of value in the *pseudodiphtheritic* pharyngitis, rhinitis and laryngitis, *e. g.*, membranes due to strepto-, staphylo-, and pneumococci, aphthous growth, syphilis, etc. The *scarlatinal* membrane (*q. v.*). In *Vincent's angina* (*q. v.*) described by Vincent (1898), the whitish-yellow or grayish-brown membrane may be confused with diphtheria and syphilis and is caused by the symbiosis of two otherwise innocuous parasites, the Bacillus fusiformis, and the Spirocheta darticola.

Course and Prognosis.—Aside from complications, the average course is one or two weeks. It is more favorable in diphtheria of the tonsils than of the nose. The prognosis concerns the toxemia, mechanical obstructions, as of the larynx, mixed infection, and the therapy. (*a*) Without serotherapy the danger is great, since light infections may intensify or extend. (*b*) Epidemics vary in virulence, 30 to 50 per cent. mortality having been observed twenty years ago. (*c*) In the first year the mortality is 77 per cent., and sinks with each year until it is, at ten years, 20 per cent.; after ten, 3 per cent.; after thirty and forty, 2.5 per cent. (*d*) Other factors are debility, adenitis and croup; the general diphtheritic infection or septic forms; renal complications; rapid and weak or slow pulse; ecchymoses and late vomiting; and paralysis. (*e*) In 946 fatalities studied by White and Smith, about 50 per cent. died of bronchopneumonia, usually after intubation or tracheotomy; 25 per cent. died of cardiac complications, and 25 per cent. of early severe toxemia, though some succumbed to asphyxia or late paralysis. (*f*) Coincidence with other infections is ominous, such as measles, in which the exanthem may become hemorrhagic, and a lethal respiratory catarrh usually results. Diphtheria belongs to the recurrent infections.

Treatment.—1. PROPHYLAXIS begins with the case in hand. In Boston the isolation hospital has reduced the mortality of diphtheria to one-sixth of its former rate. All suspicious throat cases should be isolated pending their cultural evolution; other children in the family should be kept from playmates and school. The patient is isolated until the throat gives two negative cultures, successively, requiring three weeks. The physician may protect himself by examining the throat through a piece of glass, and avoid carrying the germs, by a surgical gown and washing face and hands with bichloride solution. In fatal cases the body should be wrapped in a sheet saturated with bichloride, and buried privately in a tightly closed casket. Those exposed to infection may receive antitoxin, and should gargle the throat often with bichloride ($\frac{1}{5000}$). The clothing, blankets, rugs, etc., must be steamed. After convalescence, the walls of the room should be rubbed with bread which is then burned, the woodwork washed with 2 per cent. phenol solution or bichloride and the room fumigated (page 258). Under prophylaxis may be included the building up of the physiological resistance and the treatment of chronic catarrh of the nose, throat and bronchi. "Carriers" must be isolated; Hewlett reports success from vaccines. Schiötz advocated the use of a staphylococcus spray; a fresh bouillon culture is sprayed into the nose and throat, until they are moist; recent reports are encouraging. V. Behring recently reports a new prophylactic against diphtheria —a mixture of very strong diphtheria toxin and antitoxin combined in such a proportion that the mixture shows only a slight surplus of the toxin; the method is a result of experiments which shows that it is impossible to produce in a test-tube complete neutralization of the diphtheria toxin.

2. LOCAL TREATMENT.—Though local treatment of the throat is less important than before the days of serotherapy, the antitoxin does not always kill the bacilli, and does not affect the associated streptococci. Caustics are never indicated. In children such mild antiseptics as 3 per cent. boric solution and 3 to 10 per cent. potassium permanganate solution, should be used lest they be swallowed. The child is held in the nurse's arms with its arms and body firmly wrapped in a sheet. The mouth is opened by closing the nostrils, and a piece of cork is inserted between the teeth. The throat is then swabbed. It is a harsh procedure and the benefit is apparently more than offset by the resultant exhaustion. In adults 1 to 1000 bichloride, 25 per cent. hydrogen peroxide, 1 per cent. carbolic solution, or iodoform salve, may be used, or the somewhat irritating Loeffler's solution, which is as follows:

℞—Mentholis	10 parts
Toluolis	30 parts
Liq. ferri chloridi	4 parts
Alcoholis absoluti	60 parts

It should be kept corked in a dark bottle. Local injury, opening up new wound surfaces for infection, must be avoided. In nasal irrigation the tube is held horizontally, so that fluid may return by the other nostril. A kettle should be kept steaming in the room.

3. Serotherapy.—Behring (1890) borrowed Ehrlich's results with ricin and abrin to establish the units of immunization, and used the diphtheria antitoxin on diphtheria patients (1893). Antitoxin is obtained from horses which have been rendered immune by the injection of progressively increasing doses of diphtheria bacilli. According to Ehrlich's "side-chain" theory, toxins combine chemically with the cells of the body, just as food unites with cells in ordinary metabolism. Parts of the cells, which combine with toxins, are called receptors. The stimulation or injury excited by toxins causes in the process of repair, an overproduction of receptors, which are thrown free as antibodies, into the circulation. Thus the horse serum unites with toxins and spares the cells in human diphtheritic toxemia. Immunity may result from the body cells being devoid of receptors or an excess of receptors in the blood may unite with the toxins and spare the cells.

1. *Technique.*—The glass syringe is the best; it should be cleaned after each injection, with water to remove the serum, and then washed with alcohol, which should be thoroughly removed lest it coagulate the antitoxin at the next injection. With ordinary surgical antisepsis, inject the antitoxin into the muscular tissue of the thigh in adults, and into the interscapular region in children. Intravenous injections act in 16 hours, intramuscular in 24 hours, and subcutaneous in 72 hours.

2. As we cannot measure the toxins the *dosage* is empirical, depending on the localization, age, severity of the disease and the results obtained (the discharge should lessen, the membrane exfoliate, the fever fall, and toxemia decrease). After eight hours, three times as much is necessary as at the beginning; after twenty-four to thirty-six hours, eight times as much. The author prefers *an initial dosage of* 5,000 *units to insure results and avoid frequent repetition.* The unit is 1 c.c., which counteracts ten times the minimum dose of diphtheria poison fatal to a 300 gm. guinea-pig. The decreased bulk of present preparations allows a larger dosage. In severe cases twice the above amount should be given. McCallum emphasized the importance of full doses, 10,000, 30,000 or 50,000 units in desperate cases. Sutherlin gave a total of 498,000 units in one case. The present practice reverts to smaller doses, 1200 to 2000 in mild, and 3000 to 6000 in severer infections.

3. *Effects.*—(*a*) The mucosa becomes redder, and the membrane exfoliates in one to three days, with decrease in glandular swelling; (*b*) The fever falls; antitoxin is given until the fever falls. (*c*) The pulse and hyperleukocytosis decrease. Light cases rarely become severe, and non-septic rarely become septic. Paralysis and heart failure are less frequent with early serotherapy; many patients now live who previously died before developing paralysis. (*d*) The mortality is reduced from 40 to 15 per cent. (Siegert's collection 42,000 cases); to 10 per cent. (Wenner, 9.8 per cent., 132,500 cases, American cities); or to 6.7 per cent. (Chicago Health Department). According to Bayeux, the mortality in 230,000 diphtheria patients was 55 per cent. before, and 16 per cent. after, the introduction of antitoxin. The fatal cases and severe complications are lessened in direct ratio as the antitoxin is given early; if on first day of disease, less than 1 per cent.; second day, 2 per

cent.; third day, 8 to 10 per cent.; fourth day, 14 per cent. Failure to use antitoxin is criminal neglect. (*e*) Antitoxin is a prophylactic, and should be given in special instances, *e. g.*, to weak or very young children (in smaller doses); this passive immunity lasted for two or three weeks in all but 0.7 per cent. of 1000 cases (Zuppinger). (*f*) The sequels are: (i) Abscess formation; (ii) nephritis, always due, however, to the diphtheria itself; (iii) *serum disease and anaphylaxis.* In one or two weeks fever and exanthems appear in 33 per cent. of cases—urticarial mostly, but also morbilliform, scarlatiniform and petechial. In 3 to 4 per cent. there is also acute pain and swelling in the joints or tendons. Another type comes on more rapidly (12 hours to 6 days), the "accelerated reaction"—with the above symptoms plus vomiting, albuminuria, prostration, and weak heart. A third type appears at once, the "immediate reaction," with high fever, convulsions, dyspnea, cyanosis, vasomotor paralysis, and rigors. In a fourth form, sudden death ensues, of which some 35 instances are on record (in all but 2 instances, death followed the first injection); most deaths occurred in persons of asthmatic tendencies or in those experiencing discomfort in the neighborhood of horses. This condition is termed serum disease, hypersusceptibility, anaphylaxis, allergy, etc. In its varying degrees it is due to hypersusceptibility of the tissues to a specific foreign proteid; thus, the first dose of antitoxin causes a gradual increase of the antibodies which have the properties of a ferment; if a second injection be given 8 to 10 days later, the antibodies (now increased) acting like a ferment, produce a rapid disintegration of the proteids, with liberation of toxic substances, causing the anaphylaxis, which is closely related to the immunity problem. Auer and Lewis proved that the lungs, in fatal cases, are enormously distended, due to a tetanic muscular contraction of the finer bronchioles; the heart is also paralyzed. How may these disasters be avoided? First: There is greater danger of anaphylaxis in immunizing serotherapy than in its use for actual diphtheritic infection; the writer has seen high fever, renal suppression and the most alarming vasomotor instability (pallor, cyanosis and scarlet flushing occurring in succession). Immunizing serotherapy must not be indiscriminate. (Schick uses 0.1 c.c. of toxin solution intradermically and finds that subjects not immune to diphtheria develop a reaction similar to v. Pirquet's in tuberculosis). Second: One large dose is less dangerous than doses repeated at frequent intervals. Third: A small dose (0.5 c.c.) is given first and if there is no reaction, a second full injection is given. Fourth: The second or later injections should be from some other animal than the horse, *e. g.*, sheep antitoxin, etc. Fifth: Give the smallest possible bulk of serum. Sixth: Use atropine before the second dose, and if anaphylactic symptoms have developed, use salt solution and adrenalin (1 to 40,000); digitalis is injurious. (iv) Seibert collected 18 cases of tetanus following antitoxin.

4. Symptomatic Treatment.—A rich *fluid diet* of milk, eggs, etc., is given. In marked dysphagia, rectal feeding is indicated. Forced feeding by the nasal catheter injures the throat.

Sepsis.—Alcohol, saline enemata, strychnine and iron should be given; all measures fail if tens of thousands of units do not give relief.

Paralysis.—Electricity is of no avail; massage, baths and strychnine are useful in the later stages.

Heart Failure.—The ice-bag is indicated for stormy heart action; strychnine, coffee and camphor should be given (dosage *v.* page 269) for severe abdominal pain and vomiting, usually evidences of cardiac failure, and champagne with menthol and carbolic acid, and twenty drops each of adrenalin and tr. belladonnæ in nutrient enemata. The heart must be watched far into convalescence.

Nephritis.—Alcohol, tincture of iron, beef-tea and eggs are avoided; milk diet, baths, laxatives and care of the skin are indicated.

Local Diphtheria.—Two per cent. nitrate of silver in balsam of Peru affords relief in vaginitis; ice applications, atropine and silver in ophthalmia; local diphtheria necessitates antitoxin.

Croup.—Steam tents and ice locally are applied; the child's head may be held over a pail of water into which live coals are thrown; emetics may be administered. Retraction of the chest walls, suffocation and cyanosis necessitate intubation or tracheotomy.

O'Dwyer's Intubation.—Advantages.—It is bloodless, easier and quicker than tracheotomy, and is less objectionable to the family.

Disadvantages.—Membrane may rise beneath the tube and the tube sometimes produces false passages, necrosis, perichondritis and stricture. The tube is usually replaced two, three or four times, and rarely need remain more than five days when the serum is used. It is removed by the extubator or pressure.

Tracheotomy is indicated by heart weakness, asphyxia, much secretion, or gangrenous, diffuse or septic infiltration. The after-treatment requires a moist atmosphere and daily cleaning of the tracheotomy tube. The tube is not permanently removed until all stenotic symptoms and all plaques disappear, which, with serotherapy, may be done on the fourth day. The prognosis depends upon possible wound-infection with erysipelas or diphtheria, phlegmon, thrombophlebitis, mediastinal suppuration, ulceration of trachea and inhalation pneumonia. Rapid rise of temperature is ominous. Antitoxin reduced the mortality from 66 per cent. to 25 per cent.

CEREBROSPINAL FEVER.

Definition.—An acute infection, caused by the meningococcus, characterized anatomically by fibrinopurulent exudation in the meninges and marked clinically by headache, rigidity of the spine, hyperesthesia, vomiting, Kernig's sign and often by paralyses.

In 1805 Vieusseux described epidemic meningitis; in 1806 Danielson and Mann, in New England, made the first autopsies.

Bacteriology.—The *Diplococcus intracellularis meningitidis* of Weichselbaum or meningococcus resembles the pneumococcus, though wider and less lanceolate. It lies in pairs or tetrads and resembles two coffee grains with their flat sides apposed. It is usually enclosed in the polymorphonuclear leukocytes. Unlike the pneumococcus, it has no capsule, although one may develop in serum cultures. It decolorizes by Gram's method. It develops best on agar or blood serum as white, viscid, shin-

ing colonies. Subcutaneous inoculations are usually negative, therein differing from the pneumococcus, but it produces inoculation-meningitis and peritonitis. The meningococcus is found in the nose, from which it is thought to invade the brain, in cases with and without meningitis. Von Lingelstein found it in the nasopharynx in 94 per cent. of cases in the Silesian epidemic and every time in 68 personal observations. He asserts that the disease is communicable only by direct contact with the mucus from the nasopharynx of patients or of those in contact with them. Its presence has been demonstrated in the blood in 25 per cent. of cases by Elser; kidneys, herpetic vesicles, conjunctiva, joints, spleen, ear, tonsils, lymphatic nodes, pleura, lung, heart and more rarely in the sputum and urine; it travels by way of the lymph or bloodvessels. Meningococcus septicemia without meningitis is recorded in four instances (Bovaird).

Predisposing Etiology.—Fifteen per cent. occurs under two years of age and 60 per cent. between the second and fifteenth years; it is often epidemic in barracks, prisons and tenements or closely follows the movements of armies. Its contagiousness is proven. As many as seven members of a family have been successively stricken. Between epidemics, 1.7 per cent. of persons are said to be "carriers," and during epidemics, 2.5 per cent.; Flügge estimates that the healthy carriers outnumber by tenfold those sick with the disease. Fortunately the coccus has little resisting powers outside the body. Most cases are seen in the late winter and early spring; 3455 deaths occurred in New York in 1904 and 1905. In a Silesian epidemic in 1905 there was a mortality of 57.6 per cent. in 3102 cases. Councilman collected five instances of recurrence. The disease is also seen in the horse and goat.

Pathology.—The findings are those of the suppurative form, viz., those of an acute fibrinopurulent, or less often a seropurulent leptomeningitis. The exudate is rich along the fissures, fossa of Sylvius and at the base about the chiasm or the surface of the pons and cerebellum. The inflammation follows the lymphatics and vessels penetrating the brain and cord, resulting in encephalitic or myelitic foci, small abscesses, parenchymatous and interstitial changes. Flexner and Barker observed rows of cells "like miniature tubercles," two to eight times the size of the leukocytes. Hydrops ventriculorum is frequent in cases of long standing. The cerebral dura is seldom, and the spinal dura often, involved. In other organs the changes are those of acute infection; the muscles are brownish-red, dry and degenerated, there is parenchymatous degeneration in the heart and kidneys, and sometimes acute splenic tumor, arthritis, pneumonic foci or swollen glands in which the coccus may be found.

Symptoms.—The *incubation* lasts from a few hours to three to eleven days. Prodromes are vague, as malaise, headache or coryza. The onset may be gradual or sudden, sometimes with a chill and usually with fever. The course is attended by fever and brain irritation; an almost constant triad of symptoms, *headache, rigid neck and hyperesthesia;* vomiting, mental, motor and sensory symptoms, and in fatal cases death results from paralysis, convulsions or coma.

1. General Nervous Symptoms.—(*a*) *Headache* is very rarely absent except in very young children. It is early, most severe and generalized. It is constant, with exacerbations, and in children provokes the short plaintive, clear meningeal cry, *cri hydrencéphalique* (Coindet, 1817). The headache is throbbing or lancinating, even under narcotics, and persists even after delirium and coma have set in. Its cause may be compression of the dura, inflammation of the nerve trunks, or ventricular exudation. (*b*) *Hyperesthesia* or hyperalgesia affects the special senses, causing intolerance of light and noise, or general sensation—affecting the arms most, the trunk less and the legs least. It is rarely absent, always suggestive, and due to root neuritis. (*c*) *Rigidity of the neck and spine* is caused by inflammation of the nerve roots. The *head is retracted*, sometimes so severely that the occiput lies between the shoulders, producing dysphagia and decubitus; marked *opisthotonos* may be present. The entire body may be lifted by the head. The most common attitude is the lateral, with the head retracted, the arms flexed and the knees drawn up. Flexures in the ankles, knees and hips result when an attempt is made to flex the neck (Brudzinski's "neck sign"). Closely associated and caused in the same way is severe *spinal pain with tenderness*, chiefly in the neck and loins. It may radiate to the trunk and in 33 per cent. of cases to the extremities. (*d*) *Contractures of the limbs* are caused by root inflammation, increased pressure, or lesions in the pyramidal tracts. *Kernig's sign* comes under this head; when the patient sits or the thighs are at right angles with the trunk, in 85 per cent. of all meningitis cases it is impossible to extend the knees (Fig. 11). The legs and thighs are flexed (flexor contracture) and the knees cannot be pressed down when the patient is sitting, but this is possible when he is lying. It indicates involvement of the spinal meninges, pyramidal tracts or posterior sensory roots. It lasts well into convalescence, whence, as Netter states, "a retrospective diagnosis may be made." It occurs in other conditions, but in infancy is generally meningitic. (*e*) *Mental symptoms* occur early, especially in children and include unrest, insomnia, delirium sometimes with periods of normal intelligence, and followed by incomplete coma. (*f*) *Vomiting* in the initial stage is due to vagus irritation; later it results from ventricular hydrops or increased cere-

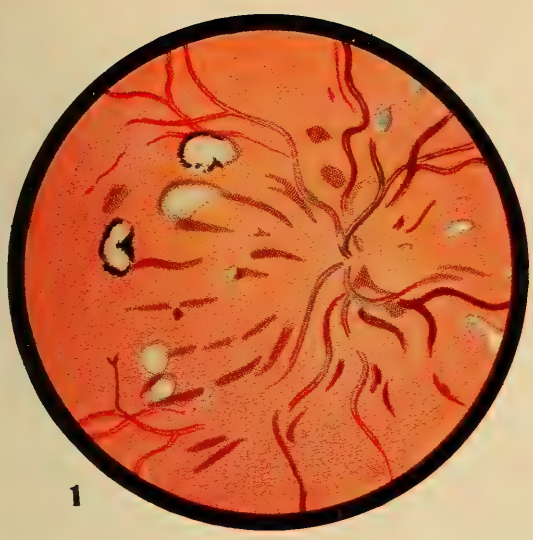
Fig. 11.—Kernig's sign, showing retraction of head and back and prominence of knees in the erect posture, *i. e.*, the impossibility of extension of knees. (Dieulafoy.)

brospinal pressure. It is of the cerebral type, *i. e.*, it occurs without nausea or relation to eating, is often projectile and is not amenable to gastric therapy. Vomiting is more typical and common in children. (*g*) The *pulse* at first is increased, perhaps disproportionately, considering the fever. Sometimes it slows later because of brain pressure, but ultimately it is fast again. It may be irregular in rhythm and oscillating in rate from 84 to 144 within a minute. Blood-pressure increases in proportion as the disease is severe. (*h*) *Respiration* is irregular in 20 per cent. of cases; Cheyne-Stokes breathing develops toward the end in 53 per cent. (Conner). Deep breathing with apneic pauses and undulatory respiration (Biot's breathing) are less common than in the tuberculous type. (*i*) *Convulsions* are most common in the young, but are ambiguous from their frequent occurrence in other diseases of childhood.

2. Focal Nervous Symptoms.—These are partly irritative and partly paralytic. (*a*) *The cranial nerves* are most involved in basal localization, while the limbs are most affected in that of the convexity. Optic neuritis is frequent, if the disease lasts over four days. The disk is swollen and its margin is "washed"; hemorrhage and choking are uncommon. (See Plate IV, Fig. 4.) The pupils at first are usually small, often unequal, and sometimes variable. Later they widen spontaneously or on movement of the rigid neck and irritation of the skin, but do not react to light. Ptosis, diplopia, nystagmus and strabismus are frequent and may vary from time to time. Complete paralysis of the third nerve is not common. Involvement of the fifth nerve is unusual, although trismus, grinding of the teeth and trigeminal neuralgia have been noted. Involvement of the facial nerve ranks next to that of the third nerve; basal paralysis is usually total; cortical palsy is of the cerebral type, and its upper third escapes. The facial expression indicates suffering, the brows are slightly elevated and the forehead is corrugated. The auditory nerve is often bathed in pus, and extension may induce hemorrhagic inflammation in the labyrinth. The tongue on protrusion may deviate to one side; dysarthria or dysphagia is sometimes seen. (*b*) Paralysis of the limbs is not frequent. Hemiplegia is rarely complete, occurs with or without participation of the tongue and face, and is caused by involvement of the cortex or pyramidal tracts. It is frequently accompanied by rigidity of the limbs and aphasia. It is most frequent in children and may be temporary or permanent. The less frequent monoplegia most often concerns the face or arm. Choreiform or athetotic movements, twitchings and tremors are occasional. Convulsions are frequently Jacksonian in type. The tendon reflexes may be increased at first and are often decreased or abolished late in the disease. The skin reflexes are variable.

3. General and Somatic Symptoms.—(*a*) The almost invariable *fever* is usually irregular or remittent, follows no definite cycle, and lysis is usual in cases which recover. A temperature of 107° may be registered before death. (*b*) The *blood* shows leukocytosis, which is early and constant; the white cells may number 25,000 to 40,000; the lymphocytes increase during convalescence; the meningococcus is found in 25 per cent. of cases. (*c*) The *abdomen* is often retracted and scaphoid,

PLATE IV



Retinal Findings in the More Important Diseases.

1. Retinitis diabetica. 2. Atrophy (and pigmentation) of retina and optic nerve. 3. Choked disk in brain tumor. 4. Optic neuritis in meningitis. 5. Medullated nerve fibers (normal).

although less so than in the tuberculous type; it is due to retraction of the abdominal walls or possibly of the intestines from irritation of the vagus. (*d*) The *spleen* is often palpable. (*e*) The *urine* is febrile, albuminous (33 per cent.) and may show peptonuria, hematuria in malignant cases, and rarely glycosuria or polyuria. (*f*) *Skin:* The *tâche cérébrale* has no differentiating value, for it occurs in many other conditions. Eruptions are more common in epidemic than in sporadic cases. *Herpes* varies with the epidemic (40 to even 90 per cent.); it is most frequent on the face, but may develop anywhere; it spreads more rapidly than in any other disease; in one case the author saw it extend from the lip to the ear, neck, chest and shoulder. *Purpura* ("*spotted fever*") occurs in severe types.

Clinical Forms.—(*a*) The ordinary *acute* type in which the general nervous symptoms enumerated are followed by focal nervous symptoms, somatic symptoms, death in coma, or slow recovery; (*b*) the *foudroyant form,* with violent onset, rapid course and death in three to thirty-six hours, with apoplectic symptoms, purpura, high fever and early coma; (*c*) the *abortive form,* with low fever and symptoms so mild that the disease is recognized only during an epidemic; and (*d*) *Heubner's chronic form,* which lasts two to six months, with marasmus and recurrent fever.

Complications and Sequels.—Pneumonia, tonsillitis, pleurisy, parotitis, endo- and pericarditis, arthritis and nephritis are the most common. Pneumonia is usually pneumococcic, but Councilman found the meningococcus in isolated foci in the lung. The arthritis is poly- or monoarticular, simple or suppurative, and may heal even when suppurative; it occurs in 25 per cent. Mental alteration, paralysis, contractures, obstinate headache, optic atrophy, deafness or Ménière's syndrome, chronic hydrocephalus, vomiting and convulsions may remain after recovery. In a Heidelberg epidemic, Moos found deaf-mutism in 60 per cent., deafness in 31 per cent. and ataxia in 50 per cent. of the cases.

Prognosis.—The mortality averages 37 per cent., but it varies with the epidemic, and may reach 75 per cent. Fifty per cent. of deaths occur in the first five days. Recovery is exceptional after deep coma, repeated convulsions, high fever or vagus paralysis. Obstinate vomiting, disturbed respiration, sudden drop of temperature and inanition are ominous signs.

Diagnosis.—The *first step* is the diagnosis of meningitis, and the *second,* its variety, cause and localization.

Cardinal Symptoms.—(*a*) The *early triad* of headache, retracted rigid neck and hyperesthesia and (*b*) *general symptoms,* as vomiting, abdominal retraction, pulse and respiratory changes, fever or convulsions, are important in their grouping. (*c*) *Lumbar puncture* (Quincke, 1890) is of great diagnostic value. Its *technique* is as follows: The patient sits or lies on the side with the knees drawn up and the body bent as far forward as spinal rigidity allows; an aspirating needle is introduced in the median line in children, and a quarter to half an inch to one side in adults, between the third and fourth lumbar vertebræ and is pushed forward toward the median line and a little upward, 1.5 inches deep in

children and 1.5 to 2.5 inches in adults, until it passes into the canal; 15 to 30 c.c. should be withdrawn. *Results:* (i) Normally the pressure with the patient in the lateral position, equals 100 to 150 mm. of water and the escaping fluid only trickles out. In meningitis, the *pressure is greatly increased* (200 to 700 mm.) and the fluid often spurts out. (ii) The normal fluid is clear, has a specific gravity of 1.007–09 and contains little albumin ($\frac{1}{2}$ to 1 gm. per mille). Though the fluid may be clear in all forms of meningitis, it is usually clear in the serous form, but *cloudy, flocculent* or sometimes bloody in other forms; in the tuberculous variety it is usually clear or slightly yellow with a whitish clot forming on standing; in the epidemic form, it may be turbid or clear, with a yellowish clot; in the purulent type it is opaque, and in rare cases pure pus. The specific gravity is about 1.010 in the serous, 1.011 in the tuberculous, and 1.015 in the purulent or epidemic type. The percentage of albumin is small in the serous, 2 per mille in the tuberculous, and 3 or even 9 gm. per mille in the suppurative forms. The Nonne, Noguchi and Ross-Jones globulin tests are positive. (iii) *Bacteriologically* in the purulent type the streptococcus, staphylococcus, pneumococcus, typhoid and colon bacilli are found; in the epidemic variety, the meningococcus was positive in 100 per cent. in the last New York epidemic; in the tuberculous type, tubercle bacilli exist in 80 to 100 per cent. in the small clot, which forms after standing. (iv) The *cellular morphology* is a valuable aid. In purulent and epidemic forms the leukocytes are largely polymorphonuclear; in the tuberculous form the lymphocytes predominate (see Cytodiagnosis, Pleurisy). (*d*) *Local signs* usually follow the general signs, and are more important, but their late development lessens their diagnostic value.

Diagnosis of the Variety (*v. i.*).—Cases of tuberculous meningitis sometimes increase when the epidemic form prevails. Sporadic cases may be indistinguishable from primary pneumococcic meningitis, save by lumbar puncture.

EPIDEMIC FORM.	TUBERCULOUS.	SUPPURATIVE.
Onset: Sudden, with few prodromes.	Longer prodromes; family history; other evidences of tuberculosis.	Secondary to ear disease, acute infections, pneumonia, etc.
Fever: Fairly high; pulse more rapid.	Less; inversed type frequent.	Especially high, with chills or pyemia.
Eruptions: Herpes and purpura very common.	Rare.	Rare—polymorphous septic rashes.
Leukocytosis: Regular and marked.	Exceptional.	Frequent.
Rigidity of neck: Most frequent.	Spinal symptoms less frequent.	Sometimes absent (pneumococcic).
Other symptoms: Peptonuria.	Choroidal tubercles.	Peptonuria.
Course: Rapid — more recoveries.	Subacute, slower, fatal.	More acute, very rarely recover.
Lumbar puncture: Meningococcus; leukocytes, polymorphonuclear.	Tubercle bacilli; mononuclear.	Pyogenic cocci, pneumococcus; polymorphonuclear.

Diagnosis of Location.—(*a*) *Convexity* localization is characterized by delirium, Jacksonian convulsions, mono- or hemiplegia, less cranial

nerve paralysis and less optic neuritis; (*b*) basal localization, by more frequent cranial nerve neuritis and optic neuritis; (*c*) the *ventricular* form rarely involves the cranial nerves.

Differentiation.—1. From Nervous Toxemia.—Jenner pointed out that toxemic headache ceases when delirium begins in acute infections. Meningitis without focal symptoms may resemble uremia (*q. v.*), but the albuminuric retinitis, cardiovascular changes, albuminuria and cylindruria are usually distinctive. Acute *inflammation of the labyrinth* may mimic meningitis, for it sometimes produces headache, vomiting, fever, convulsions, stiff neck and even optic neuritis; if labyrinthitis involves the meninges, the facial nerve is likely to be affected. Such conditions resembling meningitis Dupré called *meningismus*, and Bouchut pseudomeningitis (see pages 34, 46, 47).

2. From Brain Disease.—See *syphilitic* and *tuberculous meningitis*, *brain abscess*, cerebral hemorrhage and encephalitis.

3. From Hysteria.—Hysteria is marked by psychical alteration, limitation of the field of vision, anesthesia and other stigmata. Hysterical strabismus is always spastic and convergent, and is often associated with small pupils; meningitic strabismus is often divergent (always a sign of organic disease), and the pupils are unequal.

Treatment.—Prophylaxis; the nose should be douched with a weak resorcin solution.

Flexner's and Jobling's Serum has given 70 per cent. of recoveries in 1300 cases; given on first to third day the mortality was 18 per cent.; fourth to seventh, 27 per cent.; after the seventh day, 37 per cent. The results are (*a*) the meningococci become more intracellular, and degenerate; (*b*) the exudate becomes less turbid or purulent; (*c*) leukocytosis decreases rapidly; and (*d*) complications are few and recovery is usually complete. The mortality under 1 year was 50 per cent.; 1–2 years, 31 per cent.; 2–5 years, 28 per cent.; 5–10 years, 15 per cent.; 10–20 years, 37 per cent. The serum is bactericidal rather than antitoxic, although some antibodies exist in the serum. In some failures, the parameningococcus should be considered; it was discovered by Dopter and is distinguished by the fact that it does not agglutinate in antimeningococcus serum. *Technique:* Lumbar puncture is performed and from the trickling fluid, cover slips, cultures and a leukocyte count are made. If possible, 30 to 45 c.c. are withdrawn; an aspirating syringe is then attached and injects an equal amount of serum, warmed to body temperature by standing in warm water. Injections are repeated daily for 4 days, and if improvement follows, injections are discontinued for 4 days, and then repeated. Now and then, increased intradural pressure is evidenced by disturbed, even stertorous breathing, cyanosis, cold sweats and fall of blood-pressure. Sophian, who has given 1500 injections without accident, employs a gravity method rather than a syringe, which allows of more ready control of the amount of serum injected; he takes the blood-pressure during the injection. Anaphylactic death has been reported. When intraventricular effusions occur, the ventricles should be tapped and filled with the serum.

The patient should be kept quiet in a dark room, the head elevated

without flexion, an ice-bag applied to the head and spine, or an ice-pillow used. *Nutrition* is maintained by feeding with the nasal tube or by nutrient enemata, if retraction of the neck causes dysphagia. The *bowels* are mildly relaxed, and the bladder is watched. *Vomiting* is treated as in acute gastritis (*q. v.*), but therapy is usually unavailing, for the cause is cerebral irritation; of drugs the bromides are in general most serviceable. Feeding by the rectum, sucking of ice, and the remedies indicated for the headache give the greatest relief. Counter-irritation by blisters or cautery is dangerous because the skin sloughs readily. *Headache*, restlessness and convulsions necessitate the hypodermic use of morphine or the tincture of deodorized opium by mouth with atropine; no other analgesic is equally potent. Chloral is used for convulsions and unrest. *Warm baths* are considered almost specific by Netter. *Stimulation* is indicated when the heart weakens. *Resorbents*, as iodides and mercurial inunctions, are inefficacious although they seem to modify serous meningitis. Crowe recommends *urotropin*, ℥ss–j daily; formalin is found in the spinal fluid 30 minutes after ingestion.

ACUTE POLIOMYELITIS.

Definition.—An acute general infection, characterized by (i) diffuse cerebrospinal lesions, especially in the anterior horns (acute anterior poliomyelitis), with sequential acute, flaccid paralysis, wasting of the muscles, loss of reflexes, reaction of degeneration and contractures; (ii) lymphoid hyperplasia in the lymph glands, spleen and intestine; and (iii) parenchymatous changes in the lungs, liver and kidneys.

History.—Infantile paralysis, acute poliomyelitis, atrophic paralysis, was first described clinically by Heine (1840). The famous contribution of Charcot and Joffroy (1870) referred the trophic relation of the special cells involved to the atrophy of the nerves and muscles supplied and stated that the disease was due to primary inflammation of the cells themselves. The first epidemic in America occurred in Louisiana, in 1841. Medin described it definitely as an infectious disease, the epidemic character of which was fully considered by Wickman in his monograph. Among recent investigations are those of Landsteiner and Popper, who inoculated the disease into rabbits, followed by the brilliant work of Flexner and Lewis, Landsteiner and Levaditi, etc. In the United States, Boston and New York are the chief centres, probably invaded from Northeastern Europe. In New York there were 2500 cases in 1907, and in 1910 over 8000 in the United States. It exists epidemically and sporadically. In some statistics it constitutes even 8 per cent. of the diseases of childhood.

Etiology.—I. The virus, which passes the finest filters, is *found in the brain and cord*, tonsils, nasopharynx, lymphatic and salivary glands, blood, cerebrospinal fluid, stomach and intestine. Flexner regards the nasopharynx as the atrium to the nervous system and also as the point of exit; its *ingress* is along the lymph channels, around the filaments of the olfactory nerves, and its *egress*, in the reverse direction, along the same paths.

Müller holds that, as in rabies, infection progresses along the nerve sheaths, but Flexner maintains that it occurs by the cerebrospinal fluid. The virus was inoculated into monkeys, and from one to another through 25 animals successively. Flexner and Noguchi cultivated the virus; it is anaërobic; small bodies are found, 0.15–0.3 microns, single, double, in chains or masses. The virus is extremely *resistant* to external influences, as freezing or drying, and persists in the nasopharynx from six weeks to six months after the disease, *in monkeys*, and after it had disappeared from the brain and cord.

II. Dissemination.—The nasal, bronchial and intestinal secretions, the house- and stable-fly, and dust, may convey the disease.

III. Carriers.—In epidemics a certain percentage of cases occur of the abortive type, in which few symptoms develop. Frost and Anderson proved that two-thirds of these cases were actually poliomyelitis, by the biological test (the serum of one who has had the disease, injected into monkeys, protects the latter from inoculation). This may explain the apparent immunity of children in households in which the frank form of the disease prevails; they may have a light infection, overlooked save for the presence of an epidemic, or may carry the virus without themselves contracting the disease.

IV. Secondary Factors.—Sixty per cent. of cases occur in the first three years, and 87 per cent. in the first ten years, and 80 per cent. of cases occur in the *summer months*.

Pathology.—The affection is called poliomyelitis (Kussmaul), because it is an inflammation of the anterior gray matter, supplied by the central arteries, branches of the anterior median artery of the anterior longitudinal fissure. A given vessel branches up and down, supplying a cylindrical vertical area of two inches or more, the usual dimension of a poliomyelitic focus. Recent reports place equal emphasis on changes in the central vein and in other arteries. The cord changes are always accompanied by a round-cell infiltration of the pia mater and the arachnoid, such as occurs in rabies and syphilis, but not in diseases of bacterial origin. The meningitis is most marked in the lumbar and sacral regions of the cord, next in the cervical; it is most intense over the anterior surface, whence it follows the pia into the depths of the anterior fossa, along the sheaths of the vessels, the posterior root fibers and the arachnoid covering the spinal ganglia. This inflammation of the pia, arachnoid and ganglia is responsible for the irritative symptoms of the disease. The inflammation may extend back of the anterior horns into the anterolateral columns. The fresh focus is rarely seen, since death is unusual; to the naked eye it is red, soft, juts above the cut section and is mostly confined to the anterior horn, *i. e.*, the beginning of the lower motor neurone. Microscopically, the lesion in the anterior roots is inflammation in and about the vessels above named, even before they enter the cord; the perivascular spaces are full of round cells and granule-cells; there is exudation of red and white cells, with actual hemorrhage in severe cases; the ganglion cells in the anterior horn suffer secondary changes; their dendrites may shrink, their nuclei disappear, or the whole cell may disintegrate, while here and there some cells escape. The cells involved depend

solely on the vessel affected; involvement is sometimes bilateral and in severe cases is distributed through several segments, especially in the lumbar cord.

An inflammatory edema is detected throughout the cord, macroscopically, by the moist appearance of its cut surface; it explains the transitory nature of certain symptoms. As easily anticipated, disease of the trophic cells of the lower motor neurone must be followed by degeneration of their axis-cylinders, nerve roots, peripheral nerves and muscles (which become granular, fatty and later indurated). In *old cases* the unaided eye detects shrinking of the anterior horns, with lack of differentiation between the gray and white matter. Microscopically, the formerly inflamed vessels are thickened and pigmented; groups of ganglion cells have disappeared and their nerve fibers are degenerated or wholly wasted, which led Charcot to consider the process essentially in the ganglionic cells, but the thicker connective tissue is rich in cells and stains deeply with carmine. In *severe cases*, identical changes are found in the medulla, pons, centrum ovale, and even in the cortex or cerebellum. Acute poliomyelitis differs from acute myelitis and disseminated myelitis only in its localization. The visceral pathology, other than the nervous, consists of lymphoid hyperplasia of the lymph glands, spleen and intestinal follicles (which may be the atrium of the infection), changes similar to those of typhoid; the kidneys are cloudy, foci of necrosis appear in the liver, and there is bronchitis and often bronchopneumonia.

Symptoms.—Wickman's classification of poliomyelitis includes eight types as follows:

I. **The Spinal Type.**—There are *three clinical periods* after an incubation of 5 to 10 days. (1) The acute stage. (2) The diffuse paralysis when degeneration appears and regeneration begins. (3) The stationary stage, with determination of the paralysis to given muscles.

1. Acute Stage.—The typical onset is acute, appearing in a child previously well, with *fever* (75 per cent.), often with *vomiting*, headache, somnolence and sometimes with *convulsions* or coma, which are followed by a *sudden complete and flaccid paralysis* in one or more limbs; it corresponds to the acute inflammation in the anterior horn. The fever averages 101°, may reach 105° or more and lasts about twenty-four hours. Convulsions are not as common as in the cerebral type, and occur mostly in younger subjects, are attributed perhaps to teething, once thought the cause of the paralysis. The motor paralysis usually reaches its maximum in twenty-four to forty-eight hours and is not progressive. In many cases *prodromata* exist, as *tonsillitis* or *angina*, coryza, bronchitis, swollen joints, malaise, pain in the back and limbs or diarrhea; Müller anticipates poliomyelitis in a general infection beginning with profuse sweats, hyperesthesia and leukopenia; tympanites and weakness of the abdominal reflexes and abdominal muscles are also emphasized. *Variations* in onset; the fever may last one or two weeks before the paralysis develops, it may last a few hours only or may be absent. The paralysis may develop during the night; sometimes it is not noticed until, for example, the child fails to walk. Convulsions involve principally the extremities, sometimes as clonic muscular contractions.

2. Stage of Degeneration.—The localization in the anterior horn produces paralysis, disturbed nutrition, vasomotor changes and loss of reflexes. (*a*) The *paralysis* is motor only; it is sudden, complete, flaccid and non-progressive; at first diffuse, it usually regresses, although incompletely, leaving some permanent paralysis. *General localization:* (i) Paralysis, most frequently of one leg, then (ii) of both legs, and (iii) and (iv) of all four limbs or of one arm (with equal frequency), (v) of arm and leg of same side (spinal hemiplegia), (vi) of arm with opposite leg (crossed paralysis), (vii) of three limbs and least often (viii) of both arms. The back, abdomen, neck, face and eye may be involved in severe and diffuse foci. As to its *special localization,* the *leg* is *paralyzed in* 75 *per cent.* of cases; in one form the lasting paralysis affects the quadriceps femoris with the adductors (upper lumbar lesion), but the sartorius is not involved; another form involves the muscles below the knee supplied by the peroneal nerve—the tibialis anticus, extensor digitorum communis, ext. longus or brevis, ext. hallucis longus, peronei (lower lumbar and upper sacral lesion), of which some muscles may escape, as the tibialis anticus. If both legs are involved the paralysis is rarely symmetrical. Poliomyelitis usually involves several segments because of the arterial distribution. The *arm* is paralyzed in 25 per cent., chiefly in the form of the combined shoulder paralysis of Remak, involving the deltoid (sometimes alone), biceps, brachialis, supinator longus with the supra- and infraspinatus and teretes (lesion in upper cervical enlargement, fifth to sixth segment); in some cases the forearm type may be seen, with involvement of the flexors, possibly the extensors, while the supinator longus often escapes (lower cervical lesion, seventh and eighth segments). The trunk or neck muscles usually show improvement in this stage. (*b*) As to *disturbed nutrition,* the muscles and nerves corresponding to the level of the poliomyelitis rapidly degenerate, as shown by early reaction of degeneration and muscular atrophy. The *reaction of degeneration* consists of failure of the nerve and muscles to respond to the faradic current and usually appears in one or two weeks; it also includes a lack of response of the nerve to the galvanic current and finally a too prompt response of the muscles to the galvanic current and more quickly to the positive than to the negative pole; this latter reaction lasts for two, three or six months and then gradually disappears in a year or two. *Muscular atrophy* is more clearly seen in the arm than in the leg or trunk, where adipose deposit may obscure it. Fibrillary contractions are frequent. The wasted flaccid member hangs and swings in a lifeless fashion. (*c*) *Vasomotor paralysis* causes the low temperature (1° to even 10° below normal) and the livid, clammy skin. (*d*) The skin and tendon *reflexes* are *abolished* in the limb at first; the skin reflexes usually return later. The *sphincters* are almost always intact and sensation is very rarely affected. Trophic changes are almost unknown.

3. Stationary, Chronic, Residual Stage.—This stage is marked (*a*) by accentuation of *muscular atrophy,* connective and sometimes adipose tissue replacing the contractile substance, especially in the legs. (*b*) The *bones* become flexible, friable, arrested in development and therefore shorter; the bone cortex wastes and its medulla becomes more

spongy; scoliosis may result. (*c*) There may be pronounced vasomotor lividity. (*d*) In the *joints*, nodules may appear as in cerebral hemiplegia; the joint structures are relaxed and the "flail joint" may develop with luxation or subluxation. (*e*) *Contractures* are due principally to the mechanical influence of the unopposed antagonist muscles on the posture of the joint; if all the muscles are paralyzed they sometimes result from static causes, as from standing or attempting to walk; the arm, shoulder, wrist and fingers are flexed and the elbow usually escapes involvement. In the leg, deformity (pes equinovarus) is more common than in the arm.

II. **The Ascending or Descending Type.**—This variety, more often ascending than descending, may exactly mimic Landry's paralysis. In one epidemic (1905), among 159 poliomyelitis deaths, 28 per cent. were of this form.

III. **Bulbar or Pontine Type.**—Perivascular exudation may crowd closely around these vital nuclei, without injury to them, yet in all fatal cases they are affected; death is caused by disease of the respiratory centre. The third, fourth, fifth, sixth and seventh nerves are often affected.

IV. **Encephalitic Type.**—This is identical with Strümpell's polioencephalitis (see INFANTILE CEREBRAL PARALYSIS). Convulsions may antedate paralysis by days; hemiplegia, epilepsy and mental deficiency may be its legacy.

V. **Ataxic Type.**—Involvement of the tract between the dentate nucleus of the cerebellum and the contralateral red nucleus may occasion a coarse intention tremor, some paresis and rigidity and increased muscular tonus.

VI. **Polyneuritic Type.**—While the sporadic forms are often painless, the epidemic forms are often exquisitely painful, not merely during the acute phase but for weeks afterward.

VII. **Meningitic Type.**—Without spinal puncture, differentiation may be impracticable, because of the rigidity of the neck, Kernig's sign, twitchings, convulsions, and herpetic, petechial or other rashes.

VIII. **Abortive Types.**—Positively proved to be poliomyelitic by inoculations (*v. s.*), many or most cases would be mistaken, in the absence of an epidemic. The subforms are (*a*) those evidencing only fever or toxemia; (*b*) those with symptoms of meningitis or meningismus; (*c*) the pseudo-influenzal (pains and hyperesthesia); and (*d*) gastro-enteric symptoms as diarrhea, which in some epidemics are the first symptoms, even in 90 per cent. of cases.

Diagnosis.—The initial vomiting, fever and convulsions are usually misinterpreted until the diffuse motor paralysis appears and consequently the physician is unjustly blamed for what no one can anticipate. The rapid atrophy, reaction of degeneration and loss of reflexes are positive diagnostics.

DIFFERENTIATION.—(*a*) The various forms of *muscular atrophy* in children are gradual in onset, progressive, symmetrical and often familial. The atrophy precedes and exceeds paralysis and there is no arrest in development. The neural progressive muscular atrophy of Hoffmann

is progressive, familial and involves sensation. (*b*) *Multiple neuritis* (*q. v.*) has several distinctive features. Poliomyelitis is due to a local inflammation, is unilateral and spinal in type—or if bilateral, it is asymmetrical. Multiple neuritis is hematogenous and toxic, and therefore affects the spinal, and often the cranial nerves symmetrically and bilaterally and usually involves the arms and legs; ataxia, sensory disturbance and local tenderness over the nerve trunks are common; its onset is more subacute, the reaction of degeneration less intense and the issue more favorable. (*c*) The *cerebral palsies* of children (*q. v.*) never produce marked degenerative atrophy or loss of faradic irritability. The hemiplegic form involves the face and the paraplegic form is spastic. Disturbance of the intellect, convulsions and hemichorea are common. (*d*) *Myelitis* also begins acutely, but usually in older subjects. Poliomyelitis may be confused only when it is bilateral, located in the cervical or lumbar enlargement, when sensory changes occur (2 per cent.) or the sphincters are involved, producing a flaccid atrophic paralysis of an arm and a transitory spastic paresis of the leg. Bed-sores so common in myelitis, are almost unknown in poliomyelitis. (*e*) Painful affections causing immobility of a joint, hip-joint disease, Parrot's syphilitic pseudoparalysis and infantile scurvy but superficially resemble poliomyelitis. (*f*) Meningitis shows its appropriate microörganism (tubercle bacillus, meningococcus, etc.). Lumbar puncture of the poliomyelitic shows a clear, possibly slightly opalescent fluid, under increased pressure, exhibiting a mononucleosis, a slight fibrinous clot and positive globulin reactions.

Prognosis.—(1) *Regarding life;* there is little danger after the fever subsides; life is rarely threatened from extension to the medulla oblongata. In epidemics the mortality may reach 15 to 33 per cent., but averages 7 per cent. (2) *Regarding recovery,* improvement is the rule and recovery is due to the recession of the congestion and edema; in some epidemics abortive cases number 33 to 50 per cent. (Trethowan, 5298 cases, 1912). A localized initial paralysis may show little improvement, or an extensive initial paralysis may diminish greatly. A definite prognosis cannot be made for two weeks and then only by the electrical examination. Muscles which show no reaction of degeneration after fourteen days will probably recover; those showing slight loss of faradic irritability will recover in great part; after six weeks only those muscles are paralyzed which will remain paralyzed; if there is no recovery in three months there will be none or it will be slight.

Treatment.—In *prophylaxis,* isolation should be practised, and carriers should be quarantined. The nose and throat require special care in those ill of the disease, in carriers and in those exposed to infection; two parts water to one of hydrogen peroxide, and urotropin are advised (*v. i.*). In the *acute stage* rest, a light diet, laxatives and diaphoretics are indicated. After administration of urotropin, Cushing found formaldehyde in the spinal fluid; Starr recommends urotropin, gr. v, every four hours; Flexner and Lewis found that urotropin given to monkeys, inoculated with the virus, prolongs the incubation and often prevents paralysis. After the fever the patient should be kept in bed for weeks;

immobilizing the limb lessens spinal irritability. Electricity is contraindicated. As the paralysis recedes a fuller diet and careful passive movements are beneficial. Hot baths are quieting and repeated lumbar punctures apparently are beneficial. In the *chronic stationary stage*, with its inevitable and permanent paralysis, fresh air and liberal nourishment are important. Strychnine is valuable because of its general tonic action and its effect on the motor side of the cord. It is given for three weeks after the paralysis becomes stationary. Full doses may induce muscular spasm, pain and irritating paresthesia, and of late its use is far less popular. Electricity cannot restore destroyed tissue, but it hastens recovery in partial lesions; if applied to the spine it is useless, but the constant stream to the muscles and the faradic current to the skin are helpful. Contractures and deformities due to neglect may be modified by gymnastic movements, massage and orthopedic apparatus. The child should be kept in bed for a long period. In some cases tenotomy, arthrodesis and tendon transplantation (by attaching the tendons of sound muscles to those of paralyzed muscles) and nerve transplantation are indicated.

INFLUENZA (LA GRIPPE).

Definition.—A specific infection, caused by the Bacillus influenzæ, and characterized by its protean symptomatology and particular affinity for the respiratory tract.

Varieties.—(1) Influenza vera, pandemic, endemic or epidemic, due to Pfeiffer's bacillus. (2) Influenza notha s. nostras, pseudogrippe, catarrhal fever, of variable bacteriology.

Pandemics originate in Asiatic Russia. No other pandemic is so widely or rapidly distributed as influenza, which is due to the enormous virulence of the virus; to the universal susceptibility; and to the fact that many moderately sick individuals frequent public places. Epidemics usually follow pandemics; they occur every few years whereas pandemics are separated by decades. The first epidemic occurred in 1173 and the first pandemic in 1510.

Etiology.—Most cases occur between the twentieth and fortieth years. The immunity conferred by one attack is less enduring and absolute than in other infections. The organism discovered by Pfeiffer (1891–2) is the smallest *bacillus* cultivated. Its ends are rounded and it resembles a diplococcus when two bacilli lie end to end. It has no capsule, is nonmotile, it lies free in the sputum in the early stages and later within the leukocytes, but often disappears early from the sputum. The bacilli lie in lines "like schools of fish." It is best stained by Gram's method and then ten minutes' staining with carbol fuchsin; if pneumococci and streptococci are present, they are stained deep purple, and the influenza organism red. The bacillus is aërobic, short-lived and very susceptible to drying. It grows characteristically on blood serum as closely compressed yet discrete colonies, clear as water and almost microscopic in size; it is easily overgrown by saprophytes. The germ has also been found in rare

instances in the blood, cerebrospinal fluid, gall-bladder, joints, pneumonic and encephalitic foci, peritoneum (puerperal sepsis) and other parts, and possesses rather marked pyogenic characters. Its *atrium* is probably respiratory.

Symptoms.—There is scarcely another acute infection with such varied symptoms, sequels and complications.

After a *short incubation* of one to three days, the grippe begins suddenly with chill and fever, headache, general pains, respiratory catarrh and intense nervous and sometimes digestive symptoms.

Types.—In the *simple toxemic type* there are coryza, moderate soreness of the throat, possibly some cough and toxemia, evidenced by fever, depression, and pains in the head, back and limbs. In the *severer toxemic forms*, these symptoms obtain, but the patient suffers profound depression both with the fever, and after it. The intensely *respiratory variety* (75 per cent. of all influenzas) is attended by fever and toxemia, the bronchitis is severe, perhaps intractable and not uncommonly followed by pneumonia. The *gastro-intestinal* grippe is attended by gastric intolerance and diarrhea. Of the septicopyemic type, only seven instances are reported.

The *duration* is one to three days and the diagnosis is usually easy without the detection of the bacillus. Recurrences are frequent; most frequently, an initial respiratory catarrh is seen with nervous manifestations in the recrudescence but the order may be reversed. In rare instances influenza may cause chronic symptoms. Convalescence is marked by great nervous depression, neuralgias, possibly cardiovascular atonicity, and tardy return of vigor.

Special Symptoms.—1. Fever.—The rise is usually sudden to 103° or 104°. It is entirely atypical. Remittent fever is more common than continuous fever, which may suggest typhoid. It is occasionally intermittent, resembling malaria. Hyperpyrexia is observed particularly in encephalitis. Certain nervous forms are afebrile. The fever runs one to four days, or longer as a result of complications.

2. Nervous System.—The nervous system is second in importance only to the respiratory tract. The most frequent disturbances are functional and psychical. Headache is observed in nearly all cases, and often is very severe. *Influenza comatosa* results from very acute intoxication, which also may cause epileptiform seizures, or severe delirium. The postinfluenzal *psychoses*, due to toxemic exhaustion, are observed eight times as frequently as in any other acute disease, and most cases are seen in the young. They usually follow the attack and chiefly are of the hypochondriacal type. Suicides in Paris increased 25 per cent. during the last pandemic. In *encephalitis grippalis*, acute foci of inflammation, usually discrete and multiple, occur in the gray substance of the cortex or ganglia and vary in size from the "flea-bite" spots of inflammatory hemorrhage with surrounding softening to the size of a cherry or pigeon's egg—due to embolism by the Bacillus influenzæ (Pfuhl and Nauwerk). Mono- and hemiplegia or sudden apoplectiform onset may mark the process, or there may be diffuse cerebral symptoms without definite focal localization. Encephalitis is usually fatal. *Meningitis*

grippalis generally develops early, the virus reaching the brain by the blood stream or through the basis cranii. Slawyk first found the organism in the cerebrospinal fluid. Nearly all are under ten years of age; of Flexner's 58 cases (1911), all but six died. Transverse myelitis has been observed. *Neuritis* is usually postinfluenzal; neuralgia affects most often the fifth, sciatic and intercostal nerves.

3. The respiratory tract is the atrium of the bacillus and is involved in 75 per cent. of cases; *coryza* is seen in 70 per cent. of cases, *angina* in 33 per cent. and *tonsillitis* in 60 per cent. Laryngitis (10 per cent.) occurs with hoarseness. *Tracheitis* (65 per cent.) causes pain, tenderness and severe paroxysmal cough, induced by irritation at the tracheal bifurcation.

Bronchitis is diffuse or more characteristically occurs over one lung or lobe. At autopsy are found great congestion, hemorrhage, round-cell infiltration into the mucosa, and thrombosis of the smaller vessels, which causes necrosis of the mucosa. The sputum is characteristically nummular and greenish-yellow. It is often blood-stained, viscid and contains the bacillus. Rales abound near the liver and lingual lobe. We may observe dyspnea without physical findings. Cyanosis is inauspicious in the young, aged and decrepit. Acute *bronchiectasis* is not uncommon, but usually escapes clinical recognition.

Influenzal pneumonia (in 6 per cent. of cases) is the *most important complication.* Pfeiffer's bacillus is its most frequent cause, alone or combined with the pneumococcus, streptococcus and staphylococcus. Influenzal pneumonia, strictly speaking, shows no fibrinous exudation or granulations, but is *catarrhal,* with infiltration of cells about the bronchi as discrete foci. Clinically, the pneumonia is *atypical* and begins without a chill, but with insidious increase of the prior cough and dyspnea. It usually develops during or after the influenzal attack, although pneumonia may be the first sign of influenza. Its symptoms are more pronounced than its physical signs, and the disproportionate cyanosis and dyspnea sometimes suggest miliary tuberculosis. Foci of dulness may be found but distinct bronchial breathing is rare; bronchophony is a much more reliable symptom. There may be oligopnea (slow breathing) with difficult, prolonged expiration. The heart is often weak and a peculiar redness of the face and head, profuse sweats at the beginning, and characteristic paroxysmal coughing are symptoms not seen in ordinary pneumonia. The foci are bilateral or multiple in 60 per cent. of cases and the apices are involved frequently. The sputum is purulent or bloody. *Issues:* (1) solution by lysis; (2) death from early edema pulmonum; the patient may die later, with early gray hepatization; the mortality is 20 to 30 per cent.; (3) chronic influenza-pneumonia, with hectic fever and suppuration, simulating tuberculosis; during a grippe epidemic an increased mortality from tuberculosis is noticed; (4) abscess or gangrene of the lung. A grippe epidemic may double the number of *lobar pneumonia* cases; when it complicates grippe, it is less completely lobar and granular.

Primary *pleurisy* occurs in 27 per cent. of the fatal cases of influenza; its symptoms are frequently severe and protracted.

4. Circulation.—The heart-muscle is directly injured, especially affecting the lesser circulation. Weakness or syncope may occur during convalescence, especially from preëxisting arteriosclerotic changes. The pulse is labile, often arrhythmic or dicrotic; it may be rapid and out of proportion to the fever; cyanosis, dyspnea and bronchiolitis are frequent in such cases. A slow pulse occurs in 50 per cent. of the severer types. Acute endocarditis sometimes follows abscess of the lung; in three cases the influenza bacillus was found. Acute phlebitis is seen mostly in severe cases with cardiac weakness, most frequently in the upper extremities. Arteritis is rare, occurs mostly in the popliteal vessels, and may end in gangrene. Leukocytosis occurs in a third of the cases Hemorrhages into the skin and mucous membranes indicate severe intoxication.

5. Alimentary Tract.—Digestive symptoms are not important. The coated tongue is rarely typhoidal. Vomiting (34 per cent.) is sometimes persistent (grippe hyperesthesia of the stomach). Constipation is more frequent than diarrhea. There are the usual febrile degenerative changes in the liver. Icterus is rare (2 per cent.). The spleen is enlarged in 15 per cent. of cases.

6. Genito-urinary Tract.—Albuminuria may result from toxemia (10 per cent.). Altered red blood cells in the urine are rather frequent. Acute glomerulonephritis occurs in 1 per cent. of cases. The diazo reaction and indicanuria are noted frequently.

7. Skin, Muscles, Joints.—Urticaria, herpes (5 per cent.), miliaria, erythematous, scarlatiniform or morbilliform eruptions or purpura may be noted (16 per cent.). Polysynovitis, pyarthrosis, bone involvement and myositis are not common. Eye involvement (7 per cent.), conjunctivitis, keratitis, herpes and otitis media (35 per cent.), usually purulent and often hemorrhagic, are due to the pyogenic cocci, pneumococci and the influenza bacillus.

Diagnosis.—During an epidemic the diagnosis is simple. Respiratory catarrh, headache, pains in the joints and back, nervous depression and tardy convalescence are significant. The diagnosis of the toxic, nervous and alimentary forms is more difficult and many errors may be made. The importance of bacteriological diagnosis has been over-rated, but positive results are decisive.

Differentiation.—A clinical picture, identical with the grippe, may be due to pneumococcemia, without hepatization. A peculiar streptococcus causing influenzal symptoms, is reported by Müller, Seligmann and Richardson in septic sore throat.

Typhoid fever may be considered when severe influenza begins with step-like rise of the fever and tympany, enteritis, intestinal hemorrhage, swelling of the spleen and roseolæ; at autopsy, swelling and even ulceration of the stomach mucosa and Peyer's patches occur. However, the existence of an epidemic and early catarrhal symptoms indicate influenza.

Simple coryza and bronchitis are distinguished by the sudden rise of fever in influenza, its nervous manifestations, bacteriology, pains in the head, splenic tumor and exanthems. Lord found the bacillus of

Pfeiffer in 30 per cent. of bronchitis cases in Boston, in which there was no suspicion of influenza.

Measles.—The early respiratory catarrh and the measles-like eruptions in influenza may cause confusion, but the measles fever-curve, rising after the prodromes, with the exanthem and Koplik's spots, determine the diagnosis on the third or fourth day.

Miliary tuberculosis is suggested only in severe respiratory types. Meningitis, apoplexy, acute psychoses and acute poisoning are rarely simulated by influenza.

Prognosis.—(1) The morbidity is enormous (50 to 75 per cent. of the population, pandemic of 1889–90). (2) The mortality is 0.6 to 1 per cent. Influenza increases the number of deaths from pneumonia and tuberculosis, especially in the weak and aged. In some cases the course is chronic, the bacillus being found in the sputum for months, especially in the remarkably susceptible tuberculous subject.

Treatment.—There is scarcely any *prophylaxis* except avoiding crowds. Isolation should be practised in asylums, hospitals and prisons. Gargles and nasal douches possibly prevent infection and are indicated in grippe patients. There is *no specific treatment.* Influenza must be treated as a serious disease, and complications avoided by rest in bed and a restricted diet.

1. Pain.—At the onset, Dover's powder and acetphenetidin, āā grains x, are given for pain (phenacetin is superior to acetanilide and is less depressing). These measures failing, the salicylates (or acetylsalicylic acid, aspirin) are used as in rheumatism. Cold baths are poorly tolerated.

2. Catarrhal Symptoms.—For conjunctivitis, boric acid solution is excellent; for coryza (*q. v.*) douches with Dobell's solution, tincture of belladonna and of aconite in two- and one-drop doses, respectively, every half-hour for six doses, and the alkaloid cocaine, gr. j to albolene ʒj, should be used locally; for pharyngitis, silver nitrate (20 per cent.); for tonsillitis, nitrate of silver is introduced into the follicles which are first opened; for bronchitis (*q. v.*), heroine or morphine is indicated; for enteritis, bismuth and paregoric, āā ʒj, are given after each bowel movement.

3. Nervous symptoms are somewhat relieved by warm baths. Neuralgia indicates acetphenetidin, camphor, iron and arsenic. Headache is treated as in typhoid. Flexner and Wollstein prepared a serum against experimental influenzal meningitis.

4. Heart.—Strychnine may be indicated.

5. Convalescence concerns chronic tuberculosis and bronchitis.

PERTUSSIS (WHOOPING-COUGH).

Definition.—A specific infection, characterized by a cyclic course and a severe convulsive cough, which ends in a whoop. Cullen's definition was "Morbus contagiosus, tussis convulsiva strangulans cum inspiratione sonora iterata, saepe vomitus."

History.—Pertussis was described by Ballonius (1578).

Etiology.—(*a*) *Age:* It usually occurs in children and is most frequent at the fourth year. It is extremely infrequent after the twentieth year. (*b*) *Sex:* Sixty-six per cent. of cases occur in girls. (*c*) Most cases occur in March and April.

In 1906 Bordet and Gengou obtained in pure culture a small, short bacillus, with rounded ends. It stains feebly and shows polar granules with carbol methylene blue. It occurs in the sputum unmixed with other organisms at the onset, in the catarrhal stage and for a few days after the whoop appears. As the disease progresses, other organisms, especially the influenza bacillus, overgrow it. It has been cultivated from the blood and lungs. The blood in convalescent cases rather feebly agglutinates the organism. Specific antibodies have been demonstrated in the serum. Mallory and Horner find the bacillus characteristically between the cilia of the trachea and bronchi. It interferes mechanically with the movements of the cilia, preventing the normal removal of secretion. One attack confers almost absolute immunity. *Incubation* lasts from two to eight days.

Symptoms.—1. The *stadium catarrhale* is characterized by headache, photophobia, conjunctivitis, coryza, angina and a cough which becomes dryer and harder toward the end of this stage. In a few cases there is moderate fever. This stage averages one week, or less during an epidemic.

2. The *stadium spasmodicum* dates from the first "whoop." The fever ceases, aside from complications. The *seizures* are paroxysmal, convulsive and accompanied by dyspnea and vomiting. The child is well except for the paroxysm, which has for an aura, tickling in the larynx, thoracic constriction, vertigo or a creeping sensation, when the child braces himself and terrified, runs for support. Then the explosion comes in the form of *three* to *ten or more short expiratory coughs* following *in rapid succession* until the breath is lost, when the "*whoop*" is heard as a deep, singing or whistling inspiration. Then thin tenacious mucus is expectorated and vomiting constitutes the crisis of the paroxysm. In severe cases there may be apnea and cyanosis. One to two minutes elapse between expiration and inspiration, and inspiratory and expiratory spasms or generalized convulsions may develop. The eyes are injected, the nose runs, the jugular veins are large and the skin is clammy. Involuntary evacuations occur; the pulse is small; ecchymoses sometimes occur in the conjunctivæ, scleræ, neck or face. After the attack, consciousness returns, respiration is fast, and there is fatigue, stupor, sweating and pain in the abdominal or other muscles from the strain of coughing. The attacks occur especially toward morning. The attacks average twenty daily. In the free interval there may be euphoria or great depression, gastric disturbance, inanition or diarrhea. A marked leukocytosis is observed early in the disease; lymphocytosis occurs in 85 per cent. of cases.

3. The *stadium decrementi* follows, but this stage is longer in proportion as the case is mild. From its complications (*v. i.*) it is the most dangerous stage.

Course.—The incubation lasts one week; the catarrhal stage one to two, the spasmodic, three to six, and the decline, three weeks.

Complications.—1. Increase or Extension of the Usual Symptoms. —*Catarrhal inflammation* is common in the initial stage—stomatitis, laryngitis, diarrhea (10 per cent.) and otitis media (10 per cent.). Bronchitis (bronchiolitis) is not common in the spasmodic stage. It may occur with fever, a pulse of 120 to 150, respirations of 30 to 50, many rales, cyanosis and atelectasis. *Bronchopneumonia* is the *most frequent and severe complication,* occurring particularly in rhachitic children between the third and fifth years, most often in the convulsive stage. The onset is gradual with fever of 100° to 102°, pulse of 130 to 160, respirations of 30 to 80, with cyanosis and dyspnea. The mortality may reach 25 per cent. and an almost absolutely bad prognosis must be given in the very young. Sometimes the pneumonia greatly lessens or entirely abolishes the spasms. Lobar pneumonia, exudative pleurisy, endocarditis, pericarditis, meningitis and nephritis are very infrequent. *Spasm of the glottis* in nervous or scrofulous children is largely nocturnal in incidence, and may cause death from asphyxia even in the lightest cases. *Convulsions* of the carpopedal type, especially in nervous children, are most frequent at the height of the apnea. The eyes are rolled upward and outward, the pupils are dilated and coma follows.

2. Mechanical Complications.—*Hemorrhages* may occur into the *skin,* conjunctivæ, nose, ears and throat. Valentine summarized 83 instances of paralysis from cerebral hemorrhage. Extravasation of blood into the retina, lungs, stomach and intestine is rare. There may be *edema* of the eye-lids, rupture of the tympanum or *frenulum* of the tongue, the *ulceration* of which is almost constant. *Vomiting* occurs regularly, as a mechanical crisis to the paroxysm. Other mechanical complications are cardiac dilatation, pulmonary emphysema (less often interstitial than alveolar), bronchiectasis, pneumothorax, involuntary evacuations, prolapse of the rectum or uterus, aneurysm, muscular rupture, hernias and rib fracture.

3. Toxemia.—Marked toxemia is rare. Parenchymatous degenerations are found in the fatal cases. Nephritis is not common.

Sequels.—The hysterical coughing which sometimes follows pertussis may very closely resemble it. *Tuberculosis* may involve the lungs or lymph glands, especially *after pertussis with measles,* both of which infections predispose to tuberculosis. Preëxisting tuberculosis renders the prognosis of whooping-cough very unfavorable. The heart-muscle may suffer permanent weakness.

Diagnosis.—Diagnosis is made by (1) the definite stages of the disease; (2) the presence of an epidemic; (3) the characteristic convulsive seizures; (4) the sublingual ulceration, swollen eyelids, facial edema, petechiæ and tender areas in the larynx; (5) pressing the tongue back with a spoon often elicits a characteristic attack of coughing. The stridor may be absent in nurslings and adults. The diagnosis may be difficult between paroxysms in the early or late stages. Intercurrent diseases, like pneumonia or measles, may render pertussis atypical.

Differentiation.—In *croup* there are fever, aphonia, metallic inspiration, lividity and constancy of the symptoms. *False croup* (laryngismus stridulus) beginning with acute catarrhal laryngotracheitis, occurs at night

in healthy children. *Spasmodic cough from diseased bronchial glands* very closely resembles pertussis. Barthez and Sannée give the following differentiation:

Whooping-cough	*vs.*	Enlarged Glands.
1. Contagious, epidemic.		1. Isolated, not contagious.
2. Three periods, second paroxysmal.		2. No distinct periods.
3. Paroxysmal cough with whoop, vomiting and viscid expectoration.		3. Paroxysms, without the whoop, expectoration or vomiting.
4. Respiratory sounds normal.		4. Signs of enlarged glands sometimes present.
5. Respiration normal in interval; apyrexia if simple.		5. Asthma in some cases, febrile movements, sweats, wasting, etc.
6. Voice natural.		6. Sometimes a change in voice.
7. Acute.		7. Chronic.

Spasmus glottidis in nervous children at dentition, in rhachitis and tetany, is a sudden spasmodic closure of the glottis without prodromes, but with dyspnea, cyanosis and convulsions. *Asthma periodicum* (Millar's asthma) relates to thymic disease. *Hysterical "after-pertussis"* presents no catarrhal symptoms, fever, vomiting, sublingual ulceration, edema of the face, petechiæ or a tender point in the larynx, and other stigmata of hysteria (*q. v.*) are present.

Prognosis.—In the United States 10,000 deaths occur each year from pertussis, and its death-rate almost equals that of scarlatina (per 100,000, diphtheria 21.4, measles 12.3, scarlatina 11.6, and pertussis 11.4). The mortality averages 3 per cent., but in certain epidemics reaches 15 per cent. The longer the time of development of the convulsive stage, the better is the prognosis, and the clearer are the intervals between paroxysms. The outlook is more unfavorable in the poor, nurslings and children under five years, especially in colored children. The mortality of cases in the first year is about 27 per cent.; in the second year, 14; from the second to the fifth year, 3; from the fifth to fifteenth year, 1.8 (Hagenbach); 90 per cent. of pertussis deaths occur between one and two years of age. According to Trousseau, more than forty attacks in one day indicate a grave prognosis and over 60 attacks indicate a fatal outcome; the writer has seen recovery with over 100 paroxyms daily. Coincident *measles, pneumonia* or early bronchitis, weak heart, continued emesis, *glottis spasm*, eclampsia, skin emphysema, hemorrhages, asphyxia and *tuberculosis* cloud the prognosis.

Treatment.—1. Prophylaxis.—Isolation is indicated, particularly in tuberculous children. It is never advisable to expose a child to an epidemic. The greatest danger of infection exists during the catarrhal stage. The sputum, clothes, etc., should be cared for on antiseptic principles. It was thought that vaccination mitigated or aborted pertussis, but in eleven personal unvaccinated cases, vaccinia was ineffectual.

2. Medication.—There is no specific, as proved by the multitude of vaunted remedies. All drugs should be employed cautiously. *Quinine* is the foremost remedy ($1\frac{1}{2}$ times as many grains *per diem* as the child is years old; to children under two years, $\frac{1}{6}$ grain for each month). Children seldom object to its taste.

℞—Extr. belladonnæ . . . . . gr. iss
Quininæ sulph. . . . . gr. viij
Sacchari albi . . . . . gr. xxx
M. et divide in pulv. no. x.
S.—One powder, three times daily (Widerhofer's formula).

Camphor may be indicated as a stimulant in asthenia and bronchitis, ¾ grain at a dose. Coal tars should not be exhibited. *Bromine* moderates the course of the disease; bromoform should be kept bottled in the dark—dose one to five minims.

The *catarrh* may be treated with expectorants, alkaline drinks, a steaming kettle under a tent constructed over the bed, benzoic and tannic acids (āā gr. ij, t. i. d.).

For the *paroxysm* there is no specific. *Belladonna* is given, one-sixth grain of the extract, to a child of six to eight months, until flushing is observed; symptomatically it is also useful for the clammy, cool skin and collapse; though children tolerate belladonna well, great care must be exercised in very young children. The same caution applies to ipecac, chloral and opium.

Chloroform inhalations are dangerous. *Codeine* is less effective than *morphine*. *Chloroform* is advantageous in dry coughs (twice as many drops as the child is years old, in one dram of warm water). Adrenalin, ♏j–iij, may relieve spasmodic cough.

℞—Morphinæ hydrochloridi . . . . . gr. ¼
Sodii bromidi . . . . . ʒss
Chlorali hydrati . . . . . gr. xvj
Aquæ chloroformi . . . . . ad. ʒjss
Misce, fiat mistura.
S.—One to three teaspoonfuls for a dose, according to age.

℞—Sodii benzoatis . . . . . gr. lxxij
Sodii bicarbonatis . . . . . gr. xlviij
Aquæ chloroformi . . . . . ʒj
Aquæ anisi . . . . . ad. ʒiij
Misce, fiat mistura.
S.—One to four teaspoonfuls, according to age, every few hours.

Digitalis is used for right-heart dilatation. Intubation for the spasm of the glottis may induce ulceration from the long-continued pressure. *Bronchopneumonia* (*q. v.*).

3. Hygiene and Diet.—A warm climate modifies the course and is prophylactic against tuberculosis. The attacks are often lessened by removal to the country. Irritants, as beef-tea, stimulants, dry bread, cookies and overfeeding, provoke coughing and vomiting. Food should be given at frequent intervals in concentrated form—gruels, milk with lime-water, zwieback in milk, eggs, meat-juice, etc. Older patients tolerate more solid food. Rectal feeding is sometimes advantageous.

4. Vaccines and Serotherapy.—In reports, bacterins are claimed to shorten the course of the disease twelve to twenty-one days, but vaccines and serotherapy are still on probation.

CHOLERA ASIATICA.

Definition.—An infection caused by the comma bacillus and characterized by vomiting, severe purging, cardiac failure and suppression of urine.

History.—The name has been used since the time of Hippocrates, Galen and Celsus, but apparently not for Asiatic cholera. Asiatic cholera existed before Christ. Its first European invasion was in 1817, from which time our knowledge dates. Cholera visited America in 1832–1835, 1836, 1848, 1849, 1854, 1865, 1867 and 1873. Isolated cases have occurred since.

Bacteriology.—Koch, studying cholera in Egypt and India in 1883–1884, discovered the *comma bacillus* in the feces during life and in the bowel at autopsy. It is 1 to 1.5μ long and half as wide; smaller than the tubercle bacillus, but more plump and curved and is motile and flagellated. Gaffky (1887) first obtained pure growths. In cultures its form varies, being sometimes S-shaped, and thread- or spirillum-like. Pure cultures are grown from the stools. After several days it may be found in the bowel wall, though rarely in the stomach, vomitus, blood or viscera. It disappears from the movements after six to eleven days.

In its *dissemination,* cholera resembles typhoid, *i. e.*, infection is indirect. Koch maintained that cholera was conveyed by *water contamination.* He found the cholera vibrios in a water-tank in Calcutta from which infection was clearly traced. Attendance on cholera patients is not dangerous if the hands are kept clean. The germ is very susceptible to boiling, antiseptics and drying. There is little risk of aërial dissemination except by flies. The acid of the stomach protects the individual, and animals are infected experimentally only when the gastric acidity is neutralized and peristalsis is arrested by opium. Pettenkofer objected to Koch's teaching and even swallowed pure cultures, luckily escaping infection. Nevertheless, the terrible Hamburg epidemic (1892–1894) amounted, as Koch said, to a demonstration of almost laboratory exactness. The disease developed "explosively." On August 16 there was 1 case; by August 27 there were 1000 deaths, and September 3, there were 10,000 cases and 4300 deaths. The total mortality was over 8300. The Hamburg drinking water came unfiltered from the Elbe River, which was then at a low level. The germ was found in this supply. Altona and Wandsbeck, parts of Hamburg, had a supply of good water and escaped. The germ is difficult to find in water because it is so easily overgrown by saprophytes.

Infection by ice, milk, lettuce, flies, the washing of infected linen or in the laboratory is less common. In India, hundreds of thousands of pilgrims drink the "holy water" of the Ganges, in which they also bathe and defecate. Similar infections occur in the pilgrimages to Mecca. In former years, caravans carried infection from India through Persia and Afghanistan to Russia, which was the route of dissemination in 1892. Ships from the Orient are extremely dangerous. *Carriers:* In Manila 8 per cent. of 374 healthy individuals were vibrio carriers. In the Italian epidemic of 1911, their officials detected 41 carriers about to

embark for this country, and at New York the quarantine of 62 sick with the disease and also carriers averted an epidemic and its attendant panic.

Decreased *physiological resistance,* alcoholism, purgation, dyspepsia, overexertion, poor hygienic conditions and debilitating diseases are predisposing factors. Counting the mortality in a cholera-stricken community for a few years, the total death-rate is not increased, which indicates that weakly persons succumb to cholera and that later epidemics of other diseases have fewer victims.

Epidemics end with a lessening mortality; their ending and lessened mortality are explained by the decreasing virulence of the germ. Warm weather increases and frost usually checks epidemics; nevertheless, Russia's severest epidemic occurred in the winter.

Symptoms.—After an *incubation of two or three days,* the disease develops; several stages are described, which also correspond to clinical types of varying virulence.

1. Prodromal Diarrhea.—The thin stools contain the comma bacillus, are voided without pain and sometimes produce only moderate malaise. The trouble may cease here, when we speak of cholera diarrhea, or it may pass into the choleraic stage.

2. Choleraic Stage.—The stools, at first yellow and voided frequently, become colorless because the bile pigment is diluted and decreased, and become thin—the ominous "rice-water" stools. The stools may number twenty or thirty, aggregating a pint to five quarts daily. They have a fetid, not a fecal odor, and contain flocculi of comma bacilli, leukocytes, epithelial cells, detritus and a few red cells. There is often gurgling, although little gas is voided. The fluid thus lost to the circulation, causes some of the following symptoms: The abdomen is flat, usually neither tender nor painful, and fluid may be percussed in the bowels. *Vomiting* is frequent, painless, although exhausting, thin and profuse, even to three quarts daily; sometimes Koch's bacillus is found. The *skin* shows the lack of fluid; it becomes *shrunken,* wrinkled and when pinched, its folds disappear but slowly. The nose becomes small and pointed, and the malar bones jut out prominently; the extremities, nose and ears become cold and cyanotic, and grayish-blue rings develop beneath the sunken eyes (*facies cholerica*). If pleurisy or dropsy is present, it is rapidly absorbed. The saliva and urine decrease or disappear. The loss of fluid occasions distressing *thirst* and *muscle cramps,* especially in the calves and other muscles, which are found dry at autopsy. The immense transudation into the bowel *concentrates the blood,* whose red cells may total six to eight millions and the serum has a specific gravity of 1.040, even 1.070 (1.028 being the normal). The *heart tones* become *weak* and the pulse rises to 100, because the body cannot lose 10 per cent. of its fluid without injury. The heart has not enough blood upon which to contract and the blood is viscid; the heart also degenerates from absorption of soluble toxins from the bacilli. The difficulty in oxygenation increases the respirations to thirty or forty, and occasions precordial and epigastric anxiety. The *vox cholerica* is due to the dry larynx and weak muscles. The reflexes are decreased or abolished, the pupils tardy;

the sensorium is normal, although exhaustion and apathy are usual. The urine contains albumin and hyaline casts. While the extremities are colder by 5° to 10° or more, the patient feels hot, and the rectal temperature may be 102° or higher. The loss of weight is great (even 1 per cent. hourly). In some cases the patient dies before diarrhea develops (*cholera sicca*). Recovery is possible, even with these ominous symptoms; the diarrhea and algidity may lessen, the heart may improve and erythematous, roseolous and urticarial rashes may develop; indican, previously absent from the urine, may reappear; the urea and ethereal sulphates are increased. The later symptoms probably result from acidosis or absorption of toxins from the intestine (the so-called *cholera typhoid*).

3. Stage of Collapse.—This stage is characterized by asphyxia (literally, without pulse), algidity or collapse. The heart tones are inaudible, the pulse imperceptible, the arteries empty. In the days of venesection the blood would not drain from the incised veins, and at autopsy little or no blood escapes from the vessels. The skin is lead-colored, cyanotic, even purple. There is dyspnea, the expired air is cool, the voice gone, the mouth dry, the conjunctivæ opaque, and the lids are parted, yet the mind is clear. The evacuations cease. Pericardial or pleural rubs may be heard, due to the great viscidity found in all the serosæ at necropsy. Very few patients with cholera-typhoid recover; most die within a few hours, or a day. After death the muscles contract, to the great terror of observers; the face is distorted, the fingers flexed and the legs drawn up. At autopsy the small intestines are injected, filled with rice-water contents, their epithelium and villi are desquamated. Comma bacilli are found in the stools and intestinal wall. The muscles are dry and the kidneys show a pale cortex with great cortical degeneration, especially in the convoluted tubules, from toxemia.

Complications.—Due to the pyogenic cocci, diphtheroid changes may be found in the bowel, uterus, vagina and throat. Small hemorrhages from mucous or serous surfaces may occur. Pneumonia (20 per cent. of fatal cases), pleurisy or lung infarct; skin, parotid or other suppurations; icterus, bronchitis, venous thrombosis or postfebrile psychoses, are uncommon. Griesinger's "cholera-typhoid" is the most frequent sequel (25 per cent. of cases); acidosis, nervous complications and uremia are far less common causes than toxemia operating on the exhausted and anemic brain.

Diagnosis.—The first cases occasion the most difficulty, especially if they are light. Robust individuals may not react to the germs found in their stools; simple diarrhea may result in other cases, and a mild depression only may mark *cholerine*. On the other hand, aged or weakly persons may succumb before its characteristic stages develop. In suspicious cases, laboratory measures are necessary for diagnosis. The pseudocholera vibrios in the Elbe, Seine or Danube Canal (Vienna) are differentiated with difficulty.

Cholera nostras produces a similar, though less severe picture. In some few cases the Finckler-Prior bacillus is found, but in the majority

other microbes are present, such as the colon bacillus, and less often the streptococcus, staphylococcus and a bacillus like the Bacillus subtilis; vibrios, proteus and pyocyaneus forms; the dysentery bacillus; Gärtner's Bacillus enteritidis; in meat, the paratyphoid and anaërobic forms; and in mollusks, Zieber's Bacillus piscicidus agilis. Cholera nostras bears the same relation to cholera Asiatica as varicella does to variola or German measles to measles.

Arsenical poisoning, peritonitis, and intussusception, without bacteriological confirmation may closely resemble cholera.

Prognosis.—Cholera ranks next after the plague in death-rate, and averages 60 per cent., and sometimes 80 per cent.; 100,000 to 700,000 die yearly in India. About 45 per cent. of deaths occur in the first twenty-four hours and 17 per cent. in forty-eight hours, whence the prognosis is more favorable after the second day; when the stage of collapse is reached 85 per cent. die.

Treatment.—1. Prophylaxis.—Isolation of early cases, disinfection of the stools, soiled linen, etc., and special attention to light cases and any suspicious diarrhea are most important. In convalescence the stools may remain infective for seven weeks. Despite the sudden outbreaks in Germany in 1892–1894, sanitary measures kept the total of deaths at 10,000, while in Russia 800,000 died in the same period. Individual prophylaxis concerns the maintenance of physiological resistance; moderation in eating; boiling of drinking water and milk; care in regard to ice; avoidance of green fruits and vegetables, and contamination of food by flies. In quarantine, the stools of all aboard suspected vessels should be examined, to detect carriers. In one series the prophylactic serum of Ferran and Haffkine reduced the morbidity two-thirds and the mortality one-half.

2. Diarrhea.—Absolute rest in bed, an initial cleansing of the bowels with calomel and castor oil; thin gruels; red wine, aromatics, tea and full doses of bismuth, and morphine, hypodermically, should be given. Intestinal antiseptics are inert. The diarrhea, except the early movements, is not easily stopped, because absorption is arrested. Cantani advised that 1 or 2 quarts of 1 per cent. tannic acid solution be given slowly by rectum in the hope that it would reach the small intestine.

3. Vomiting.—Vomiting is intractable. Fluids should not be withheld. The stomach may be washed with normal salt solution. Hot wine, champagne, and hypodermics of morphine are indicated.

4. Heart.—Failing heart action and renal secretion are sometimes helped by a warm bath. Hypodermoclysis and intravenous transfusions were studied during the Hamburg epidemic and their employment often greatly benefited the heart and the general condition.

5. Muscular cramps are helped by warm baths, friction, local heat, saline infusions and morphine hypodermically. In convalescence, great care is necessary lest the diarrhea return. Other symptoms and sequels are treated expectantly.

THE PLAGUE.

Definition.—The pest, or bubonic plague, is a specific acute infection caused by the Bacillus pestis and characterized by a pronounced typhoid state, fever, buboes and carbuncles, very frequently by pesticemia, and by extremely high mortality.

History.—The first clear account dates from the times of Trajan, in the second century, and of Justinian, in the year 542. In the fourteenth century the "black death" swept over Europe with a mortality of 25,000,000 (one-quarter of the world's population). It reappeared in the sixteenth and seventeenth centuries, but in the eighteenth century it withdrew southeast and disappeared from Europe until the small Russian epidemic in 1878–1879. Egypt was the early focus for African epidemics, and European epidemics started from Syria, the leading Asiatic focus for thirteen epidemics (1773–1843). The present Oriental focus is Thibet, whence India and China have been invaded thirty times between 1823 and 1897. Uganda is considered a permanent plague home by Koch; plague is endemic in Asia near Mecca, in Mesopotamia and in Siberia. In 1899–1900, New York, San Francisco, Cape Town, South America and Australia were visited; in 1906 England, and in 1912 Cuba and Porto Rico; 4500 died in Manchuria in 1910–11. Plague appeared in New Orleans in 1914.

Etiology.—The *Bacillus pestis* was found by Yersin in 1894, and independently by Kitasato. Its ends are rounded and stain deeply; its sides are somewhat convex; it measures $1\frac{1}{2}$ to $1\frac{3}{4}$ by $\frac{1}{2}$ to $\frac{3}{4}$ μ and is encapsulated. It is polymorphous, and appears as rods or diplo-bacilli. Atypical forms are ovoid, annular, chain-, thread-, or branch-like; involution forms are the pyriform, biscuit, comma-shaped, or spermatozoid. It contains no spores or flagella. It is found in all organs, in the sputum, the urine and feces, and in the bubo and carbuncles. Its cultures are typical and superficial, and have bulging, wall-like edges. It stains with ordinary stains, but not by Gram's method.

Atrium.—It may enter (1) by the *skin,* through invisible wounds, scratches or the intact integument. Postmortem and laboratory inoculation is possible, as in the unfortunate case of Müller, of Vienna, while writing his well-known monograph. Flies, *fleas,* bed-bugs, etc., may inoculate the disease. (2) The *mucosæ,* conjunctivæ, nose, tonsils, anus and external genitalia, may be primarily infected, especially the *nose and throat* in children. (3) The bacillus may cause primary plague pneumonia.

Contagion may be direct or indirect. (*a*) *Direct:* Dissemination of plague pneumonia by the sputum is most dangerous (*v.* Flügge's experiments in tuberculosis). The pest spreads rapidly only when it is of the pneumonic type. The bacilli escape from ruptured carbuncles or buboes. The urine, the feces and the milk of nursing women are also dangerous. Direct contagion may result from autopsies or operations. (*b*) *Indirect:* This may result from fomites, or bites of animals or insects containing the germ. Epidemics among rats precede human epidemics. In Canton

35,000 dead rats were found in one district. The disease also exists among apes, cats, mice, ground-squirrels, which latter made California a permanent pest focus, and marmots (Manchurian epidemic). Infection by rat-fleas is clearly proved. Flies may convey infection from the urine or feces of infected rats. The bacillus produces *virulent toxins.*

Symptoms.—There are two factors in the clinical picture: (*a*) The local changes, as the primary localization (bubo or pneumonia), and (*b*) the successive septicemia (pesticemia) and toxemia. The average *incubation is five days.* Usually the onset *is very acute,* with marked headache, intense vertigo, severe rigor, sudden fever, vomiting and, in children convulsions. There is *early glandular pain,* profound prostration, and an early typhoid state. The *fever* reaches 104° on the second day, is continuous or remittent, in convalescing cases falls by lysis (fifth to seventh day), or may remain high until death. Crisis is seen in light cases only. The *skin* is *hot* and dry or covered with sweat as death approaches. Skin *hemorrhages,* due to embolism by the Bacillus pestis are frequent before death. Herpes never develops.

1. Local Lesion.—This is the *bubo. The lymph glands are more important than in any other infection.* The *clinical forms* of the plague are (*a*) the *bubonic* type (77.7 per cent.); (*b*) the *septicemic* type (14 per cent.); (*c*) the *pulmonary* type (4.3 per cent.); (*d*) the carbuncle type (2.5 per cent.); (*e*) mixed infection (1 per cent.); and (*f*) the ambulatory type (pestis minor, 0.5 per cent.), which is considered by Scheube as not plague but "climatic bubo." In all except the pulmonary type, *the bubo is found very early* (95 per cent.); it occurs in the external lymph glands in the region of the atrium; it is large, very painful and tender, and is attended by great periadenitis, edema, hemorrhage and vesiculation of the skin. Its size and rapid growth are due in part to lymph-medullary swelling, but chiefly to hemorrhage, which may spread along the nerves or vessels, into which it may break, disseminating the bacilli. The glands may fuse into a brawny mass, as large as an egg or a man's fist; they and the surrounding tissues contain enormous numbers of bacilli. The location is inguinal (70 per cent.), axillary (20 per cent.) and cervical (10 per cent.). The inguinal and axillary adenitis is more common in males; cervical adenitis results in children from infection of nose and throat by dirty fingers; the popliteal and epitrochlear glands, curiously, are involved in but 1 or 2 per cent. Strangely, too, the lymph vessels are usually unaffected. From the primary bubo the contiguous glands are involved, *e. g.,* after the infection of the inguinal glands, the iliac, aortic, mediastinal, subpectoral, infra- and supraclavicular or cervical glands may be involved, *i. e.,* infection occurs in the direction of the lymph current. On the fourth to the sixth day necrosis occurs, and on the eighth or ninth day suppuration develops, resulting more often from the Bacillus pestis alone (66 per cent.) than mixed infection (33 per cent.). The primary bubo is often inconspicuous and its size has no bearing on the intensity of the disease, yet subjects with marked periadenitis rarely recover. The bacilli in the blood have a remarkable affinity for lymphatic structures; these *hematogenous buboes* are smaller, less tender, painful, edematous and hemorrhagic, than the primary

bubo. The mesenteric glands, the pharyngeal follicles, stomach, intestine and lung contain bacilli in large numbers. *Carbuncles* result chiefly from pesticemia and occur in 10 to 15 per cent. of cases, often near the primary bubo; the crowning serous vesicle ruptures, discharges bacilli and leaves an ulcer, whose floor is bluish-red from hemorrhage or yellow from swarms of bacilli. A dark crust then forms. There is great peripheral infiltration and the whole limb may become edematous. These lesions indicate that the pesticemia is an integral part of the disease. The blood shows polymorphonuclear leukocytosis. The spleen is enlarged threefold. Its cut surface is finely granular and slightly glistening. It is infiltrated with white cells and bacilli, found in innumerable foci of minute necrosis. It is usually palpable on the first or second day.

2. Nervous System.—There are few anatomical changes except meningeal hemorrhages and brain edema. Clinically the toxemic "typhoid" symptoms are prominent—early headache, marked prostration and vertigo, causing a drunken, cerebellar gait. The delirium is most often dull, dreamy and docile. Twitching, rigidity of the neck and increased pulse and respiration, polyphagia or polydipsia, may suggest meningitis. Conjunctival hyperemia, sometimes with ecchymoses, is so frequent that Yamagiwa made it a cardinal symptom.

3. Circulation.—Circulatory and nervous changes measure the intensity of infection and forecast the prognosis. There are few cardiac poisons as intense as the plague toxin which operates on the medulla and its vagus nucleus. Early the pulse is full and dicrotic, with a rate of 80 to 90. Later, it increases to 120 to 150; the heart tones become muffled; tachypnea, dyspnea and all the signs of acute cardiac insufficiency develop. At autopsy the heart is lax, the right ventricle dilated, the myocardium cloudy, and the serous investments ecchymotic.

4. Respiratory Tract.—The *plague pneumonia* is of two varieties: (*a*) The *primary*, in which the lung is the atrium (or analogue of the primary bubo)—the "black death" of the middle ages. It became well known in the Indo-Chinese epidemic of 1896–1897, when Childe found the Bacillus pestis in the lung. The bacillus, if inhaled, causes confluent lobar pneumonia and salient lymphatic involvement. The bacilli exist in hordes in the lung and sputum, and may cause hematogenous buboes and carbuncles. The diagnosis is positive only from the sputum, cyanosis, dyspnea, hemoptysis, pleuritic pains, signs of consolidation, adynamia, splenic tumor and collapse. Death occurs in 97 per cent. of cases. (*b*) *Secondary* pneumonia is (i) embolic, as multiple small foci and difficult to diagnosticate without bacilli in the sputum. (ii) Aspiration pneumonia in the lower lobes follows bubonic disease of the tonsil, pharynx or bronchi and is almost invariably fatal

5. Digestive Tract.—A moist, then dry tongue, hemorrhage from the mouth, and involvement of the tonsils or pharynx are common. There may be ecchymoses and erosions in the stomach and intestines; bubonic changes in the lymph structures were found in 20 per cent. of Wilm's cases, and mesenteric adenitis in 60 per cent. Vomiting is frequent, meteorism and diarrhea occasional. The liver is swollen from degeneration or stasis.

6. Genito-urinary Tract.—The urine is decreased, and moderate albuminuria is very common. Nephritis is frequent. Anatomically, degeneration, minute areas of focal necrosis and glomerular hemorrhage from capillary embolism of the bacilli are common. Bacilluria occurs in about 30 per cent. of the cases. The bacilli have been found in the aborted fetus.

Diagnosis.—The *cardinal signs* are fever, buboes, pesticemia and positive blood cultures, inoculation and agglutination (not well marked until the second week of infection, and best developed in convalescence; negative results do not exclude plague).

Prognosis.—A patient, stricken in the morning, may die before night. If the patient outlives a week, the chances of recovery are good. Ominous symptoms are marked nervous and cardiac toxemia, digestive disorders, hemorrhages, cervical buboes, tonsillar involvement and pneumonia. The death-rate is often 80 and sometimes 95 per cent. In Toulon (1721), 77 per cent. of the population had the plague and 62 per cent. died—higher percentages than in any other epidemic disease. Nearly 1,000,000 die yearly in India.

Treatment.—1. Prophylaxis.—The plague patient should be isolated and carefully covered with netting. Infected houses should be isolated, unroofed to admit the sun and fumigated to kill insects and rats. Vigorous attacks should be made on rats and ground-squirrels. Ships should be held in quarantine until the rats in their holds are killed, the bilge-water disinfected, and all clothing fumigated. The sputum may be infective for three months and the urine for six weeks. The strictest precautions are imperative in those coming into close contact with the disease; besides ordinary antisepsis, goggles and masks should be worn. Inoculation with dead cultures and with serum from immunized animals and from convalescent patients is a measure assured of success.

2. Symptomatic Treatment.—Buboes and carbuncles are most efficaciously treated with weak sublimate dressings. Surgical extirpation disseminates infection, and injection of carbolic acid into the buboes does not stay the septicemia. Digitalis, alcohol and hydrotherapy are employed as in other toxemias. Bichloride of mercury is well tolerated and was suggested by the observation that syphilitics under mercurial treatment withstood infection.

DYSENTERY.

Definition.—An acute contagious disease, caused chiefly by the Ameba dysenteriæ and Shiga's Bacillus dysenteriæ; attended anatomically by bowel inflammation and characterized clinically by colic, tenesmus and mucobloody passages.

History.—The name dates from Hippocrates (480 B.C.), and the affection is clearly mentioned in the oldest medical script, the *Papyrus Ebers* (1550 B.C.), and in the *Veda*. It has been the scourge of armies from the times of the Greco-Persian wars. In our Civil war there were 287,522 cases among the Northern troops. Among the English soldiers in India 30 per cent. of the deaths were due to dysentery. It prevailed in the

recent South African and Spanish-American wars, but was practically absent among the Japanese forces (1904–1905). Essentially a disease of the tropics and subtropics, it is frequently observed in temperate zones, especially in prisons and in asylums. Lack of hygiene, overcrowding, eating of spoiled food or fruits and impure water are predisposing causes.

Shiga's Bacillary Form of Dysentery.—The *Bacillus dysenteriæ* was described by Shiga (1897), who found it in the intestinal wall; the bacillus and its toxins, when inoculated, produce intestinal hemorrhage. There is a specific agglutination, increasing in intensity under observation and most marked in convalescents; the serum of the immunized cases is of prophylactic and therapeutic value. Actual inoculations of man produce dysentery, as in Strong's inoculation of a criminal under sentence of death, as well as in accidental laboratory infections. The bacillus resembles the colon and typhoid bacilli in form, though less motile than the latter, and flagellated; it is a facultative anaërobe, contains no spores, stains readily but not by Gram's method, and grows on all media.[1] It is suggested that the Shiga type be called dysentery and the Flexner-Harris type paradysentery, but the general term bacillary dysentery probably will prevail. The bacilli are less abundant in the early diarrheal stools than in the later mucobloody stools; when the dejecta become purulent (last stage) the bacillus disappears. It is found in the mesenteric glands, but rarely in the blood, spleen or liver. Infection is probably carried by contaminated drinking water, milk and flies. Healthy bacilli-carriers and convalescents may convey infection. Flexner, Barker, Strong, Musgrave and Craig found this type in the Philippines; Kruse found it in Germany, Flexner, Goodliffe and others in America (1901). Duval, Bassett, Wollstein and other clinicians discovered it in infantile types. Epidemics have existed for centuries in Japan.

Symptoms.—After an *incubation* of two to eight days, there is a *sudden onset* with fever, reaching 100° to 102° (even 104°), colic and diarrhea. The simple diarrheal stools soon show mucus, and then *bloody mucus* or pure blood, voided with severe rectal tenesmus. The passages are small, numbering twenty to sixty or even one hundred a day, and contain Shiga's bacillus. The urine is scanty. In mild cases the symptoms abate in two to three weeks, and the stools become purulent and contain few bacilli.

The symptoms arise from *local inflammation* and from *systemic toxemic reaction* to solution of the bacilli themselves in the tissues; especially when lesions occur in the lower small intestine, the absorptive power of which is great, a *typhoidal* condition results, with coated tongue, nervous toxemia, weak pulse, and frequently death in the first week. Shiga names this form enterodysentery, as distinguished from the

[1] In gelatin it grows with deep, pin-point cultures, is brownish-yellow without liquefaction, and has well-marked borders; it develops best in glucose agar (grayish growth along the puncture, without fermentation), or in litmus milk (some slight acidification, followed by alkalinization and a brownish color). There are *three strains* of the bacillus: (1) The Shiga type, which attacks glucose but not other sugars; (2) Flexner-Harris type, which attacks glucose, mannite and dextrine but not lactose, and forms more acid; (3) Hiss-Russell type, which attacks glucose and mannite but not lactose or dextrine. The blood of a dysentery subject will agglutinate all types, but particularly the second.

ordinary type, colodysentery. If the stools become gangrenous the outlook is hopeless. Ecchymoses on the chest, epigastrium and inner surface of the limbs are quite frequent. Emaciation is marked in acute and chronic forms.

The symptoms are explained by the *anatomical findings:* The bacilli lodge largely in the small folds and large flexures of the colon and sigmoid. The intensely hyperemic mucosa shows various grades of inflammation—catarrhal, hemorrhagic, diphtheritic, necrotic or ulcerative. The rugæ and the entire mucosa are edematous, thickened and show coagulation necrosis, fibrinous exudation and cellular infiltration; the submucosa is infiltrated; the muscularis is sometimes infiltrated and the serosa is ecchymotic and freshly fibrinous. In marked cases the necrotic areas become ulcers, in or between the follicles; they have a flat base, occupy chiefly the folds, usually reach only the muscularis mucosæ, and in severe cases impart a sieve-like aspect. The swelling may obliterate the lumen of the intestine. Cocci and colon bacilli infest the upper but never the lower layers, where we find the Bacillus dysenteriæ in and around the glands. In the virulent cases the gut becomes gangrenous. Stenosis may follow cicatrization of the ulcers.

Diagnosis.—*Local rectal disease* (syphilis, cancer or papilloma) may possibly confuse. In contradistinction to *amebic dysentery*, it is generally acute; toxemic symptoms and hemorrhages are common, from involvement of the small intestine; the ulcers are flat, on the folds of the intestine and never undermined; liver abscess never occurs. Amebic dysentery is characteristically subacute or chronic; the small intestine less frequently suffers; liver abscess is common (20 to 33 per cent.), and necrosis begins in the submucosa and produces ulcers with undermined edges. *Agglutination*, similar to the Widal reaction in typhoid, occurs in dilutions of 1 to 20 (even 1 to 100 or 1000). It gradually increases as the disease progresses, as Förster noted in typhoid fever—a good prognostic. It is seen in the second week and most clearly in convalescence, and therefore late for diagnostic purposes.

Prognosis.—The prognosis is more favorable in *involvement* of the *rectum* and sigmoid flexure than when the upper colon or cecum is affected. The outlook is grave in patients under five or over fifty years of age. *Intoxication* is a bad prognostic. The effect of *serotherapy* is another factor (*v. i.*). The mortality is high, particularly in Japan, ranging from 20 to 25 per cent. *No immunity* is conferred. A second attack was observed in 2.7 per cent., and a third in 0.3 per cent. in Ogata's collection of 25,279 cases.

Amebic Dysentery.—Lambl (1859) and Lösch (1876) discovered an ameba in the bowel movements. Kartulis, in Egypt, found the ameba in over 500 cases and in the secondary liver abscess; (*a*) it was constant in certain forms of dysentery, in the ulcers, feces and liver abscesses; (*b*) it was inoculable in cats; (*c*) negative results were obtained with the ameba found in normal feces. The home of amebic dysentery is the tropics, especially in Egypt, India, Formosa, China, Arabia and Italy. Sporadic cases occur in the temperate zone, especially in Germany and America, where it is the most frequent form. The author has seen sixteen

cases, clearly acquired in Chicago. In the Philippines it constituted 67 to 80 per cent. of Strong's series. The ameba is found in the pus, glairy mucus or blood-tinged mucus of the evacuations; introducing a rectal tube, the fecal matter in its eyelets shows the ameba. It is round in the quiescent stage or irregular from protrusion of pseudopodia, when the slide is slightly warmed; for diagnosis it must be motile. It is unicellular, and measures 10 to 50μ. Schaudinn distinguishes the innocuous Entameba coli from the pathogenic Entameba histolytica (Ameba dysenteriæ), which is larger, has a highly refractile hyaline ectoplasm (more clearly differentiated from the endoplasm), contains more vacuoles, has a sharper nucleus (5 to 7μ), and more frequent red-cell inclusions. Encysted amebæ are resistant, dangerous forms, resembling the gametes of malaria (Musgrave and Clegg). It stains readily by the Wright-Romanowsky method and in tissues best with thionin. Wooley and Musgrave obtained it in pure cultures. The ameba probably enters in the food or drink. Unhygienic conditions favor infection. Amebic dysentery is probably perpetuated by "carriers," who have had the disease or carry the parasites without reacting to them. Intestinal amebiasis (Musgrave) occurs largely in males in the third or fourth decade.

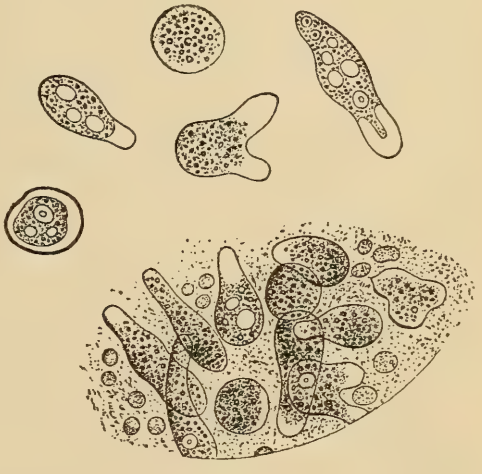


Fig. 12.—Amebæ (Hallopeau).

Symptoms.—The symptoms of *acute amebic dysentery* begin abruptly with much the same picture as in Shiga's type, although the fever is lower and rarely lasts over two or three days. The movements are pultaceous, fluid and yellow at first and later, mucous and bloody. Sometimes in one movement feces are voided first, followed by mucus and blood, or blood-tinged fluid containing amebæ, glairy clumps like swollen sago or frog spawn, pus cells and detritus. The stools may be chocolate-colored, or masses of necrotic tissue are voided. The stools number 5 to 20 or more daily; their odor is fetid when feces are present; they are odorless when only blood or mucus is present; and are stinking when gangrene occurs. The repeated and painful movements cause straining, sweating and sometimes syncope. *Colic* and borborygmus are present; pain is referred to the navel; tenderness is marked over the sigmoid, colon and frequently over the cecum, and tenesmus and a burning anal pain mark involvement of the lower bowel, though less than in Shiga's type. The rectum may prolapse and vesical strangury appear. The abdomen is sometimes distended. *Very light cases* show only catarrhal movements, but blood, pus, membrane or shreds indicate necrosis and ulceration; strong men may be ill for months or even die before the malady is suspected. Constipation was noted by Musgrave in 50 patients, the autopsy showing incipient ulceration or extensive destruction of the colon mucosa; abdominal aching, flatulency, loss of weight and sweating

characterized these cases. *Severe cases* may begin with chills, fever of 103° or 104°, vomiting, herpes and symptoms like those of cholera; *e. g.*, 20 to 100 movements daily, leg cramps, scanty urine, failing pulse and rapid emaciation. *Peracute cases*, with chill, high fever, great abdominal pain, collapse, and even sudden death, exceptionally result from rapid necrosis or gangrene. The average course is one and a half to three weeks. Perforative peritonitis (in 19 per cent.), or exhausting hemorrhages sometimes occur. Neglected cases easily become severe or chronic; spontaneous recovery is uncommon.

Chronic amebic dysentery results from the acute form or is subchronic from the beginning. The symptoms are the same but less intense. Some patients maintain their weight and suffer only from recurrent diarrhea every few weeks, or from irregular moderate intestinal derangement. In the average case the tongue is lacquered and red, the abdomen is somewhat distended and tender and the lower colon and sigmoid are palpable as a thick cord. The *outcome* is (1) recovery after two to four months; severe infections generally leave intestinal cicatrices or chronic catarrh; after a year's illness recovery is seldom complete; (2) recurrent attacks for months or years, constipation alternating with diarrhea; (3) complications, such as *liver abscess* (*q. v.*) in 20 to 33 per cent. Strong reports 10 cases of profuse fatal enterorrhagia coincident with liver abscess. Perforative peritonitis, brain or splenic abscess, arthritis, myelitis, neuritis and less frequently, endocarditis or chronic nephritis are other complications; (4) gradually increasing anemia, marked or extreme emaciation and death from exhaustion.

Pathology.—The early changes in the mucosa are catarrh, inflammatory injection, swelling and hemorrhage, mostly in the large intestine. The essential change is in the *submucosa* (John Hunter, Councilman and Lafleur). The amebæ enter by the tubular glands or sometimes the bloodvessels, and reach the submucosa where they cause fibrinous exudation and *cellular accumulations* which are not leukocytes, but connective-tissue cells. These foci undergo coagulation necrosis and the mucosa which at first covers the small submucous cavities, afterward sloughs, and exposes the primary grayish-yellow necrosis. When the necrotic tissue sloughs out, the *dysenteric ulcers* are more apparent. They lie in the whole of the large intestine (50 per cent. of cases); in the colon descendens and sigmoid (25 per cent.); in the cecum, with ascending or descending colon, or in the rectum (25 per cent.); in the rectum alone (20 per cent.); in the cecum alone (5 per cent.); in the appendix (7 per cent.); ileum (3.5 per cent.) and the entire bowel (80 per cent.). The deep ulceration exceeds the superficial ulceration, and early ulcers may, by fistulous tracts, tunnel the apparently healthy mucous membrane; their edges are undermined. The ulcers may be round or serpiginous and are usually discrete. They may penetrate to the muscularis or perforate the serosa, causing diffuse or localized peritonitis. The lymphatic structures of the bowel are less affected than in Shiga's type. Secondary changes include streptococcic diphtheroid deposits, diffuse sloughing of entire blocks of the intestinal wall and gangrene. In chronic cases the muscularis frequently thickens and narrows the lumen. Healing ulcers produce

pigmented stellate scars which sometimes cause stenosis. Amebæ may enter the mesenteric bloodvessels, by which they reach the portal circulation (pylephlebitis; liver abscess). The *liver shows* toxemic focal necroses, and, in a large percentage, *abscesses;* the solitary, large abscess generally develops in the right lobe near the diaphragm or colon. The multiple small abscesses are also superficial. In both types there is central necrosis, the contents are yellow, green or chocolate-colored, contain fat droplets, liver detritus and the amebæ on the abscess walls. In older cases there is a firm connective-tissue capsule, but round-cell infiltration is as infrequent as in the intestine. Amebæ may be found in the sputum when the abscess ruptures into the lung. The mortality averages 6 per cent.; 7 per cent. of deaths in Cairo are dysenteric.

Indeterminate Dysenteries.—Of these forms, some are primary and others secondary infections (colon bacillus, proteus, pyocyaneus, pneumo-, strepto- and staphylococcus). Varieties: (*a*) *Catarrhal* dysentery, in which the mucosa is injected, cloudy and infiltrated; the solitary follicles are swollen and become round, deep and small ulcers—the "acute follicular colitis" seen especially in children. The symptoms are mild pain and mucobloody evacuations. (*b*) The *croupous* or diphtheroid dysentery represents a severer stage or degree. The epithelium necroses, the submucosa is infiltrated, and there are membranous deposits on the rugæ over slight superficial ulcerations, or there may be deeper, wider ulcers with great thickening of all the intestinal layers. This may occur (i) as a primary infection, with dysenteric symptoms, marked typhoidal symptoms, fever and chill, and frequently with a fatal outcome; or (ii) as a secondary incident in pneumonia particularly, typhoid, scarlatina, diphtheria, syphilis, cardiac affections, cachectic conditions, mercurial or other poisonings, uremia, etc.; there may be symptoms of clinical latency. (*c*) *Gangrenous* dysentery results in enormous swelling, softening, and sloughing of dark, gangrenous shreds, and sometimes in tubular sloughs of the larger part of the intestinal circumference. Perforation sometimes occurs. In the Cochin-China endemic form, enlarged glands, splenic tumor and intumescence of the liver are found. Coprostasis, foreign bodies and parasites are promoting factors, *e. g.*, the cercomonas, Trichocephalus dispar, Anguillula stercoralis or Distoma hematobium, causing dysentery, bloody urine and strangury, hypertrophy of the mucosa and ulceration.

Treatment of all Forms.—1. Specific Treatment.—Shiga's bactericidal and antitoxic serum reduces the mortality from 35 to 9 per cent., and shortens the course; in mild cases ʒiiss are injected once; in medium cases ʒiiss twice, and in severe cases ʒv daily for three days. The best results attend early injection. As Shiga states, specific treatment is more potent in reducing the mortality of the disease than in its prophylaxis. Shiga's serum does not help the Flexner-Harris type, nor conversely. Vaccines are uncertain, but apparently have checked institutional outbreaks.

2. Prophylaxis.—Drinking water should be boiled, and fruits and vegetables should be cooked. In the tropics, sudden variations of temperature are to be avoided. "Carriers" require consideration.

3. Diet.—Acute and chronic cases must be kept in bed. Boiled milk, raw or slightly cooked eggs, or the whites of eggs beaten with water are given to relieve colic and tenesmus. Lactose may benefit (Kendall). Broths and cold drinks excite peristalsis. Some lemonade is permissible. In convalescent, acute or in chronic cases, solids should be given late, commencing with well-cooked, tender meat and starches, and avoiding vegetables and fruit until convalescence is absolutely established.

4. Eliminative Treatment.—Sodium sulphate is given in early cases to cleanse the bowel and to minimize the danger of ascent of the virus from the lower intestine (℥j every two hours until the bowel is emptied). Aromatic sulphuric acid, ♏xx p. c., is sometimes given after the saline treatment. Castor oil or calomel, in frequent and small doses, is of little use except in the early stages.

5. Antisepsis.—Beta-naphtol and phenylis salicylas, āā gr. x, q. i. d., probably do not reach the larger intestine.

6. Ipecac was introduced from Brazil, by Piso, in 1648, and is considered almost specific in India and Brazil. It reduced the mortality from 11 to 5 per cent. in Frayer's series; food is withheld for five hours, and then a hypodermic of morphine, or opium by mouth, is administered to quiet the stomach and lessen nausea; 15 grains of powdered ipecac are administered every hour for two or three doses. If the first dose is vomited, the opium and ipecac are given again. Given in salol- or keratin-coated pills, ipecac passes the stomach and does not induce vomiting, whence morphine or opium can be omitted. Vedder's experiments and Roger's clinical results have revived the use of ipecac in the form of emetin hydrochloride, given once or twice daily, by mouth or hypodermically (gr. $\frac{1}{3}$ in 30 drops of water; $\frac{1}{3}$ gr. equals ℥j of ipecac).

7. Pain.—Opium quiets the patient, enforces rest and decreases tenesmus and peristalsis. It is given most efficaciously by rectum in a suppository, or by enema, mixing Tr. opii deodorat. ♏xx with an ounce of starch water.

8. Local Measures.—*Bismuth* by mouth is local in its action; doses of ℥j–ij are necessary, and even two ounces daily are given. It may cake in the ulcers; bismuth poisoning (from liberated nitrous oxide or arsenical contamination) is avoided by using the subcarbonate. *Lavage* is difficult because of the tenesmus, and is not without danger in copious irrigations. The rectal tube must be introduced carefully as the rectum is very sensitive, first using cocaine, opium or iodoform (5-grain) suppository.

℞—Ac. tannici . . . . . gr. xv
Extr. opii . . . . . gr. iv
Extr. belladonnæ . . . . . gr. j
Olei theobromatis . . . . . q. s.
M. et ft. suppos. no. iv.

Bellei employs as an antiseptic and anesthetic douche, carbolic acid ♏xx, tannin ℥j, marshmallow root ℥ij to a quart of warm water. Thymol is Musgrave's main reliance. Among the other solutions of value are quinine, of which Strong advises two quarts of a 1 to 3000 solution, the bag being three to four feet higher than the bed and the flow being slow.

A weak peroxide solution for the amebic variety is recommended by Harris; all enemata should be warm. Strong silver solutions (1 to 500 or less) cause great pain, and weak dilutions are inert; silver, however, is most suitable for chronic cases.

9. Turpentine Emulsion may be used for tympanites, and Leiter's coil with opium may be used for hemorrhage.

10. Colostomy.—In chronic cases colostomy has been advocated, and recently, wide-open cecostomy (A. B. Herrick).

MALTA FEVER.

Definition.—A specific infection, caused by the *Micrococcus melitensis*, somewhat resembling typhoid, but characterized by a longer course, by recurrent fever, severe sweats, splenic tumor, constipation, orchitis, and rheumatoid or neuralgic pains.

History.—It was first described as a separate disease by Marston (1858). Marston, Bruce and Hughes, English army surgeons, are the largest contributors to our knowledge of Malta fever. Its distribution is well covered by its names—Mediterranean, Rock or Gibraltar fever, Malta fever, Neapolitan or Levant fever. It is also seen on the Danube, Red Sea, Bosphorus; in China, India, the Philippines; in the Canaries, Azores and Antilles; in Venezuela (Caracas fever), Brazil and South Africa. About a dozen cases have come to this country; H. H. Smith reports one from Boston and Gentry and Ferenbaugh 7 cases in Texas.

Etiology.—The *Micrococcus melitensis* (Bruce, 1887) is round or oval, measures 0.3μ, often occurs in chains, is non-motile and stains with methylene blue or gentian violet, but not by Gram's method. Cultures on slightly acid beef-water, peptone and agar, grow characteristically as small pearl-like spots; these after a few weeks become yellowish-brown. The bacillus is found in the blood, milk, feces and urine (even two years after recovery), and in the liver and spleen. Inoculated apes show a typical clinical course and postmortem findings. The disease prevails chiefly between the sixth and thirtieth years, in the hot, dry weather from May to mid-October, and in old unhygienic buildings. The *atrium* is the lungs (dust), or alimentary tract (food or water). In Strong's laboratory infection, the conjunctiva was the point of entrance. It is not directly contagious. Mosquitoes and goats convey infection; the organism is found in the blood of goats in 50 per cent. and in their milk in 20 per cent. In the Texas cases infection came from goats, 34 per cent. of which gave a positive agglutination test (*v. i.*); in 1905 the Government imported Malta goats, in which the micrococcus was found.

Symptoms.—Septicemic symptoms appear after an *incubation of six to ten days*. Some symptoms so resemble those of typhoid and malaria that the three diseases were formerly confounded. The *fever* rises gradually to 104° or 105°. The face is red, there are chilly sensations, frontal headache, depression, and pains in the back and legs. The tongue is coated, at first moist, and then dry and fissured. The tonsils are frequently swollen, and the pharynx is red or ulcerated. Anorexia, nausea,

epigastric pain and tenderness and constipation, are the rule; vomiting, icterus, meteorism and diarrhea, with dark, malodorous, mucous and even bloody stools are sometimes seen. Bruce denies that there are any anatomical findings in the intestines. There is considerable bronchitis and the sputum is often streaked with blood. Dyspnea is quite frequent. The pulse-rate, at first between 80 and 90, becomes faster later. Severe, repeated sweats occur, usually after midnight, accompanied by sudamina (*febris sudoralis*). An acute splenic tumor and hepatic intumescence are due to degeneration and infiltration by round cells.

After one to three weeks, the continuous *fever* remits, and convalescence is apparently established; but the symptoms again return, frequently several times. The fever curve shows waves accompanying each relapse (literally "undulant fever"). The patient becomes emaciated and pallid. There is no leukocytosis and micrococci are found in the blood in 60 per cent. of cases.

Complications.—*Pseudorheumatism* (in 50 per cent. of cases) is accompanied by redness, swelling and pain in the joints and sometimes in the bursæ, periosteum and fibrous tissues. *Neuralgias* are common. *Orchitis* and epididymitis are usually painless, unilateral and ephemeral. Occasionally *polyneuritis* or psychoses are sequels.

Erythematous or hemorrhagic *skin eruptions* occur in the relapses, sometimes accompanied by hemorrhages from the nose, tongue, gums or lungs; desquamation and falling of the hair or a reddish or violet circumscribed edema of the feet and ankles are common. In convalescence, tuberculosis not infrequently develops.

Course and Prognosis.—The majority of cases relapse after three weeks. The average duration of the disease is 120 days; Bruce records a case which lasted over two years; Melland found that 50 per cent. of cases in the Canary Islands lasted from two to three weeks, and in only 10 per cent. did the protracted undulating type occur. Hughes described three types: (*a*) The *undulant*, the most common type; (*b*) the *malignant*, which ends in death in one to three weeks from weak heart or hyperpyrexia (110° or 111°); and (*c*) the *intermittent*, which lasts for months. The death-rate in the last century was over 3 per cent., but it is now under 1 per cent. One attack usually confers *immunity*.

Diagnosis.—The differentiation from typhoid is often difficult. In Malta fever the roseolæ, diazo reaction, Widal and the bacillemia are absent. The course is longer, the sweats more severe and the joints inflamed; orchitis and neuralgias develop. A positive diagnosis can be made by the use of Wright's *serum reaction* (1897), after the fifth day; agglutination is obtained with living or dead cultures; the dilution of the blood is 1 to 50, and the time limit, one hour. The severe bronchitis, fever and sweats may simulate tuberculosis and the older writers spoke of Malta fever as "Mediterranean phthisis." The bronchi are intensely hyperemic and the sputum is frequently blood-tinged. Malaria, ulcerative endocarditis, liver abscess, etc., are differentiated by the agglutination test.

Treatment.—Among English troops quartered at Malta, 2229 cases developed (1898–1904), with 77 deaths; in the fleet there was an equal

number. Avoidance of goat milk reduced the morbidity to 7 cases (1907). There is no specific treatment. Quinine and arsenic are ineffectual. Headache and backache are relieved by acetanilide and morphine; insomnia by bromides, chloral and hyoscine; diarrhea by tr. ferri chlor.; pseudorheumatism by heat, wool investment, tr. iodi, locally, and by salicylates, internally; orchitis, by local heat and suspensory elevation. The general therapy is the same as in typhoid. Dalton advocates a solid diet. In convalescence a change of climate is necessary.

ANTHRAX.

Anthrax is one of the *zoönoses* (diseases acquired from animals), which also include glanders, foot-and-mouth disease, vaccinia and hydrophobia. Anthrax is also called splenic fever, pustula maligna and carbunculus contagiosus.

Definition.—An acute infection caused by the Bacillus anthracis, and usually communicated to man from the herbivora.

History.—Over a century ago Morand and Fournier distinguished between the simple carbuncle and anthrax. The bacilli were seen by Pollander (1855) and Branell (1858), but Davaine (1864–1873) noted that the organisms were bacteria. It remained for Koch with his epoch-making technique to find the spores and absolutely differentiate the disease.

Bacteriology.—The anthrax bacillus is the largest pathogenic microbe (3 to 10 by 1 to 1.5μ). In growths it occurs singly or in chains. The ends are slightly rounded, and the chains resemble the phalanges of the finger, the organisms lying end to end. They are non-motile, transparent, and stain by Gram's method. At high temperatures they grow in long, thread-like, winding, segmented filaments. Spores are absent in the cadaver and in the living tissues; in the living tissues the bacilli multiply by fission. In attenuated cultures, one spore develops for each bacillus; these become bacilli which are extremely resistant to drying and heat.

The bacillus is an obligate aërobe. Stab cultures of agar are characteristic; a white, milky yet transparent and band-like growth develops horizontally in blood serum, and grows downward at an acute angle, so that in three days it resembles a quill with a feathered upper end. Growths on gelatin have a thick, wooly covering; the gelatin liquefies after a few days, and a white, granular sediment falls to the bottom of the tube.

Anthrax is *endemic* and *epidemic* in Europe and Asia. There are persistent foci in Saxony, parts of Bavaria and in Frankfort; in the provinces of Burgundy and Auvergne; Hungary; Russia, principally in Siberia (the "Siberian plague"); in China, India and in South America. Anthrax is rare in America, though small epidemics have appeared in Delaware and Pennsylvania. From Russia, infection may be disseminated by horse's hair and from Asia by mohair (Angora goats). Among animals, the herbivora are most often affected, especially sheep and cattle, and in Russia, horses. Omnivora are more susceptible than carnivora. Mice, guinea-pigs, rabbits, dogs and fowl rarely acquire the disease. Transmission of the spores by means of water, earth-worms, snails, flies and fleas is possible.

Of Koranyi's cases, 65 per cent. were attributed to sheep and 35 per cent. to cattle; Bourgois considers that the greatest percentage comes from cattle, the next largest from sheep, and least from horses.

I. External Anthrax.—External anthrax is by far the more common type. The atrium is an abrasion of the skin, possibly through intact hair-follicles. Koch observed that sheep were infected by fly-bites on the exposed skin of the neck. Shepherds, coachmen and those who attend diseased animals; butchers, tanners, wool-sorters and persons who handle diseased hides or meat; and saddlers, rag-pickers and gardeners are most likely to acquire the disease. Jacobi records infection from a hypodermic needle, first used on a patient with anthrax. The spores are more virulent than the bacilli.

Site of the Pustula Maligna.—In Koch's series 51 per cent. developed on the head and face, 38 per cent. on the upper extremities, 2 per cent. on the lower, 5 per cent. on the neck, and 4 per cent. on the trunk, that is, exposed parts are the usual seat of primary infection. The lesion is usually single, but several pustules may develop, possibly from inoculation by the finger-nails in scratching.

Symptoms.—Incubation lasts two or three days. The initial small red prominence resembles an insect-bite. It itches and in twelve hours develops a small vesicle, which becomes purulent if ruptured and dries with scab formation, and a dark, central, leathery slough, caused by streptococcic infection, and is surrounded by dense round-cell, and often hemorrhagic infiltration. This in turn is surrounded by edema of the connective tissue, in the deeper lymph vessels of which the Bacillus anthracis abounds. If the pustule extends, a circle or even concentric circles of new vesicles appear, which fuse and increase the infiltration. The eyes may be closed or the lips may be so swollen that the jaws cannot be opened. Demarcation now occurs, followed by sloughing, granulation tissue and cicatrization; or *general infection* develops, and the adjacent lymph vessels and glands become infected, soft and hemorrhagic—usually on the fourth to sixth day. Anthrax toxemia and bacillemia are marked by headache, backache, pain in the limbs, chilliness, leukocytosis, continuous or remittent fever, soft pulse, rapid breathing, vomiting of mucus or blood, colic, diarrhea and collapse. Hemorrhages or even gangrene may develop around the carbuncle. Bacilli have been found in the cerebrospinal fluid by lumbar puncture. The mind is usually clear, although delirium, convulsions or coma may develop, and death may occur on the seventh to ninth day with an antemortem fall of temperature. In fatal cases there is only slight rigor mortis but rapid decomposition, dark fluid blood, swelling of the lymphadenoid tissues (glands, intestine and sometimes the spleen). In the liver, kidneys, spleen, heart, lungs, brain and meninges, hemorrhages and swelling may be found; bacilli are seen less in the large vessels than in the capillaries. Bourgois describes another cutaneous form, most often on the body, in which there is great edema (*charbon blanc*, anthrax edema), but no pustule or carbuncle.

Diagnosis.—*Simple carbuncle* resembles anthrax, in that it is also hard, has a prominent central necrosis, is surrounded by edema and sometimes

is vesiculated; it usually causes great pain, and has numerous openings which discharge ordinary pus. The *anthrax carbuncle* may be differentiated by the bacilli, depression of the necrotic centre, the corona of vesicles, more rapid evolution, greater edema, remarkable freedom from pain, and lack of odor. *Glanders* may be distinguished by involvement of the mucosæ, painful nodes without eschars, and ulcerations which discharge the Bacillus mallei. Cultures from the pustule or blood disclose the anthrax bacilli.

Prognosis.—The prognosis is more favorable in children, in cases with local symptoms only, and those with simple carbuncle without edema. Even patients with bacillemia may recover. In Koch's collection, 68 per cent. recovered. Nasarow's figures show 26 per cent. mortality when anthrax occurs in the head and face, 23 per cent. when it occurs in the trunk, 19 per cent. in the neck, 14 per cent. in the upper, and 5 per cent. in the lower extremity.

Treatment.—Prophylaxis.—Infected animals should be cremated entirely. Industrial hair (especially when blood-stained), wool and hides should be thoroughly disinfected. Toussaint (1880) and later, Pasteur, Chauveau and Colbert used attenuated cultures to immunize animals. In France (1882–1893), 1,788,677 sheep were thus inoculated, with a resulting mortality of 0.9 per cent.; and 200,962 cattle were inoculated, with a 0.3 per cent. mortality.

Active Treatment.—Wounds in those exposed to infection should be promptly washed with strong sublimate solution. Cauterization with the live cautery, carbolic or nitric acid or caustic potash; surgical excision through sound tissue; and injections of iodine or corrosive sublimate have been recommended, but equally good results are obtained by elevation, the use of mercurial paste, and *expectant treatment*, which at least avoids dissemination of the parasite through sound tissues. In 1073 cases treated with Mendez's serum, the mortality was but 4 per cent. Salvarsan is recommended.

II. Internal Anthrax.—Internal anthrax is less frequent.

1. **Alimentary Form.**—In mycosis intestinalis, first described by Wahl and Recklinghausen, bacilli may enter through cuts in the mouth and throat, but usually directly invade the intestines and stomach, which are most frequently infected by spores in milk or water. The gastric juice kills the bacilli but not the spores, yet peasants often eat the pickled meat of diseased animals without being infected. In Wurzen, Saxony, there were 206 cases of intestinal anthrax in 1877.

Symptoms.—The onset is usually abrupt, with fever and vomiting of mucus or blood, followed by pain in the bowels, serous and afterward bloody movements, meteorism, dyspnea, weak pulse, and by collapse, as seen in anthrax generalization in the cutaneous form. Secondary carbuncles, or more often hemorrhages into the skin of the neck and the abdomen, may develop. The course is severe; death results in five or six days, although recovery is possible.

Diagnosis.—Bacilli in the stools and blood determine the diagnosis. Changes like the cutaneous carbuncle consist of localized induration with central sloughing, opposite the mesenteric attachment and accom-

panied by surrounding hemorrhage and edema in the mucosa, and swelling of the solitary follicles, Peyer's patches, mesenteric glands and omentum and retroperitoneal tissue. The bacillus is found in these lymphadenoid structures in large numbers and sometimes in the chyle vessels and portal bloodvessels. The greatest changes most frequently occur in the upper small intestine, sometimes in the stomach or ileum, seldom in the colon and most rarely in the rectum. Thirty or forty foci may be seen in the small gut, but the lesions in the stomach or rectum are fewer and more discrete. Secondary embolism of the intestinal arteries from anthrax of the skin seldom cause hemorrhagic stools. Serohemorrhagic fluid may be found in the peritoneal cavity. Treatment by ipecac, calomel and bichloride of mercury is without effect.

2. **Respiratory Form.**—The respiratory form was described by Bell (1879), in England, as the "wool-sorters' disease;" and by Eppinger (1894) in Austria, as the "rag-sorters' disease." Infection may, on rare occasions, occur in the nose, larynx, tonsils or bronchi. The bacilli are capable of entering the uninjured bronchioles and alveoli. Wool-sorters' disease results especially from infection by hides imported from Russia and Brazil, and, in 1897, anthrax developed in this country from hides of Chinese origin.

Symptoms.—The affection begins suddenly with chill, high fever, tachypnea, dyspnea and bronchitic and irregular pneumonic findings; the viscid or hemorrhagic sputum in some instances contains the pathognomonic bacilli. The later symptoms are cyanosis caused by diffuse mediastinal infiltration; serous pleurisy, weak heart, vomiting, terminal collapse and antemortem fall of temperature. Icterus, hematuria and anthrax endocarditis have been observed. The sensorium is clear to the end in most fatal cases; convulsions and coma are due to meningeal hemorrhage. The patient may die on the second or third day. The mortality in rag-sorters' disease is 50 to 87 per cent., and rather lower in wool-sorters' disease. The bacilli enter the alveoli, wherein they produce alveolar desquamation and cellulofibrinous exudation—the analogue of the carbuncle—and much serohemorrhagic exudate, comparable to the skin edema. Where there is much cellular exudate, recovery may result from demarcation; where there is much serous infiltration the bacilli are abundant and the lymph vessels infect the mediastinum, peribronchial glands and pleuræ, which may hold quarts of anthrax-infected serohemorrhagic exudate.

Treatment.—The therapy is wholly supportive.

3. **Anthrax Septicemia.**—Anthrax bacillemia (septicemia) is a rare, fatal form, in which no atrium can be found.

GLANDERS.

Definition.—An acute or chronic contagion, mainly acquired from horses, caused by the *Bacillus mallei*, and characterized by nodules and ulcers, chiefly in the nose and skin.

Etiology.—Glanders and farcy were known to Aristotle and Hippocrates. The Bacillus mallei was discovered by Loeffler and Schuetz

(1882), and independently by Bouchard, Capitau and Charrin, which identified glanders and farcy as one disease. Weichselbaum (1885) first found the bacillus in human infection. It closely resembles the tubercle bacillus morphologically, but is shorter and thicker and measures 2 to 5 by 0.4 to 1.5μ; it is straight or slightly bent; its ends are somewhat rounded; it is usually single, and not motile. It stains with the alkaline anilines, easily in cultures but with more difficulty in sections, and the bacillus tinges unevenly, with lighter areas, resembling spores. It is a facultative aërobe and its most characteristic growth is on potato, on which a thin light yellow film develops in two days, soon becomes amber-colored and in eight days assumes a weakly red tinge with somewhat greenish-blue borders. The *atrium* is usually the skin, through which the bacillus enters by some small, perhaps microscopic lesion; or by horse-bites; infection may take place through the intact mucosa of the nose and air-passages, the conjunctiva, or very rarely, the digestive tract. Man is usually infected by chronically diseased horses; man-to-man infection is far less frequent. The bacillus is found in the nasal nodes (*glanders*); in the skin nodes (*farcy*); in the lymph vessels and glands (in which its virulence is greatest); in the excretions and blood current, where it occurs more frequently in man than in animals.

The bacillus soon dies in pus, but may endure for three or four weeks in the viscera of cadavers; it is rapidly killed by sunlight. Babes (1890) and Kalming (1891) isolated a toxin, and Kalming, during his researches, succumbed to acute glanders. Hellmann (1891) isolated mallein and Babes (1892) morvine, both of which are successfully injected for diagnosis and immunization. The horse, ass and mule are the most susceptible animals and from them other animals may be inoculated. In some localities half the horses have latent glanders (Babes). Infection is carried by the nasal or cutaneous discharge, whence glanders and farcy are chiefly observed in stable-boys, coachmen, farmers and veterinary surgeons. In 1909, Robin collected 156 human infections.

Symptoms, Pathology and Course.—These are considered under two topics: (*A*) Farcy, the cutaneous type and (*B*) glanders, the nasal type. In most cases the types are not wholly distinct.

(*A*) Farcy.—1. *Acute Form.*—The incubation is three to five days, attended by depression, nausea, headache and pains. The infected skin shows infiltration, which may heal only to break out anew. Lymphangitis and lymphadenitis are less marked than in the horse. Subcutaneous and cutaneous nodes or areas of diffuse infiltration develop. These lymphangitic farcy-buds or areas are infective granulomata, composed of epithelioid, lymphoid and white cells, and contain the bacilli, which are free, rarely intracellular and most abundant in the centre of the nodes. Early sections are succulent and show central necrosis, which is followed by suppuration and ulceration. The ulcers are painful, deep and crater-like, with sharply cut, everted borders and speckled bases; they have a thin, puriform, hemorrhagic or ichorous discharge and they often fuse. Thrombophlebitis, diffuse phlegmons or gangrene may develop. The initial fever may resemble the ladder-like ascent of typhoid, yet chills, polyarthritis and large muscular abscesses are fre-

quent. In some cases a successively macular, papular and pustular skin eruption resembles that of smallpox except that it is rarely umbilicated; it may be scant and scattered, very diffuse or even confluent; and it may affect the mouth, throat and conjunctivæ. The patient is often delirious, the pulse becomes rapid and small and fresh skin nodes develop, with ulceration or perhaps gangrene. The urine shows albumin, casts, or the diazo reaction, the frequent movements are offensive, and death occurs in two weeks from exhaustion or lung inflammation.

2. *Chronic farcy* shows the same changes, but they are gradual in onset, slow in progression and unaccompanied by essential inflammatory reaction or lymphatic invasion. For the first month or two there are pains in the limbs and joints, and then indolent swellings appear in the extremities and in the periarticular tissues, which ulcerate. The ulcers may cicatrize, but break out again after months, resembling lupus, which heals on one side and advances on another. Muscular abscesses may appear, most often in the pectoralis, biceps, brachialis and gastrocnemius. The course lasts for months or years (2–11). Recovery occurs in 50 per cent. of the cases, or death results from exhaustion and acute dissemination. In horses the greatly swollen lymph vessels and glands appear as sausage-shaped masses, whence the term "farcy" was used by Vegetius. In horses the skin eruptions and orchitis are more frequent than in man.

(*B*) GLANDERS.—1. *The acute nasal form* begins in the nose after an incubation of three days. Miliary nodules develop, which are elevated, yellowish-white in the centre, the size of a pea, surrounded by a pale red zone and ulcerate. New nodes form on the edges of the ulcers, which break down, fuse with them, and discharge a thin, puriform, dirty, sanguinolent fluid. Diphtheroid deposits or crusts form. Ulceration may erode the bone or cartilage and perforate the septum. The nose is swollen and eroded externally. The process reaches the mouth by lymphangitis or thrombophlebitis. The palate may perforate; the tongue, gums or tonsils are invaded; the angular lymphatic, parotid and submaxillary glands are swollen, and the larynx, and finally the lungs are infected. The leukocytes are increased. Beregin found microscopic nodes in the liver, kidney, spleen and brain; in the lungs they resemble miliary tubercles, or pneumonia malleosa may develop, accompanied by hemorrhage, abscess formation, gangrene, a distressing cough and raspberry, mucopurulent sputum. Secondary suppuration in the bones, muscles, joints or skin complete the pyemic picture. The course is that of acute farcy, though more rapid, and death occurs in two weeks.

2. **Chronic Nasal Form.**—This is similar to acute glanders except in its chronicity. It is less frequent, and when nasal involvement is slight it may resemble pulmonary tuberculosis, chronic coryza, pyemia or osteomyelitis. The malady may "slumber," to be aroused later by trauma or symbiosis with the staphylococcus. Death occurs from exhaustion or generalization by lymphangitis or thrombophlebitis. In Robin's series 6 per cent. recovered.

Diagnosis of Glanders and Farcy.—Typhoid, pyemia, variola, syphilis, lupus, tuberculosis and anthrax may cause confusion, but the history

and bacteriology determine the diagnosis. In thick smears, the chromatic bodies in the bacilli may simulate staphylococci (Zeit). The cultural findings are proved by intraperitoneal inoculation of a male guinea-pig, in which orchitis develops rapidly (Strauss's reaction, 1886). Injections of mallein differentiate in 90 per cent. of cases, producing fever of three degrees or more. The bacilli are agglutinated by the serum of normal horses in a dilution of 1 to 200, and of those subject to glanders in a dilution of 1 to 1000.

Treatment.—The prophylaxis of glanders is like that of anthrax—isolation of diseased, and cremation of dead animals, and strong antiseptics in wounds of persons exposed to infection. Skin ulcers are curetted and bandaged antiseptically, and if they are obstinate, touched with the live cautery. Infection of the nose demands carbolic douches, zinc chloride paste or iodoform. Arsenic, mercurial inunctions, quinine and alcohol are recommended. Injections of mallein and large doses of potassium iodide are recommended. Among others, Cramp records a recovery under vaccines.

TETANUS.

Definition.—Tetanus, or lock-jaw, is an acute infection caused by the tetanus bacillus and characterized by greatly increased reflexes, muscular rigidity and tonic spasms. Tetanus is literally stretching. It is described in Hippocrates's aphorisms.

Etiology.—In 1884, Carle, Rosenbach and Rattone produced the disease by inoculations of pus, and in the same year, Nicholaier discovered the *tetanus bacillus*, of which Kitasato (1889) first obtained pure cultures from wounds and from earth. The bacillus measures 3 to 5 by $\frac{1}{3}$ to $\frac{1}{2}\mu$ and is enlarged at one end, in which there is a spore, thus having a pin-head or drum-stick shape. It becomes thread-like in cultures. It stains readily by Gram's method, and is delicately flagellated and motile. It is anaërobic and cultures emit an onion-like smell. It grows well in sugar solutions. The bacillus is very enduring and the spores may live nineteen years. It exists in the primary wound and in the brain and cord in 44 per cent. of severe infections, so that tetanus no longer ranks as a simple toxemia. The bacillus has been found in the blood and sputum. Tetanus is traumatic; in the so-called idiopathic, rheumatic form, the *atrium* is either microscopic or pharyngeal. The bacillus produces *toxins* which are 100 to 400 times as toxic as strychnine. They are tetanospasmin and -lysin. Tetanus symptoms result from the tetanus toxins alone, but both infection and virulence are enhanced by association with various putrefactive microörganisms, and innocuous solutions of tetanus poison become lethal if combined with sterilized cultures of these putrefactive bacteria. The bacillus does not produce inflammation, though mixed infections are usual. Mice, guinea-pigs and rabbits are inoculable. In *nature* the bacillus is found in the alimentary tract of herbivora, whence it occurs in manure, garden earth, street or house dust, hay, putrefying fluids and splinters. The disease is ten to twenty times as frequent in the tropics as in temperate zones, because the bacillus thrives in heat. It may lurk in certain districts, as in Prague, the eastern

end of Long Island, or the West Indies where negroes are more often and more seriously affected than whites.

Mode of Infection.—Incised wounds are obviously less dangerous than punctures, gunshot or contused wounds or hematomata. Tetanus occurred less frequently in our Civil War than in the Spanish-American conflict, in which many cases developed among the Spaniards. It may result from circumcision, hypodermic injections, tooth-extraction and vaccination (95 cases), and Peterson (1910) collected 150 cases of post-operative tetanus, of which 70 dated since 1890; and in all, catgut was used. Parturition, lack of attention to the navels of the newborn, the use of imperfectly sterilized catgut, leech-bites or pin-pricks may cause its development. Chauffard (1903) collected 18 cases of infection from injections of gelatin for acute hemorrhage, Dieulafoy 11 cases after injections for aneurysm, Seibert 18 cases after antitoxin given for diphtheria, and preventive inoculations against the plague caused 19 deaths. The germ is very frequently found in gelatin, which should therefore be sterilized three times in very small amounts, each time for thirty to forty-five minutes at 212°. There is danger from the use of toy pistols and blank cartridges for Fourth-of-July celebrations; 415 deaths from tetanus are listed in the United States for 1903 as against 10 in 1912 (*Jour. Amer. Med. Assn.*); French clinicians describe a visceral tetanus originating from the intestines in which latter tetanus spores have been found.

Incubation.—This lasts from six to twelve hours in severe experimental inoculations, from two to nine days in less intense inoculations, and in man, in 33 per cent. of cases the symptoms appear in one, and in 66 per cent. in two, weeks.

Symptoms.—These appear suddenly. Ehrlich's side-chain theory gives a satisfactory explanation, the anterior horn cells of the cord and medulla combining with the toxins and causing the spasms. The first signs are *rigidity of the jaw* ("lockjaw," trismus) and *stiffness of the neck.* In most animals the muscles become rigid near the seat of inoculation first, and sometimes, in injuries received in war, the muscles of a limb may be first involved; the toxin travels along the nerve trunks. The horse, like man, develops trismus first. The poison incites the anterior cells of the cord and medulla to tonic contraction.

The *facies tetanica* results from zygomatic spasm, which produces the *risus sardonicus*—a smiling expression to the upper and a sad expression to the lower part of the face; the forehead is lined, the eyebrows raised and approximated, the eyeballs fixed, the masseters prominent, the teeth set and the nares dilated. The spinal extensors are tonically contracted, arching the head backward and the trunk forward (*opisthotonos*); the abdominal muscles are retracted stiffly; in rare cases the contracture is lateral (pleurothotonos), or forward (emprosthotonos), or the body is straight and rigid (orthotonos). The limbs are extended, the legs more so than the arms, and the feet and hands least. The spasms are chiefly *tonic,* but momentary clonic exacerbations may result from peripheral stimulation of the sensory nerves or those of special sense, by the least touch, jar, draught, light or sound. The *reflexes* are *enor-*

mously increased, particularly those of the skin. The spasms occasion violent muscular pain, oppression over the lower chest and epigastrium, speechlessness, insomnia and, from participation of the medulla, profuse sweats and increased and irregular heart action. The temperature may be high, normal or subnormal. After death a temperature of 114° has been observed. The *sensorium* is usually clear. Evacuation of the bladder and rectum is difficult, and erection or ejaculation may occur. The urine is decreased and sometimes contains albumin, sugar, acetone or indican, but no increase of urea and creatin. In some cases the lymph glands adjacent to the seat of infection are enlarged. In very rare cases the toxins cause death without muscular spasm; *i. e.*, they combine with other than nervous tissues. Axhausen collected 10 instances of localized tetanus, in which the disease did not extend from parts first involved.

Diagnosis.—The diagnosis is based on the nature of the wound, the Bacillus tetani, trismus (necessary for diagnosis), the facies and opisthotonos. Serum from cases of tetanus obtained by blistering, is fatal to white mice in twelve to twenty-four hours. In *strychnine poisoning* the history, tests for the drug in the stomach washings and urine, the more abrupt onset of the spasm and reflex excitability, the more diffuse and violent clonic intermittence of the convulsions, their greater involvement of the hands and feet, and the later appearance of trismus and its interparoxysmal relaxation, are entirely distinctive. *Hysteria*, hydrophobia and tetany (*q. v.*) are wholly different from tetanus. In *meningitis* the sensorium is affected. Trismus may be due to dental caries, parotitis, or temporomaxillary arthritis with a tender area in front of the tragus. Escherich's *pseudotetanus* is characterized by tonic contractures of the jaw, back and limbs after an acute infection and convalescence after a few weeks.

Prognosis.—The following factors are important:

1. Incubation.—The later after inoculation tetanus develops, the lower is the mortality; it is 91 per cent. if it appears in the first week, 81 per cent. in the second, and 53 per cent. if later.

2. Site of Infection.—Infection in the arms results in a 70 per cent. mortality; in the legs, 90 per cent. (Norris); in cephalic tetanus (*tetanus facialis* described by Charles Bell in 1830), the mortality is almost 100 per cent.; Brown (1912) found 94 records of cephalic tetanus, and in all but 1 case, the trauma was cephalic; in 84 per cent. the first symptom was trismus, followed later by various paralyses; in 16 per cent. paralysis antedated the trismus; paralysis may be facial or involve the pontine and medullary nuclei, which sometimes causes dysphagia (hydrophobic type) or paralysis of the eye muscles. The author observed these symptoms in a boy who had been kicked over the ear by a horse. Lloyd found 7 cases of facial diplegia. *Tetanus* or *trismus neonatorum*, especially described by Beumer (1887), is due to an infection of the navel in the second week of life. It prevents nursing and is fatal by the fourth day in 83 per cent. of cases. In the West Indies 50 per cent. of the children died of tetanus at one time, in the Western Hebrides, 67 per cent., and in Iceland, 100 per cent. Anders and Morgan collected

1307 cases (1850 to 1905). *Tetanus puerperalis,* particularly described by Heyse (1893), usually follows difficult labor, abortions or ministration by mid-wives, and is generally fatal. In three years there were 232 cases in Bombay (Gowers).

3. Course.—Hippocrates observed that patients who survive the fourth day may recover; 68 per cent. die in the first four days; 88 per cent. of acute and 75 per cent. of chronic cases die.

4. Trauma.—The death-rate in traumatic cases is over 80 per cent.; in the so-called idiopathic cases it is 50 per cent. or lower.

5. Sex.—More women recover than men.

6. Symptoms.—Slight trismus is favorable; high fever, involvement of the pons or medulla, respiratory spasm, dysphagia and ocular paralysis are unfavorable.

Treatment.—1. Prophylaxis concerns surgical antisepsis in *punctured wounds, hematomata,* trauma due to *blank cartridges* or *toy pistols,* in labor and care of the navel in the newborn. Immense credit is due the *Journal of the American Medical Association* in its campaign for a rational Fourth of July. Since the bacillus is anërobic, punctured wounds should be widely incised, treated with iodine, phenol or peroxide, and packed. In extensive injuries, extirpation of the regional lymphatics, or amputation may be indicated.

2. Antitoxin Therapy.—Behring and Kitasato (1890) discovered an antitoxin and were able to immunize animals by cultures heated or treated with Lugol's solution, which changes the toxins to toxoids (Ehrlich). The antitoxin is found in animals immune to tetanus, as crocodiles and chickens, and in the blood, milk and bile but not in the tissues. Tetanus antitoxin is not bactericidal, *i. e.,* it does not act on the bacilli. It neutralizes the tetanus toxin in the blood *before it has become fixed in nervous tissue.* From the above it is obvious that (*a*) serum prophylaxis may be successful in Fourth-of-July wounds; in the Prague obstetrical clinic it abolished tetanus after every other measure failed. (*b*) If used early it may neutralize circulating toxins which have not yet become attached to nervous receptors. (*c*) When the toxins have combined with the motor cells, it may abstract some of the virus fixed in the cells; 15,000 to 25,000 units for adults and 10,000 units for children (or 1500 and 1000, respectively, for prophylaxis) are injected daily near the site of inoculation. As absorption requires three days, intravenous administration has been advised (as well as subdural injection, which some consider dangerous). The antitoxin is eliminated in eight days. Anaphylaxis has resulted (*v.* page 88). Antitoxin reduces the mortality of established tetanus 20 per cent. (Permin).

3. Clonic convulsions may be minimized by absolute *quiet,* seclusion and darkening the room. *Morphine* hypodermically relieves pain and cutaneous reflex irritability, one-quarter grain every four to six hours for a day or two, but the pupils and respiration-rate must be carefully watched. An occasional corrective hypodermic of atropine should be given. *Chloral hydrate* depresses the motor elements of the cord; twenty grains may be given every two hours for three doses, but

even this amount may cause dangerous cardiac symptoms in alcoholics or arteriosclerotics. Digitalis may be given with chloral hydrate to steady the heart, and morphine and *potassium bromide* to enhance its depressomotor action and lessen the cutaneous irritability. The bromides are given, ʒiss every three hours for six doses, after which the interval should be lengthened. Inhalations of chloroform and amyl nitrite, or lumbar puncture may inhibit the onset of severe attacks.

4. Nutrition.—The nasal catheter or feeding by rectum overcomes the difficulties offered by trismus or dysphagia.

5. Other Measures.—(*a*) Baccelli secures better results with hypodermics of 2 per cent. *phenol* solution than with serotherapy (150 to 300 drops daily); in 190 cases, the mortality was only 17 per cent. (*b*) Wassermann advises subdural injection of an emulsion of sheep brains. (*c*) Meltzer and Auer use, after lumbar puncture, intraspinal injections of a 25 per cent. solution of *magnesium sulphate*—1 c.c. for each 25 pounds of the patient's weight; Kocher prefers a 10 or 15 per cent. solution; the magnesium paralyzes the motor nerves, and relieves the symptoms, giving the patient rest and ease; six out of seven of Kocher's patients recovered and in Fox's 24 cases the mortality was 46 per cent. The dangers are excessive bronchial secretion (relieved by atropine) and paralysis of the respiratory centre. In one case the writer observed retention of urine, maniacal delirium and quadriplegia. It must not be injected unless spinal fluid first runs out through the aspirating needle.

GONORRHEAL INFECTION.

Lightly spoken of as "the badge of virility," gonorrhea is often neglected. In its so-called latent form it may be conveyed to the wife, on whom it entails suffering, sterility and invalidism.

I. Regional Invasion.—Gonorrhea may invade the prostate, seminal vesicles or testes, the vagina, uterus, tubes or peritoneum, the rectum, bladder and kidneys and, in children, it causes institutional epidemics of vulvovaginitis.

II. Gonococcal Septicopyemia.—Thrombophlebitic or lymphangitic generalization has been discussed under Septic Infections. The fever is toxemic or septicemic, intermittent and often marked by great variations. The gonococcus has been found in the blood (Thayer and Blumer, 1895); myocardium; pleura (15 cases collected by Lemoine and Gallis, 1905); and in the joints (*v. i.*), kidneys, spleen, muscular abscesses and lymph glands; in meningomyelitic foci (30 cases, Barrié); in myositis (7 cases collected by Harris and Haskell, 1904); in arteritis and phlebitis (25 cases collected by Heller, 1904); in felons, osteomyelitis and periostitis; perichondritis (of the ears); horny skin eruptions or keratosis (21 instances, F. E. Simpson, 1912); 49 cases of ulcerative gonorrheal endocarditis were collected by Kulbs in 1907, of which only 12 were above criticism. Septicopyemic lesions may be due to Neisser's diplococcus alone or to mixed infections.

Gonorrheal arthritis is a form of mild septicemia—occasionally part of a septicopyemia. It was described by Selle and Sweidiaur in 1781,

but first clearly by Brande, 1854; the gonococcus was first found by Petrone (1883) and cultivated by Höch (1893). It develops in 2 or 3 per cent. of gonorrheal infections and is the most common disease of the joints. Eighty per cent. show an acute urethritis; in the balance the urethritis has subsided or become chronic. It has followed experimental gonorrhea, cuts during operating and primary gonorrheal conjunctivitis (18 cases reported by Lucas). About 90 per cent. occur in males, and 50 per cent. between twenty and fifty years of age. Too active treatment and sexual indulgence during the florid stage of gonorrhea are predisposing causes.

Symptoms.—Fever rarely exceeds 102°. The joints alone may be involved, or there may be more septicemic evidences, as endocarditis. Gonorrheal "rheumatism" is more than synovitis; the accessory bursæ, contiguous tendon sheaths and extracapsular structures are often involved—*gonorrheal arthritis.* It is often held that it affects one joint (monarticular) or at least few joints (oligarticular); though fewer joints are involved than in genuine rheumatism, in Gerhardt's series two to nine joints were involved in 87 per cent., and one joint only in 13 per cent. Gonorrheal arthritis frequently involves joints which usually escape in acute articular rheumatism, *e. g.*, the sacro-iliac synchondrosis, sternoclavicular, temporomaxillary and intervertebral joints. The knee is affected in 70 per cent., the ankle in 27, fingers and toes in 19, hip in 13, wrist in 12, shoulder in 10 and the elbow in 9 per cent., of the cases. Within the joint a greenish serofibrin exudes, and without the joint there is edema and infiltration. Suppuration may occur sometimes with or without coincident pyogenic infection, especially in the joints and tendons of the hand. Gonococci are found with variable frequency, the highest reported percentage being 75. Injection of a sterilized emulsion of gonococci may cause subcutaneous local reaction and induration, slight fever and local swelling in the joints (Bruck and Irons)—a form of cutaneous allergy. A positive complement-fixation test is indicative of gonorrhea.

König's *pathological classification* includes (1) *simple hydrops* of the joint, (2) *catarrhal* or serofibrinous *hydrops,* (3) *pyarthrosis* and (4) *gonorrheal phlegmon,* affecting the soft parts. A *clinical classification* may be made as follows: (1) *Arthritic form,* pain with exudation; (2) acute or subacute *polyarthritis,* by itself or with septicemia; (3) the *periarthritic form* (polytendosynovitis and polybursitis, often with periostitis); (4) *acute monarticular form,* with local reaction, sometimes with suppuration; (5) the *chronic monarticular hydrarthrosis* especially of the knee, a far less frequent form; and (6) simple arthralgia (the painful heel, talalgia or Jaquet's blenorrhagic foot).

Course.—The course is chronic and sometimes relapsing; iritis is fairly frequent; atrophy in the contiguous muscles may occur, or ankylosis, which is oftener plastic than osseous. In exceptional instances, sciatica, permanent arthritic changes resembling arthritis deformans (*q. v.*), exhaustion psychoses or multiple neuritis develop.

Treatment.—Recovery is a matter of time and nature, rather than of therapy. The salicylates are inert and the iodides unsatisfactory.

In the acute stage, immobilization by plaster casts or splints, and in the chronic stage, blisters, Pacquelin cautery, massage, passive movements and extension, to overcome residual exudation and contractures, are useful. Some cases are helped by Röntgen rays and superheated air (see ARTHRITIS DEFORMANS). Passive congestion frequently benefits. In intractable cases incision and drainage are indicated. Vaccine therapy is promising in chronic and subchronic complications; it is generally futile in vulvovaginitis of children, chronic deep urethritis and blenorrhagic endocarditis; in polyarthritis opinions differ; 25–100 million are used.

TUBERCULOSIS.

Definition.—A specific infection, characterized (*a*) etiologically by the Bacillus tuberculosis, (*b*) pathologically by nodes or tubercles, or diffuse tuberculous infiltration, and (*c*) clinically by symptomatology varying with the tissues or organs invaded.

History.—The term phthisis (wasting) was first used by Hippocrates. The history of tuberculosis includes several epochs:

Epoch I.—The anatomical tubercle was recognized by Sylvius (1614–1672), Morton (1689) whom some consider the real pioneer, Mangetus (1700) and Bayle (1810), who described the miliary tubercle. Laennec, in 1819, spoke for the unity of phthisis. Virchow held that caseation was only a retrogressive change and not necessarily tuberculosis. In 1857 Buhl described miliary tuberculosis. Cruveilhier, Rillet and Barthez identified scrofula and tuberculosis.

Epoch II.—Villemin (1865) positively established the inoculability of tuberculosis by experiments on animals. From the time of Hippocrates and Aristotle, tuberculosis was feared as a contagion, and the older pathologists, as Morgagni, dreaded to section phthisical subjects. The histology of the tubercle was developed by Wagner, Schüppel and Virchow; Langhans fully described the giant cells previously seen by Virchow and Rokitansky; tubercles were found in lupus (Friedländer, 1875), lymph glands (Schüppel, 1871) and fungus joints (Köster, 1876). The firm and ancient belief in its contagiousness led to the successful search for the cause.

Epoch III began with Koch's announcement, in 1882, of the tubercle bacillus. His report, as health officer in an obscure German town, was so complete that scarcely a single point has been added to his initial communication. The bacillus was found in all tuberculous lesions, such as chronic phthisis, miliary tuberculosis, intestinal ulcers, "scrofulous" lesions, bone and joint disease, lupus and in the sputum, in cattle, etc.; it occurred in no other disease; it was cultivated by him and inoculated into animals with reproduction of tuberculosis, thereby fulfilling what has become known as "Koch's law."

Bacillus Tuberculosis.—1. MORPHOLOGY.—It is a thin, slightly curved, narrow rod, measuring $1\frac{1}{2}$ to 4μ long; it contains no spores, the light unstained areas in its body being vacuoles or degeneration; it is immotile; in young cultures it is shorter than in the sputum; in old cultures and in lung cavities it is longer and is frequently thread-like. Pleomor-

phism is often noted, as bulging sides or ends, thread-like, or branching forms and forms like actinomyces. (See Plate V).

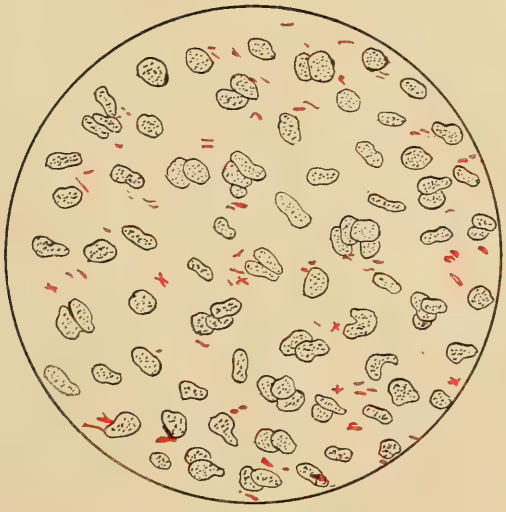
2. Staining Reaction.—It stains slowly and gives up the stain reluctantly. Ziehl's solution is the best (fuchsin 1, absolute alcohol 10 and 5 per cent. carbolic solution 100). The sputum is spread out in a dish over a black background and the yellow-green areas selected are thinly distributed on the slide which is held in the hand, and dried at some distance from the flame; it is then stained by the carbol-fuchsin, being passed repeatedly but lightly through the flame (without boiling) to deepen the stain; it is then washed, treated with 33 per cent. nitric acid, washed well in water and then alcohol, and dried. Uhlenhuth's method, as modified by Loeffler detects bacilli in 15 to 35 per cent. when not found by other methods.[1] The bacillus resists destaining because of its wax-like envelope, an almost pathognomonic feature. For clinical purposes only (*a*) the bacilli found in bronchiectasis and gangrene of the lung (*q. v.*), (*b*) the smegma bacillus and (*c*) the leprosy bacillus need be considered; the leprosy germs are intracellular, cover the nucleus of the containing cell, and are stained in six or seven minutes with alcoholic fuchsin, while tubercle bacilli are extracellular and stain slowly; the smegma bacillus causes many errors in genito-urinary examinations; it grows rapidly in twenty-four hours, does not show the beaded appearance seen in the Bacillus tuberculosis, and stained and counter-stained in the usual way, becomes tinged with methylene blue. Much found that some tubercle bacilli stain by Gram and not by carbol-fuchsin; he proved by culture and inoculation that they were Koch's bacilli and that they could be converted into the less virulent acid-fast form.

3. Cultures.—The bacillus develops best at body heat in one or two weeks; blood serum is the best medium, but growths are also successful on bouillon, glycerine-agar and potato; they are dry, thin, grayish and scale-like. The germ is a facultative aërobe and multiplies by transverse fission. It does not lead a saprophytic life in nature, outside the bodies of animals and of man.

4. Chemistry, Metabolism, Resistance, Virulence, Etc.—It contains two to sixteen times as much fat as other bacteria. A waxy substance envelops the bacillus; it also develops *tuberculin* whose chief components are peptone, albumose and alkaloidal substances; it is soluble in glycerin. It is not proved that it develops a definite toxin. Its *resisting powers* are as a rule limited. Drying is endured by the bacillus for six or even ten months. In decomposing media its average life is one to one and a half weeks. Heat (106°) kills it in a few minutes. It may survive 14° below zero for weeks. Sunlight kills the bacillus in minutes to hours.

[1] 5 to 20 c.c. of sputum are placed in a flask with an equal quantity of 50 per cent. antiformin and the mixture is boiled. Solution of the sputum occurs at once. To 10 c.c. of the cooled solution (which is sterile and from which cultures or inoculations can be made) 1.5 c.c. of a mixture of 10 volumes of chloroform and 90 volumes of alcohol are added and the whole thoroughly shaken. The specimen is centrifugalized about fifteen minutes. Chloroform is found at the bottom of the tube, and on its upper surface the sediment collects. The supernatant fluid is poured off, and with a pipette the sediment is transferred to a glass slide.

PLATE V



Tubercle Bacilli and Elastic Fibers.

Koch assumed an equal *virulence,* while the Arloing school finds that it has an attenuated virulence in glandular and osseous lesions, and Theobald Smith holds that bovine cultures are more virulent for rabbits than is human sputum. *Large numbers* are found in fresh tubercles; few are found in chronic articular, osseous or glandular foci, and inoculations may be necessary to demonstrate the nature of long-standing lesions. Arloing and Courmont found that serum from inoculated rabbits or goats, agglutinized *tubercle bacilli* in a dilution of 1 to 5 or 20, in two to twenty-four hours; agglutination was noted in 95 per cent. of the cases of lung tuberculosis and in 50 per cent. of the cases of surgical tuberculosis.

Associated or Mixed Infection.—Symbiosis has been noted with the streptococcus, staphylococcus and pneumococcus, to a lesser degree with the Bacillus pyocyaneus, influenza, etc. (*v. i.*)

Prevalence in Man.—Tuberculosis is the greatest foe of man and five million die yearly from this plague; it causes 14 per cent. of all deaths, and 33 per cent. of deaths between fifteen and forty-five years of age; it causes more deaths than war, famine, plague, cholera, yellow fever and smallpox combined; in Germany, in 1894, the deaths from diphtheria, croup, measles, scarlatina, pertussis and typhoid, together, were 7000 less than those from tuberculosis.

Quiescent (latent), obsolete or healed tuberculosis was found by Heitler in many postmortems upon those dying of other diseases (4.8 per cent. among 16,562 autopsies). Latent tuberculosis occurs in 25 to 33 per cent. of the population (Baumgarten, Birch-Hirschfeld), and 50 per cent. (Cruveilhier and Déjérine), 91 per cent. (Burkhardt) and in 97 per cent. of all necropsies (O. Naegeli).

Prevalence in Animals.—(*a*) *Cattle.*—In Germany it is found in from 4 to 20 per cent. In New York the tuberculin test was positive in 17 per cent. Theobald Smith, and later Koch, contended that bovine and human tuberculosis were separate infections; most writers incline to this view. In cattle tuberculosis is less often attended by softening of the tubercle, and the serous membranes are more often involved; the lungs and pleuræ are involved in 40 per cent., the lungs alone in 20 per cent., and the pleuræ and peritoneum in 15 per cent. (*b*) *Swine* are affected more often in Europe than in this country; in Saxony tuberculosis occurs in 1 per cent. and in Copenhagen and Dantzig, in 11 per cent.; because their infection commonly results from eating tuberculous offal, the type is alimentary (90 per cent.). (*c*) *Other mammals:* Dogs, cats, horses and sheep are rarely diseased. Monkeys in captivity die frequently (43 per cent.) from tuberculosis, referable to foul cages. (*d*) In *reptiles,* it is rarely seen except in captivity. (*e*) In *birds,* tuberculous lesions are more frequent in the liver, spleen, intestine, mesenteric glands, bones and joints, than in the lungs, though parrots suffer from skin and lung localizations. Avian, bovine, reptilian and human tuberculosis are closely related types. Tubercles are not uncommon in animals from nematodes, protozoa and bacteria and in man, from the streptothrix and aspergillus (pseudotuberculosis).

Atrium.—The details will be considered under the different clinical localizations of the disease.

1. By the Respiratory Tract.—The bacillus enters the respiratory tract by inhalation. In the vast majority of cases *the sputum* of diseased persons is *causative* both *of the disease* and its great frequency of localization in the respiratory organs. It has been estimated that a phthisical person voids three millions of tubercle bacilli daily. Cornet holds that the greatest danger lies in the *drying of the sputum* in the room, on the handkerchief, the lips, beard, hair, fingers and clothing or in the street and other open places. Flügge contends that dried sputum is rapidly sterilized by sunlight and is less dangerous than the moist expectoration, and demonstrated that in coughing, which seemingly voids no secretion, in singing, sneezing or talking, *small and almost invisible drops* of moisture containing the bacilli are voided; he cultivated bacilli from glass plates covered with solid culture media and held near the patient; he believes that minute particles remain in suspension in the air and thus infect other individuals. The ubiquity of the tubercle bacillus is exaggerated, though of great importance in poorly ventilated, dark dwelling-rooms, hospitals, cloisters, hotels or prisons, frequented by victims of tuberculosis. Cornet found the bacilli in the dust of such rooms in large numbers, and demonstrated that animals confined in rooms with dried tuberculous sputum readily acquired tuberculosis. Straus collected the germ from the nose in 31 per cent. of the attendants in tuberculous wards. *The closer the contact* with tuberculous subjects, *the greater is the danger of infection.* In crowded prisons in Austria the prison mortality is 40 to 60 per cent., against the general mortality of 11 to 15 per cent. In nursing sisterhoods, the death-rate exceeded 75 per cent., as observed by the writer in one small, closely cloistered community. In Philadelphia, Flick's studies showed that 33 per cent. of infected houses contributed more than 1 case. In our dispensary service, 7 rapidly fatal cases were observed in one family (Harpole). Oral breathing, caused by adenoids and inhalation of coal-dust or stone-dust, are favoring factors.

Dusting, sweeping or shaking the bedclothes disseminates the virus; there is less danger of infection when the windows and doors are open and when dusting is done with moist cloths rather than with feather-dusters. In hospital wards there are more germs in the air during the cleaning hour than during the other twenty-three hours. However, if bacilli are in the dust, they are not necessarily in the inhaled air; if inhaled, they may be caught in the upper air-passages whose secretions antagonize their development, and are often waved back by the ciliated epithelium of the lower air-passages; when bacilli are inhaled into the respiratory tract (*a*) no lesions may result, (*b*) the lungs may become diseased, or (*c*) the germs may pass the mucosa of the mouth or bronchi and cause cervical or peribronchial tuberculous adenitis. Cornet holds that the statement that "every man lives in the environment of tuberculosis" is exaggeration. Married persons, because of their intimate association, may contract the disease from each other, as was noted by Ettmüller and Morton in the seventeenth century.

2. By the Digestive Tract.—Bacilli may pass the mucosa, especially in children, and produce the first lesions in the mesenteric glands. Park and Krumwiede hold that in young children 6 to 10 per cent. of tuberculous deaths are due to the bovine type of bacillus, and that juvenile tuberculous adenitis and abdominal tuberculosis are more often due to bovine than the human bacillus. In pulmonary tuberculosis in adults, Park and Krumwiede did not once find the bovine bacilli in the sputum in 1042 cases; Kossel found in 709 sputa, 705 bacilli humani, 2 certain and 1 doubtful bacilli bovini and 1 with both types; in other words, the bacillus humanus is found almost exclusively in pulmonary tuberculosis of which twelve times as many people die as from all other localizations combined. In cows, a tuberculous mastitis was formerly thought necessary to milk infection, but milk may be infective from cows with tuberculosis which does not invade the udders. Pigs, fed on tuberculous milk, develop intestinal or pulmonary tuberculosis. Butter may contain tubercle bacilli. Meat is less dangerous, as it is usually well cooked, but tubercles in raw offal frequently infect swine. Harbitz's figures show that the entry of tuberculous infection is (i) respiratory in 41 per cent., (ii) digestive in 22 per cent., (iii) respiratory or digestive in 21 per cent., and (iv) in the lymph nodes, elsewhere or doubtful in 16 per cent. Gohn holds that 95 per cent. of infections in children are pulmonary or aërogenic, not lymphadenogenic, in origin.

3. Direct Inoculation.—Inoculation through the skin, genito-urinary tract, trauma or operations is far less significant.

Predisposing Factors.—*These are important, but tuberculosis is best considered and treated as an infection, communicated and acquired by direct personal contact.*

1. Heredity.—(*a*) *Hereditary Predisposition to Tuberculosis.*—A tuberculous family history is obtained in infected individuals in 25 per cent. Kuthri found essentially the same percentage of family tuberculosis in non-tuberculous as in tuberculous cases. Tuberculous parents may infect their children (50 per cent.), as they may infect others, but 97 per cent. escape the disease when removed from their parents. According to Rieffel, 3 per cent. of children acquire tuberculosis when both parents are sound and 22 per cent. when both parents are tuberculous. Hereditary predisposition is no obstacle to prevention and treatment. Cornick argues that heredity promotes resistance.

(*b*) *Congenital Tuberculosis.*—Baumgarten thought that children actually inherit the bacillus, which remains latent for years, and then develop an active tuberculosis. Congenital tuberculosis can be acquired only through the spermatozoön, ovum or placenta. The spermatozoön and ovum can not carry bacteria. Placental tuberculosis has been observed according to Warthin and Cowie in only 5 human cases, and in slightly over 100 cases in animals (Friedman, 1905). Virchow never saw a case. Usually there is a placental focus before the fetal blood is infected, but the bacillus may pass directly to the fetus. Schmorl and Geipel found tuberculosis in 50 per cent. of placentæ of tuberculous mothers.

The arguments against hereditary tuberculosis are (1) that children

born with the disease die very early; (2) that tuberculosis in the first months of life is extremely rare; (3) in congenital infection, the liver is chiefly diseased because of its vascular relations to the placenta; in tuberculosis of children, the lungs and glands are more often diseased, *i. e.*, respiratory infection is extra-uterine. (4) Removed from their tuberculous parents, children thrive well.

2. Environment.—Environment is but *another name for exposure or contact-infection.* Dampness and poor drainage *reduce the physiological resistance.* Trudeau showed that inoculated animals, kept in dark, damp cellars, die, while others, allowed to run at large, outlive the infection. Density of population raises the proportion of tuberculous cases. In recent years the large cities have shown an apparent decrease of tuberculosis, *e. g.*, Chicago, Glascow, New York and London. In Massachusetts the total is less by half than fifty years ago. The decrease is attributed to more intelligent hygiene. It is possible that this decrease is explained by the fact that pneumonia carries off the less resistant of the population; in Munich the death-rate from tuberculosis was 12 per cent. when typhoid was epidemic; when typhoid was practically abolished the tuberculosis-rate increased to 16 per cent., *i. e.*, 4 per cent. of those with tuberculosis had previously died from typhoid.

3. Occupation.—Work in close, damp rooms, in dust, in *crowded quarters,* particularly when associated with poor food, alcoholism, worry and overexertion, *lowers resistance* and promotes tuberculosis when *exposure to infected individuals* exists. Clerks, stenographers or waiters are more easily infected than are those working in the fresh air. Miners do not suffer more frequently, as dampness prevents the drying of infected sputum.

4. Age.—Cornet's figures show the following death-rate per 10,000 of population: First year 2 to 3, the child being in close contact with the mother. These figures grow smaller, until the lowest figure is reached, namely, ½ from the fifth to tenth years; fifteenth to twentieth years, 17 to 19; twentieth to thirtieth years, 32 to 36; thirtieth to fortieth years, 44; fortieth to fiftieth years, 55; fiftieth to sixtieth years, 76; sixtieth to seventieth years, 100; seventieth to eightieth years, 70; over eightieth year, 20. In the first decade of life, glandular, meningeal, osseous and cutaneous tuberculosis are more frequent.

5. Sex.—Before twenty years of age girls are more prone to tuberculosis, because their life is more sedentary. After twenty years males more often contract the disease, because of their greater exposure to infection. Repeated pregnancies and protracted lactations lower resistance to infection.

6. Climate.—Warm, dry climates, high altitudes and lack of sudden variations in temperature are inimical to tuberculosis. The French Alps and other localities were long free of tuberculosis until crowded by infected subjects. The chief advantages of many resorts seem to be their sparse population and inaccessibility.

7. Race.—In this country the Irish and negroes are most frequently diseased. The Indians have justly come to dread the "coughing white man," and they die in large numbers from consumption. These instances

are explained by exchanging a country for a city life. Infection among Jews is peculiarly infrequent.

8. Individual Predisposition.—The oldest medical writers described the winged scapulæ, paralytic thorax and depressed sternum; but poor development is often an expression of already existing tuberculosis; phthisis also occurs in splendidly developed chests, *exposure to infection* being the most important factor. Tuberculosis confers no immunity to relapses or to new localizations; a lung lesion may heal while a coincident intestinal lesion progresses.

9. Trauma.—Bacilli in the blood may be localized in a bone or joint by trauma; a blow on the head or spine may initiate meningitis in a tuberculous subject; pleurisy or pulmonary tuberculosis may be initiated by injury to the chest, arousing latent lesions in the peribronchial glands.

10. Other Factors.—These are *diabetes*, in which 25 to 50 per cent. die of tuberculosis of the lung; *psychoses*, especially of the depressive type; anemia, which, however, is more often the result than the cause of tuberculosis; *heavy colds*, *measles* and *pertussis*, and less often scarlatina, influenza or diphtheria, may predispose to infection, or more probably often awaken slumbering glandular tuberculosis by the irritative action of products absorbed from the bronchi or lungs. Enterocolitis in children or typhoid in adults may in a similar way awaken or initiate abdominal tuberculosis. *Contracted kidney*, *cirrhotic liver*, *sclerosed arteries*, stenosis of the pulmonary artery, aneurysm and a hypoplastic aorta are predisposing factors. Rokitansky noted that valvular disease of the left heart prevents or checks infection, because it congests the lungs; Bier's congestion treatment of surgical tuberculosis is based on this point; Birch-Hirschfeld, in 4359 autopsies, found chronic pulmonary tuberculosis in 21 per cent., and in 107 autopsies of valvular heart disease it was present in but 2.8 per cent. Potain, in 54 cases of mitral stenosis, found pulmonary tuberculosis in 16.6 per cent. (see Valvular Disease of the Heart). Syphilis, cancer, pleurisy (*q. v.*) and hemoptysis (*q. v.*) are occasional predisposing factors.

General Histopathology of the Tubercle.—The only *pathognomonic feature of the tubercle is Koch's bacillus*, as similar infective granulomata are seen in strongylus in sheep, in actinomycosis, syphilis, etc. Baumgarten described the changes following inoculation of the anterior chamber of the eye: (*a*) On the first day the bacilli increase and spread along the lymph spaces. (*b*) By the sixth day, the fixed connective-tissue cells proliferate under the stimulus of the bacillus; these *epithelioid* or *endothelioid cells* are large, rounded or polygonal, with distended nuclei, sometimes contain tubercle bacilli and show karyokinetic figures; the bacilli multiply and the endothelial cells of the vessels proliferate. In this stage it is called the epithelioid tubercle. (*c*) On the tenth day leukocytes emigrate, an inflammatory reaction to the toxins secreted by the bacillus; most of the white cells are polymorphonuclear, many of which degenerate; later there are *mononuclears* which do not suffer retrogressive changes; the mononuclears crowd the epithelioid cells and dominate the histological picture, especially with complicating trauma (lymphoid tubercle). (*d*) The *reticulum* of the tubercle consists of the

stretched fibers of the infected tissue. Tubercles contain no bloodvessels. (*e*) The epithelioid cells increase in size and are often multinucleated. From them or the extravasated leukocytes, by growth or fusion, *giant cells* develop; they contain many large, oval nuclei and tubercle bacilli; the bacilli may number 50 to a cell in very active processes or even may be the chief histological finding in very chronic forms, such as lupus, scrofulous glands or fungoid joint disease; they are few or absent in miliary tuberculosis; there seems to be an antagonism between the nuclei and bacilli, for if the nuclei lie on the margin of the cell, the bacilli are centrally grouped, and if the nuclei lie at one pole of the cell, the bacilli concentrate at the other pole. The cellular inclusion of the bacilli is possibly a phagocytosis. The completed miliary tubercle is non-vascular, cellular, millet-seed (miliary) in size, gray in color and translucent or opaque from later caseation (*v. i.*); it is the invariable pathological unit of all tuberculous lesions and, more or less, is a continuous process. In the diffused inflammatory tubercle, the diffuse areas result from the fusion of myriads of miliary tubercles.

The tubercle then undergoes one of two *degenerations*, caseation or induration: (1) *Caseation* results from degeneration of the tubercle, beginning at its centre, from ischemia (vascular obliteration) and coagulation necrosis (the bacillus and its toxins). Inoculations with dead bacilli may cause tubercles and even caseation. The area of coagulation necrosis then gradually becomes more yellow and granular, and finally results in an amorphous, homogeneous, cheesy debris. Caseation is a dangerous, destructive degeneration. (2) *Induration* or encapsulation results from growth of connective tissue around the conglomeration of tubercles; it limits the tubercle, is the measure of the resisting power of the surrounding tissue, of the process of bacteriolysis, and is a conservative change. It occurs most frequently in the peritoneum, but also in the lungs and other tissues. *Inflammation*, varying with the tissue involved, may complicate the histological findings. Suppuration may result from mixed infection with streptococci, though Koch held that pus may be formed by the tubercle bacillus itself; the ordinary tuberculous "cold abscess" is not actual pus, but merely thick, amorphous, granular debris.

Extension.—(1) Tubercles develop at the point of infection and in the nearest lymph glands, which permanently or temporarily arrest the process. (2) Infection may occur through the uninjured mucosa or skin, but the process rarely passes beyond the tributary lymph glands. (3) The later advance of the process can be traced clearly at autopsy, along the lymph vessels.

I. Tuberculosis Miliaris Acuta.—Definition.—An acute general, sometimes almost universal, embolic dissemination of the tubercle bacillus in great numbers by the blood or lymph stream, resulting pathologically in the wide-spread formation of miliary tubercles in various tissues, and clinically in fatal toxemia, pulmonary, meningitic or other symptoms.

Etiology.—1. DEVELOPMENT.—Acute miliary tuberculosis *always develops from a tuberculous focus*, which is found in 97 to 100 per cent. by careful dissection; it may be small or even miscroscopic; von Buhl,

who first fully described the affection (1856), recognized that miliary dissemination occurred by absorption from old tuberculous foci, as pyemia results from local suppuration. It is curious that miliary tuberculosis occurs from foci so small as to be easily overlooked, and that large tuberculous lesions seldom result in miliary dissemination.

2. Lesion.—A description of all primary foci would enumerate every possible localization of tuberculosis. *Pulmonary tuberculosis* causes over 50 per cent. of cases of acute miliary tuberculosis. Glandular lesions rank next, and articular, osseous, genito-urinary and serous-membrane tuberculosis cause most of the balance. The more the tubercle is indurated, the less is the danger of dissemination. Acute generalization only follows the introduction into the circulation of *large numbers* of *bacilli*, for they do not multiply in the blood. A few bacilli reaching the circulation cause a solitary tubercle. Repeated ruptures of moderate numbers of bacilli cause the so-called chronic miliary tuberculosis (*v. i.*).

3. Access of Bacilli into Blood Stream.—Sir Astley Cooper, in 1798, found tubercles in the thoracic duct. In 1877, Weigert demonstrated that *tubercles invade the vessel walls*, which Rokitansky had declared immune. Tuberculosis reaching a vessel usually causes its gradual obliteration and thus localizes the process, but in some cases bacilli reach the lumen of the vessel and are distributed in the direction of its current. The most frequent process is periangitis, by which a tubercle adheres to and empties into a vein. Less common is endangitis, by which bacilli lodge in the intima, and, grouping there, further disseminate the infection. The most common method is *periadenitis of a tuberculous bronchial gland*, its adhesion to a pulmonary vein and final rupture into it. Tuberculous lesions may rupture into the azygos, cavæ, pulmonary artery, aorta, thoracic duct or jugular or other veins.

4. Organs Involved.—(*a*) Rupture into a pulmonary vein floods the *arterial circulation* with bacilli, whence miliary deposits occur in the brain, choroid, liver, spleen, kidneys, myocardium, bones, serous and mucous membranes and in the bloodvessels themselves. (*b*) Rupture into a portal venous radicle affects chiefly the liver. (*c*) Into the pulmonary artery, the lungs and (*d*) into a peripheral artery, the tissue supplied by it.

5. Predisposing Factors.—Miliary dissemination is most frequent *in children*, especially between one and four years of age, because the circulation is faster, metabolism is quicker, thrombosis of the vessels is less frequent, tissue reaction is less marked and the lymph vessels and spaces are wider. *Depleting factors*, pregnancy, the puerperium, trauma, bone operations, *measles*, *pertussis* and mixed infections are somewhat predisposing causes.

Symptoms.—The clinical picture varies greatly, depending on the quality (virulence) and quantity of bacilli; the toxins thrown into circulation from the primary focus; and the localization of the tubercles formed. *Toxemic precede local symptoms.*

Types.—Waller (1845) distinguished the following forms:

1. Typhoid Type.—The febrile onset may be gradual or sudden, with or without chills. The patient often exhibits only *great toxemia*

with little local reaction from the tubercle, therefore with few local findings; this type may be due to a large caseous focus flooding the blood stream with toxins. The *status typhosus*, delirium, headache, continuous or weakly remittent fever, enlarged spleen, diazo reaction and bronchitis suggest typhoid but the fever is more variable and the pulse runs higher; less frequently roseolæ, meteorism, diarrhea and intestinal hemorrhage may cause even a closer resemblance to typhoid (see page 46). Unless the meninges or lungs are involved the diagnostic confusion may be insuperable. Epistaxis is less common; hyperesthesia and herpes are more common but all symptoms may be deceptive except the typhoid bacillemia and Widal reaction in typhoid, as opposed to the choroidal tubercles, etc., in miliary tuberculosis. Acute miliary bacillemia is more common in children than in adults, and runs a course of ten to twenty days, or somewhat longer. The writer saw typhoid and miliary tuberculosis in the same subject.

2. Respiratory Type.—In this type *the toxemia* is *subordinated to the local miliary eruption. Tachypnea, dyspnea* and *cyanosis, without proportionate physical findings* are almost unmistakable; the respiratory urgency and rapid pulse, without cardiac disease and with a *tympanitic pulmonary resonance* declare the diagnosis. The following forms are less characteristic: *Pneumonia* may be closely simulated by the fever, chills, pain, cough, rusty sputum, increased breathing, dyspnea, cyanosis, or even by tubular breathing (in children). *Capillary bronchitis* (bronchopneumonia) requires differentiation after recent measles, or after pertussis with patches of atelectasis. Diagnosis should be made from senile bronchitis and emphysema in the aged, in whom the miliary type is frequently afebrile. Tubercle bacilli in the sputum or urine may occur accidentally. The course averages three or four weeks.

3. Meningeal Type.—Tuberculous meningitis appears as the only apparent trouble, whereas it is but a part of a miliary dissemination. Meningitis occurs in 80 per cent. of cases of miliary tuberculosis. The increased respiration, deep breathing and increased irregular pulse are suggestive and the choroid findings, lumbar puncture and Kernig's sign are corroborative. In children who are prone to this type of miliary tuberculosis, toxemia may closely simulate actual meningitis, as in Henoch's cases, where only brain edema was found.

4. Other Types.—The mention of these infrequent types shows the liability of diagnostic error; the *latent type*, marked only by catarrhal symptoms, and often called grippal; the *ambulant type*, with death after a few days of indisposition; nephritic; apoplectiform, delirium tremens, suffocative or asthmatic type; and the fulminant type, where the patient dies in a few hours. Sepsis, miliary carcinosis, uremia or the cyanosis of nitrobenzol poisoning may be suggested.

Individual Symptoms.—1. Temperature.—Continuous fever may be observed in very diffused acute forms, but it is lower than in typhoid. *Remittent or intermittent* fever is more common; miliary tuberculosis following pulmonary phthisis may change neither the hectic fever of the latter nor its euphoria. The *typus inversus* usually lasts but a few days. In 17.5 per cent. of cases the course is afebrile; involvement of the

meninges, hemorrhages, vomiting or diarrhea may depress the fever; low temperatures are common in the aged, in ambulant, bronchitic and cardiac types.

2. Circulation.—(*a*) The *pulse is fast in proportion to the fever,* 120 to 140–160; the blood-pressure is low but dicrotism is infrequent; a pulse under 100 is very rare (in old people or meningitic localization). (*b*) The heart is somewhat dilated and an apical systolic murmur, with accentuation of the second pulmonic sound, is frequent. On rare occasions a pericardial rub is due to roughening of the serosa by the miliary eruption. (*c*) Tubercle bacilli were first found at autopsy in the blood by Weichselbaum and Meisels, and clinically by Lustig and Rütimeyer. In the death agony from lung tuberculosis, bacilli may enter the blood. (*d*) The *leukocytes* are usually not increased; Matthes notes increase in the polymorphonuclears.

3. Respiration.—Early symptoms are frequent. (*a*) There is very often a severe *continuous cough, always a suspicious symptom.* It is due to the toxins or to irritation of the vagus twigs by miliary nodes in the lung; it causes muscular pain, voids little sputum and ceases later if the brain becomes involved. (*b*) The *breathing* is *increased* to 40 to 60 in adults, and to 80 to 90 in children. (*c*) *Dyspnea* and *cyanosis* develop from irritation of the vagus or involvement of the diaphragm. The *tachypnea, dyspnea and cyanosis are peculiarly significant* diagnostically in that they have no adequate explanation in the physical findings. (*d*) The sputum is scant, foamy white and mucous; it seldom contains dots of blood, and is rarely rusty without complicating pneumonia. It may be more abundant in subacute cases. (*d*) The *physical findings are not as marked as the urgent respiratory symptoms would indicate.* A *tympanitic note* is very frequent, resulting from relaxation of the lung, or acute emphysema from coughing. Dulness only results from older tuberculous foci in the lung, or from complications. Auscultation reveals *fine rales,* especially suggestive over the upper chest, where stasis is uncommon. The *x*-rays reveal small, bright pin-head spots early in the disease. Jürgensen noted a fine pleural rub, due to pleural tubercles. Terminal pulmonary edema is frequent. At autopsy innumerable translucent nodes are scattered through the lung, most apparent on oblique illumination.

4. Nervous System.—Nervous symptoms may predominate, and are toxemic or organic: (*a*) The *toxemic symptoms* are headache, dulness, vertigo and tinnitus; these are followed by delirium, muscular adynamia and tremor, and eventuate in stupor, broken by fits of delirium, and coma. In rare instances coma may develop early in an apoplectiform fashion, or the sensorium may be normal. (*b*) *Meningitic symptoms* may develop, such as headache, retraction of head, spinal tenderness and rigidity, paralysis of the cranial nerves, Kernig's sign, convulsions and coma. The cerebrospinal fluid is generally under increased pressure and contains globulin, lymphocytes and often tubercle bacilli. The findings at autopsy are those of tuberculous meningitis (page 150).

5. Digestive System.—The tongue is coated and there is anorexia and constipation. Nausea and vomiting are usually meningitic. Diarrhea

is infrequent, but bloody stools may occur with tympany. Ascites is infrequent in miliary tuberculosis of the peritoneum (as distinguished from tuberculous peritonitis). Litten once heard a friction rub over the liver. Miliary tubercles may occur in the stomach and bowels, most frequently in children.

6. Choroidal Tubercles.—These occur in 16 to 75 per cent. of cases, after one or several weeks. They occur singly or in large numbers in very generalized tuberculosis, usually in both eyes, as whitish-yellow protuberances with irregular washed borders. They become large and cause so much atrophy of the retinal pigment layer that they can be seen through it. Daily examination for them is necessary. (See Plate VI, Fig. 6.)

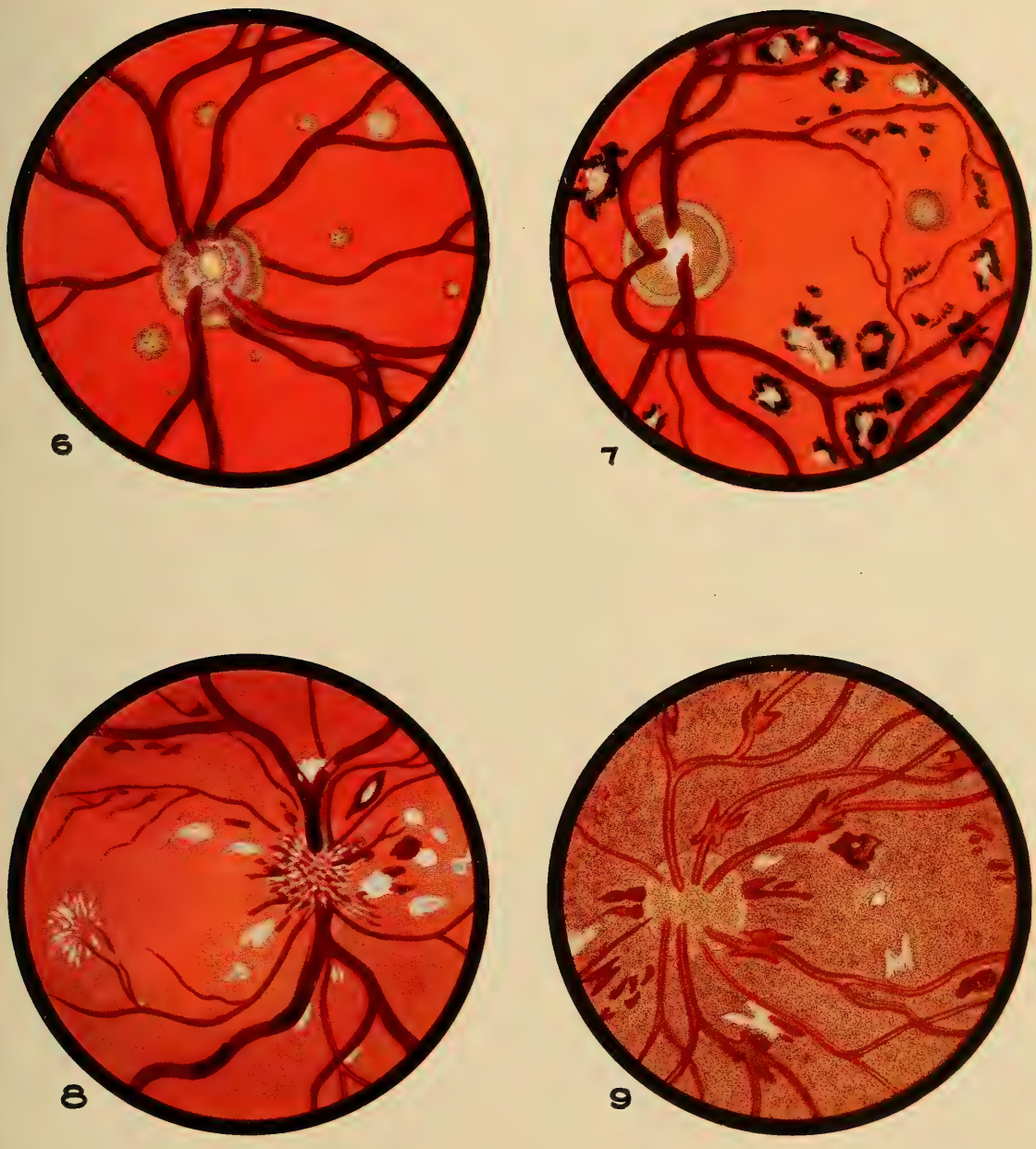
7. Other Symptoms.—One of Wunderlich's cases was reduced to one-quarter of his ordinary weight. The *spleen is enlarged* in 70 per cent., but is usually smaller than in typhoid, except in children, where it may assume great dimensions, with swelling of the lymphatic glands. The urine is concentrated and dark, may contain albumin and often shows the diazo reaction. Hemorrhages into the gums, nose, retinæ and intestines are infrequent. The skin is pale, cyanotic, damp and sudaminous. Herpes may be observed, as well as rose spots, petechiæ and terminal edema; miliary tubercles in the skin were found by Tileston, especially in children; the pin-head eruption is a red papule, becoming vesicular and finally encrusted and appears in crops on the buttocks, thighs and genitalia; in 71 per cent. the tubercle bacillus was found.

Remissions may occur, even for a month or two, the toxemia subsiding while the tubercles continue to develop. The fall of the fever resulting from brain implication may be deceptive. As a rule the course is acute, even stormy or peracute, and death may occur before the tubercles have time to form. Severe infection is fatal in two or three weeks, moderate infection in one or two months. Chronic miliary tuberculosis is rare. Recovery is almost impossible; Waller and others believe that it exceptionally occurs, even with bacilli in the blood. Therapy is unavailing; symptoms should be treated expectantly, as in typhoid or meningitis.

II. Tuberculous Meningitis.—Etiology.—Most cases occur between the *second and tenth years* (75 per cent.), some in adolescence and few late in life. Tuberculosis in children is usually generalized, and miliary tuberculosis is in 80 per cent. associated with meningeal disease. Tuberculous meningitis is always secondary to pulmonary or glandular tuberculosis, less frequently articular, genito-urinary, cerebral and other localizations. *Measles, whooping-cough,* typhoid or pneumonia may seemingly arouse latent tuberculous foci. Trauma is of occasional clinical and medico-legal importance. In the case of a baggage man thrown upon his occiput an acute tuberculous meningitis resulted in death; the autopsy disclosed an apical focus, from which the bacilli infected the injured brain. In one instance, meningitis and coma followed an anesthetic, given to examine a tender spine, and was fatal within a week.

Pathology.—The diagnosis is made *macroscopically* in the majority of cases. The exudate is gray, yellow or green, gelatinous, purulent or serous and covers the base from the chiasm to the medulla (basilar

PLATE VI



Retinal Findings in the More Important Diseases.

6. Tubercles in choroid. 7. Syphilitic choroidoretinitis. 8. Albuminuric retinitis. 9. Hemorrhages in pernicious anemia. (From Sahli.)

meningitis). Tiny tubercles are found on the nerves and along the Sylvian artery where "they look like lice eggs on a hair." Careful search is sometimes necessary for their detection. They are more clearly seen in the translucent arachnoid, and on superficial examination look like minute air bubbles. They are seen frequently over the convexity or between the two hemispheres. The ventricular fluid is increased and tubercles and inflammation of the ependyma are not uncommon; the external and internal communications of the ventricles may be obliterated. The substance of the brain shows ischemic or encephalitic softening. The cerebrospinal fluid is increased and is clear or cloudy, rarely purulent. In the cord the exudate may be a quarter of an inch in thickness, and is largely disposed over the posterior part of cord because of the patient's dorsal decubitus; the membranes sometimes appear granular, as "though sown with fine sand." Wartmann found tubercles in the substance of the cord in 87 per cent. of his cases. The cranial and spinal nerve sheaths are bulging. *Microscopically,* exudation is found. The nodes are proved to be tubercles; bacilli are found in them and in the cerebrospinal fluid. The microscope in some cases proves the existence of tuberculous meningitis not visible to the naked eye. Neuritis of the cranial and spinal nerves is constant. The optic, oculomotor and facial nerves are the most often affected. The usual histopathology of tubercle is seen with tuberculous endarteritis and periarteritis, small encephalitic and myelitic foci, swelling of the neuroglia and degeneration, especially of the axis-cylinders.

Symptoms.—*Prodromes* are more prominent in children than adults; fever, emaciation and anemia are due to preëxisting lung or glandular tuberculosis. Irritability, change in disposition, tendency to cry, inability in children to play or in adults to work, unrest, insomnia and headache may develop in a week to three months. Trauma, measles or pertussis may be the inciting cause. The onset, in 86 per cent. develops more gradually than in the epidemic form, so that the classical description comprises *three stages:* (*a*) The *stage of irritation,* with headache, vomiting, remittent fever reaching 102°, the *cri hydrencéphalique* of Coindet, night terrors, pain and rigidity in the neck and spine, small, irregular pupils, delirium, grinding of the teeth, hyperesthesia, rigidity of the limbs, the neck sign (page 91) and Kernig's sign (in 22 of Koplik's 82 cases); (*b*) the *stage of brain pressure,* with paralytic and irritative conditions in the eye muscles, as ptosis or strabismus; with paralysis of other cranial nerves or monoplegia or hemiplegia from involvement of the cortex, and frequently aphasia and dulness; with general or Jacksonian convulsions; slow, irregular and variable pulse; retracted abdomen, constipation, continued vomiting; sighing respiration; and very frequently with optic neuritis; and (*c*) the *stage of paralysis,* in which the vomiting and often the headache cease, absolute coma develops, and the rapid pulse vacillates in rate and rhythm. Biot's alternating apnea and hyperpnea or Cheyne-Stokes's breathing appears, emaciation is extreme, the pupils are wide and immobile, the eyeballs roll upward, and a typhoid state with dry tongue or abdominal distention prevails. Temporary remissions of days or weeks are occasional, in which the

pulse remains irregular, but the patient lapses again into coma, the fever rises very high (107°) or falls low (even to 93°), and death occurs in some weeks from cardiac or respiratory failure, dysphagia or inanition. This division into stages is not wholly satisfactory, because the stages may overlap but the progression of symptoms is somewhat as described.

Tubercles in the choroid are found clinically by Eichhorst in 17.5 per cent. of cases. Heinzell did not find them in 41 cases. At postmortem examination Litten found them in about 75 per cent. (See Plate VI.) *Leukocytosis* is absent and the spleen is enlarged in 33 per cent.

Many variations exist. The process may be wholly *latent*, being masked by miliary tuberculosis (or meningeal symptoms may dominate when the greatest pathological changes are in the lungs). Coma may be sudden, resembling apoplexy, especially in adults; in these cases the hemiplegia probably results from changes in the arteries. Only rarely is the course very chronic. It may appear to be a psychosis. In adults the course is more rapid, the headache and delirium more marked and the convexity more frequently involved than in children. In Holt's series, three-quarters of sporadic meningitides were tuberculous and most arose from contact with infected adults.

Death is usual, but Bokay collected 37 undoubted recoveries (1914). (See CEREBROSPINAL FEVER for differentiation and treatment.)

III. Tuberculosis of the Lungs (Phthisis, Consumption).—The lungs are involved with great frequency, not because they are peculiarly susceptible, but because aërogenous infection is the most frequent cause of tuberculosis. Hematogenous infection, as in miliary tuberculosis, and lymphogenous infection also occur.

Pathology.—The tubercle bacillus localizes in the bronchi (tuberculous lymphangitis), bronchioles or alveoli (tuberculous pneumonia), and causes tubercles as described in the general pathology. There is cellular hyperplasia of the alveolar, epithelial and endothelial cells, and fibrinous inflammation develops. Isolated tubercles, by fusion cause the conglomerate tubercle.

Extension.—(*a*) *Lymphatic extension* is the most common form. In young individuals the lymph paths are wider and absorption greater, so that intoxication and bacillary migration are more active. Absorption is greater after trauma, pneumonia, measles, pertussis, emaciation, *e. g.*, as convalescing typhoid, etc. Bacilli may pass the bronchi and reach the glands at the hilum, which in turn may infect the blood or break into the lung. Extension is usually along the lymph vessels in the bronchial walls (lymphangitis tuberculosa). (*b*) Extension by the *air passages* is common; when a tubercle ruptures into the bronchi, its contents may be coughed or waved upward by the ciliated epithelia if they are intact; on the other hand, a deep breath may aspirate, or coughing may impel, infected particles into sound areas, where they develop new foci; if the bacilli are living, an active tuberculosis develops; if dead, a lobar or lobular pneumonia is more common. (*c*) Extension by the *bloodvessels* may cause a miliary dissemination in the lungs or in the entire body. Preliminary to the clinical description in which special pathological features will be described, it may be said that some caseated

areas fuse into large infiltrations; some encapsulate or calcify and thus effect a temporary arrest or a permanent recovery; other caseated areas soften, forming ulcers and cavities.

Symptoms.—A clinical description covering all types is impossible, as the course may be galloping or extremely chronic; one form is marked pathologically by caseation, another by ulceration; one symptom, as hemoptysis, may indicate the type and may be the first and chief symptom; in some persons the symptoms suggest other diseases and the physical findings are indeterminate; in others the findings are remarkably dissociated from the usual symptoms. Therefore the symptoms and signs usually found will be covered first and the special types will be dealt with afterward:

1. Local Respiratory Symptoms.—These are usually the first noted, but may only appear months after the primary focus has begun to spread; they are due to the local tubercle and its toxin.

(*a*) *Cough.*—Cough is one of the first symptoms and is *the most constant symptom;* it is usually present from the incipiency to the close of phthisis. It is due to *toxins irritating the vagus* twigs, which irritation is referred to the medulla, from which issue muscular contractions with the glottis closed, to evacuate the slight secretion; the sensitive interarytenoid space and tracheal bifurcation initiate this reflex. Less often coughing is due to pleurisy, pharyngitis, enlarged bronchial glands or laryngitis. It is slight at first, and has no characteristic, other than being dry, difficult and paroxysmal. Later it becomes more frequent but less difficult as the secretion increases. In some cases it is most severe in the morning, the secretion having accumulated over night, while in others it is most severe at night, for the lungs become congested by the dorsal decubitus; saliva reaching the larynx during the night, may cause a sudden explosive cough. It is provoked by dust, smoke, variations in temperature, opening windows or getting into the cool bed, by exertion, laughing or taking alcohol. It is less conspicuous in the phlegmatic, aged and insane. When obstinate it may invoke constant vomiting and induce great emaciation.

(*b*) *Sputum.*—The sputum is first *caused by the toxins*, and later by the *secretion* from the tuberculous focus. At first it is glairy and contains myelin, sago-like bodies composed of transformed alveolar cells. It later becomes yellow from admixture of pus, yellow, green or red from saprophytic organisms, and blood may give it a red, rusty, flesh-like or chocolate hue. Other bacteria and saprophytes are frequent (*v.* Bacteriology). It tastes sweet and smells slightly fetid. Its amount varies from a few globules to over a pint. It is most abundant in cavities; it stagnates before death, because of the patient's muscular weakness; children are prone to swallow the sputum. *Nummular sputum* from cavities is flat, "coin-like," greenish-gray, extremely abundant and sinks in water, as it is airless; it occurs in other cavities as in bronchiectasis. The *only characteristic* of the sputum is the *tubercle bacillus* (*v. s.*); it may be seen before the advent of any physical finding, but in other cases it must be sought repeatedly, as it appears *only when ulceration communicating with a bronchus* develops. The tubercle bacillus is found

in only one-third of the cases of incipient tuberculosis. Falk and Tedesko found that salicylic acid may be detected, after administration by the mouth, in any of the serous fluids and inflammatory exudates, but not in the saliva or bronchial secretion. If found in the sputum it is believed to be evidence of involvement of the lung substance itself. *Elastic fibers* are found in 80 per cent., and originate from the vessels or parenchyma of the lung. They are elongated and twisted, and sometimes shreds of the vessel wall are seen; those from the lung substance often show its alveolar arrangement. Elastic fibers can often be located by flattening the sputum between two plates of glass and picking out the yellowish areas for microscopic examination. Treatment with caustic potash and centrifuging, facilitate their detection when they are scanty. They merely denote a destructive process, hence are also found in abscess, gangrene, infarction and pneumonia (see Plate V). Detritus, pus cells, red cells, alveolar, bronchial and buccal epithelium, myelin droplets and pigment, are also found. The so-called lung calculi are sometimes voided; they are calcified particles from old areas of caseation, from bronchial obstruction, or from peribronchial lymph glands; from the glands large calcareous masses may cause fatal obstruction of the trachea, or suffocation may be averted only by immediate low tracheotomy. The significance of albumin in the sputum, emphasized by Roger and Levy-Valensi, is not great.

(*c*) *Hemoptysis.*—Hemoptysis (hæmoptoë, spitting of blood) occurred in 37 per cent. of Prior's 1000 cases; others place it at 66, even 90 per cent.; it is most frequent in adult males and is rare before puberty. (*a*) *Early hemoptysis* is due to *erosion of a small vessel* by tubercles in its perivascular lymph sheath. The toxins, by causing coagulation in the vessels in and near the affected lung area, usually prevent extensive early hemorrhage. Hemoptysis is the first symptom in 4 per cent. of phthisis cases and in 16 per cent. the only symptom; in most cases, however, other symptoms develop; bacilli and elastic fibers may be seen at the time of bleeding. Reiche recently corroborated the old statement that early hemoptysis is not unfavorable. The bleeding develops in quietude in 66 per cent. of cases but may be induced by coughing, laughing, exertion or excitement; there may be a premonitory rise of blood-pressure. Often without cause, it comes on suddenly with a warm, salty taste in the mouth and the evacuation of a dram or more of bright frothy blood, clear or mixed with mucus; small particles of darker blood are brought up, for a few days, after which it disappears; in 4125 of the Brompton Hospital cases, 69 per cent. evacuated less than half an ounce. Bubbling rales may be heard on the side of the lesion, and moulds of the bronchioles may be voided. F. Stricker collated 900 cases of hemoptysis in the German army; in 480 cases there was no apparent cause and of these 87 per cent. were tuberculous; of 43 cases brought on by exertion, 74 per cent. were tuberculous; and of 24 cases due to trauma, half were tuberculous. The occurrence of early hemorrhage, before other symptoms of phthisis, led Hippocrates, Boerhaave and Van Swieten to think that hemoptysis caused phthisis, while Laennec, Louis and Traube correctly regarded it as a symptom of already estab-

lished phthisis; the earlier idea is explained by the fact that blood and bacilli may be aspirated into other parts of the lung, in which the process develops anew; fever, rapid pulse and dyspnea following hemoptysis are ominous (*v. i.* ACUTE FORMS). (*b*) *Late hemoptysis* may be slight or profuse. Profuse bleeding is usually due to erosion of aneurysmatic bloodvessels running across, or in the walls of cavities (Rasmussen); Kidd found ruptured aneurysms in 70 of 82 fatal cases; occasionally larger vessels, as the pulmonary artery, are ulcerated. A pint or a quart may spurt out of the mouth causing acute anemia, suffocation or rapid exsanguination. Blood may accumulate in large cavities, never reaching the air passages. Hemoptysis is usually a sign of tuberculosis but also occurs from other causes (*v.* DISEASES OF THE LUNGS).

(*d*) *Dyspnea.*—Just as it is absent in pleurisy of *gradual* onset, it is often absent even when the respiratory area is enormously contracted (great infiltration, great cavity formation or induration of two-thirds of the respiratory space); in sudden miliary tuberculosis the dyspnea is great. In some cases dyspnea may follow pressure of glands on the

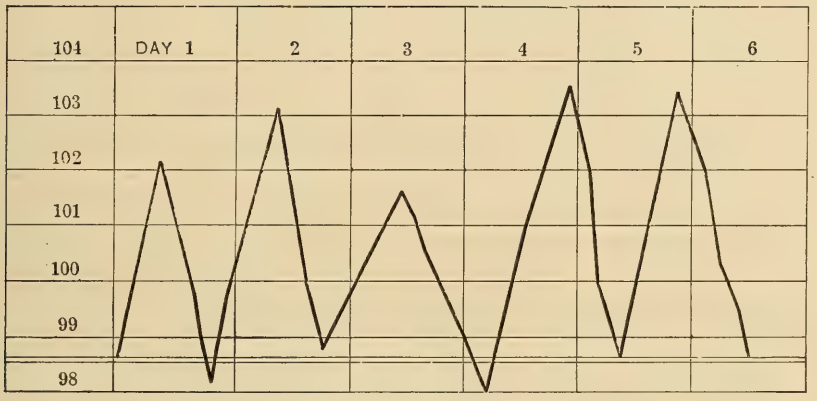


FIG. 13.—Hectic fever curve in phthisis.

vagus, stagnating secretion, extensive vicarious emphysema or massive pleural adhesions, yet it is *most often due to the rapid heart action.* In the last stages it may cause orthopnea and result from intercurrent pneumonia or pneumothorax. Germain Sée described a tuberculous pseudoasthma.

(*e*) *Pain.*—Pain is observed in 66 to 75 per cent. of cases. Its location may be apical, diaphragmatic, anteriorly from the clavicle to the third rib or under the scapula. Its character is sticking and intermittent, but not intense; the skin is often hyperesthetic. Its cause is *chiefly acute pleurisy,* for lung disease and chronic pleurisy are generally painless; other causes are muscular fatigue from coughing, intercostal neuralgia and pneumothorax.

2. GENERAL SYMPTOMS.—These result *from absorption of the toxins* secreted in the local lesions.

(*a*) *Fever.*—This is present in the great majority of cases and is almost invariable in the early stages. Probably the dominant cause is the tuberculous toxin. Fever marks the tubercle formation, fusion of foci, advance of the disease, degeneration of the tubercle, and probably

secondary infection, though the associated streptococci are usually far from virulent. Enzymes and their action upon the body cells (autolysis) may be causative. The cessation of fever is synchronous with temporary or permanent arrest of the disease, as manifested by improved health, strength and weight. It is easily increased by exercise, excitement or trivial causes.

Types.—There is no typical curve nor can fever be classified as initial or ulcerative fever. The vesperal hectic is the most common curve, and indicates slow disintegration and absorption of toxins. Intermittent fever may resemble malaria and usually indicates rapid and marked tissue-disintegration; remittent fever most often occurs late in the clinical course; continuous fever occurs early or in acute consumption, but rarely resembles the typhoid curve; in some unfavorable cases the fever falls from high to subnormal registers, sometimes with collapse; the *typus inversus* with morning exacerbation and vesperal fall is very uncommon. Fever may be absent in patients with old cavities and fibrous induration. Chills have no relation to the fever or its intensity. As fever is indicative of toxemia, it is often associated with anorexia, accelerated pulse and emaciation.

(*b*) *The Blood.*—(i) The red cells rarely number less than three million; anemia may antedate all other symptoms, whence the frequent confusion with chlorosis. Rarely severe anemia of the aplastic form or with hemolytic icterus develops. Again, anemia may be inconspicuous or late. In some cases the red cells are increased, notably in individuals prone to hemoptysis. (ii) The hemoglobin averages 75 per cent.; it may be decreased more than the number of red cells (*chloranæmie tuberculeuse*). (iii) The leukocytes may be normal; they are increased in cavity formation (secondary infection), caseous pneumonia or protracted pyrexia; after injection of tuberculin the eosinophiles show a decided increase. Steffen holds that the lymphocytes are increased early and the polynuclears later (mixed infection). (iv) Recent reports of positive bacillemia are ascribed to acid-fast bacilli in the water employed in staining.

(*c*) *Sweating.*—Night sweats, more important than in any other disease, occur in 90 per cent. of cases (Louis), and are due to the action of toxemia on the sweat-centre. Heavy coverings sometimes induce sweating. The term "night sweats" refers to their usual occurrence between 2 and 5 A.M. They are more frequent in acute than in chronic phthisis, in younger than in older subjects, when cavities are forming and the fever is variable.

(*d*) *Pulse.*—The rapid pulse is associated with indigestion, muscular adynamia and malnutrition (*v.* page 166).

3. Physical Signs.—In adults *the apex* of an upper lobe, more often the right than the left, *is first affected* (Laennec). The reasons ascribed are various, such as a poor apex blood supply, slower lymph current and less expansion—which are scarcely proved. More probably coughing drives infection upward from the bronchi, and the clavicle or first rib compresses the bronchi passing to the apex and thus lessens its ventilation. The summit of the apex is not invaded first; Birch-Hirschfeld,

in early cases which died from causes other than tuberculosis, found that in 24 per cent. the disease began in the fourth or fifth order of bronchi in the upper lobe and that the posterior were affected earlier than the anterior divisions.

(*a*) *Inspection.*—(i) The nutrition may be good, but, later, emaciation is marked. The face is pale, the cheeks hectic, the nose pointed, the eyes hollowed and glistening, the neck thin and the chest is altered in various ways; in some cases it is long and narrow, with wide interspaces, sharp costal angles, prominence of Louis's angle, atrophy of the scapular muscles and more vertically directed ribs; in others it is flattened anteroposteriorly and the ribs are closely apposed; in still others the funnel-chest may be seen. Laennec recognized that the "paralytic chest" was the result and not the cause of phthisis. Examining the patient in a good light, the following may be noted: (ii) *Deficient expansion* above or below the clavicles, especially when examined from behind and above; in the lower chest, impaired excursion of the diaphragm. Deficient expansion may be due to pleural adhesions, to atelectasis of the lung resulting from caseous nodes obstructing the bronchi, to a cavity, or to induration, an effort toward encapsulation and recovery. (iii) Distention of the chest may be due to vicarious emphysema, effusive pleurisy or pneumothorax. (iv) The cardiac impulse may be widened by retraction of the left lung away from the heart.

(*b*) *Palpation.*—Palpation shows (i) *decreased expansion*, elicited by standing behind the patient with the thumbs above either clavicle and the fingers below it, or by standing in front of the patient and spreading the hands over the anterolateral parts of the thorax. (ii) *Increased vocal fremitus*, obtained when the lung is infiltrated (caseous foci or pneumonia), indurated or the seat of cavities, all of which increase conduction of the voice-sounds from the bronchi. Superficial show more clearly than deep lesions; thick, pleural adhesions or plugging of the bronchi with caseous material decrease the fremitus. (iii) The systolic distention of the pulmonary artery and the second pulmonic closure are better felt when the lung retracts from the heart or when it is infiltrated in its vicinity. (Pottenger describes intercostal rigidity over the disease focus.)

(*c*) *Percussion.*—(i) Incipient infiltration gives a short percussion note, which later becomes *dull;* the apex is first involved in 88 per cent. of cases. Dulness may be found under the middle of the clavicle, at which level Birch-Hirschfeld found the anatomical beginning of tuberculosis. The apices are best percussed when standing behind the patient; the right apex is normally less resonant than the left. The lung lying behind the clavicle can be tested by gently tapping the clavicle with one finger. To detect slight early alterations in note, percussion should be gentle, symmetrical parts should be compared, the patient should hold his breath in full inspiration and the muscles must be relaxed; each patient has his own percussion note, varying with his muscle, fat and bone. The apex reaches 1 to 2 inches above the clavicle, and behind to the *vertebra prominens*. A focus as large as a cherry may cause dulness, but usually impaired resonance is produced only by consolidation measuring $1\frac{1}{2}$ by $2\frac{1}{2}$ inches in area and within $\frac{3}{4}$ inch of the surface, and foci of these

dimensions lying deeper than 2 inches escape detection. Small, multiple or deep foci give a tympanitic note. The dulness may extend from the clavicle to the second and third costal cartilages, then to 1.5 inches below the apex of the lower lobe (level of the fifth dorsal vertebra); this area is almost always involved when the upper lobe shows distinct consolidation; extension occurs outward and downward along the interlobar fissure; then it extends to the apex of the upper lobe on the other side.[1] Often the first dulness of consolidation is found behind, in the supra- or interscapular region. (ii) Shrinking, fibrous induration and a thickened pleura give apical or other dulness. Sometimes the lung becomes vicariously emphysematous about an old focus which is thus hidden clinically and pathologically. (iii) A tympanitic or cracked-pot resonance may be obtained over cavities (*v. i.*).

(*d*) *Auscultation.*—This is *the most important aid*, next to the microscopic examination of the sputum. (i) The vesicular murmur, especially at the apex, is altered, for detection of which the patient is best examined without deep breathing or coughing; the jerky "*cog-wheel*" *breathing*, described by Jackson (1833), may be due to small tubercles in the bronchi, somewhat stenosing their lumina; it also occurs in normal individuals from forced, muscular breathing, or pounding of the heart on the lung during inspiration. The breathing may be *wavy*, *weak*, *indeterminate* or *puerile*. Rude, rough inspiration is due to peribronchial tubercles compressing the bronchioles. The *expiration* is frequently *prolonged*, *rougher* and *higher pitched*, in apical "catarrh;" it is long, sharp and bronchial, in older foci; what seems clinically to be catarrh is often consolidation pathologically. In comparing the breathing, inspiration on one side should be compared with that of the other, and expiration similarly noted. The breath-sounds are physiologically higher pitched on the right than on the left side. (ii) *Bronchial breathing* is caused by various consolidations (tuberculous, pneumonic, neoplastic or gangrenous) and by cavities, tuberculous and otherwise. Bronchial breathing of moderate degree is physiological in the interscapular region, but is less intense than that of consolidation. (iii) *Rales* are rather an evidence of infiltration (consolidation) than of "catarrh." If they are heard in the apex, the presence of tuberculosis should be suspected, though they may occasionally occur there physiologically, or after influenza. The vibrations of the accessory muscles of respiration, when the patient takes a long forcible breath, are easily differentiated from rales after a little experience. Rales may not be heard unless the patient breathes deeply or coughs; auscultation should be made before, and then after, coughing noiselessly and inspiring with the least muscular force. In incipient consolidation, small, crackling, crepitant rales are heard over the alveoli and smaller bronchioles at the end of inspiration; they later become larger, ringing and resonant, indicating that the alveoli and bronchioles have become airless over a considerable area, or that softening or formation of cavities is in progress; scattered, clear, large, crackling rales are heard over small cavities in the apex or the smaller bronchi, and indicate

[1] There is a non-tuberculous shrinking of the right upper lobe, in mouth-breathers, due to collapse of the apex [Krönig], or to tuberculous glands about the right bronchus (Bing)

a viscid secretion. Clicking rales are generally attributed to softening; they may occur near the heart as it beats upon an adjacent infiltration. Gurgling and consonating rales suggest cavity or bronchiectasis. In emphysematous patients, whose chests exhibit sibilant and bubbling rales, diagnosis of developing tuberculosis may be most difficult. (iv) *Bronchophony* (a bronchial quality imparted to the voice-sounds) is heard when the patient speaks during auscultation; it indicates infiltration or cavity formation but is absent when the bronchi are plugged. Often testing the whispered voice is of value. *Egophony* is merely a higher grade of bronchophony and occurs as Skoda demonstrated, in cases of consolidation, in medium-sized cavities and above pleural exudates. (v) *Pleural friction* results from fibrinous pleurisy or rough turbercles in the pleura. (vi) A systolic bruit over the subclavian artery is probably due to pleural adhesions to the artery.

(*e*) *Röntgen Rays.*—Shadows may be due to consolidation, thick-walled cavities, calcified foci, gangrene, pleural callus or pleural exudate. The excursion of the diaphragm can be followed; shadows, due to the muscles, may be readily eliminated by moving the arms.

Summary.—The above findings may be grouped together, because they are found together clinically, one part showing consolidation, another, cavity formation or induration. (*a*) The *signs of consolidation* are decreased expansion, increased vocal fremitus, initial tympany followed by dulness, high-pitched expiration, then distinct bronchial breathing, crepitant rales and bronchophony. (*b*) The *signs of induration* are retraction or lagging behind of the fibrous area on breathing, increased vocal fremitus, dulness, usually large rales, bronchial breathing and bronchophony. (*c*) The *signs of cavity formation* are: (i) Increased vocal fremitus, if it is near the surface; (ii) tympanitic note, which may increase in intensity when the cavity is percussed with the mouth open (Wintrich's change of note), when percussed during inspiration (Friedreich's change of note), or with the patient in the sitting posture (Gerhardt's change); the note may be metallic; the cracked-pot note, Laennec's *bruit de pot fêlé*, is obtained in cases of superficial cavities measuring $2\frac{1}{2}$ inches with thin walls, which communicate with an open bronchus by a small opening; variation in the percussion note from accumulation (dulness), or emptying of secretion (tympany), is most suggestive of cavity formation; percussion may cause the patient to cough and void much sputum; (iii) the breathing, which is loudly bronchial, or if the cavity is quite large, metallic, amphoric or metamorphosing (which begins normally and suddenly becomes bronchial or *vice versa*); (iv) bronchophony and egophony; (v) large, resonant rales, which are the most frequent and important finding; metallic rales in the apices are most suggestive of cavities; they may acquire an amphoric character in very large, thin-walled excavations; (vi) nummular sputum; (vii) systolic, sometimes booming or metallic murmurs, due to propagation of the heart tones; they may be heard at some distance from the patient.

Cavities are due to the breaking down of caseous areas and are usually connected with the bronchi, into which their emptying is partly favorable, in that the infective material is evacuated; and partly un-

favorable, because it opens up avenues for infection by aspiration. Once formed, they generally remain or increase; they may leave fistulous cicatrices (Laennec's *cicatrices fistuleuses*); if they are held by adhesions the cavities persist, contracting cicatrization being impossible; there is danger of their further extension by secondary infection, or of gangrene. The cavity has a well-marked limiting membrane, from which pus, nummular sputum, elastic fibers, and grayish, granular detritus composed of masses of tubercle bacilli are voided; the cavities are frequently multilocular, may heal on one side and extend on another, and sometimes occupy an entire lobe or even an entire lung. They may rupture into the pleura, pericardium or bloodvessels; the author saw a rupture into the mamma, resulting in diffuse subcutaneous emphysema.

Stages of Pulmonary Tuberculosis.—Tuberculosis usually proceeds for six to eighteen months before any symptoms appear.

1. Phthisis Incipiens.—The general and local symptoms appear as above described, with fever, anemia and rapid pulse. The modes of onset are various. The most frequent picture is that of a patient with an obstinate cold or a "grippe" which does not regress; the sputum may show tubercle bacilli and the apex some rales. In others, disordered digestion and a rapid pulse awaken suspicion. The voice may be lost, passing as a simple laryngitis. There may be only a moderate depreciation of health, languor and neurasthenic manifestations; in young girls a chlorosis may be hastily diagnosticated. Malaria is a common escape in explaining an obscure fever. Tuberculosis may appear in other more directly suggestive forms, such as hemoptysis, pleurisy or cervical adenitis. The physical findings are often uncertain or absent.

2. Phthisis Confirmata.—Distinct physical findings indicate not incipient phthisis, but confirmed, established phthisis.

3. Phthisis Consummata.—This is advanced consumption, with marked hectic fever, cavity formation and emaciation.

Course.—The average case of "chronic ulcerative phthisis," with the symptoms and signs already described, stands midway between the following very acute and extremely chronic groups:

1. Acute, Galloping or Florid Consumption.—This type constitutes 10 per cent. of phthisis and develops from some old focus, as tuberculosis of the bronchial glands, or a small cavity which by intrabronchial rupture disseminates the process; possibly the *virulence of the germ* is greater in these cases; diabetes, alcoholism, *measles, whooping-cough* or influenza, may be the immediate cause.

(*a*) *Acute Pneumonic Form.*—The acute pneumonic form is acute caseous pneumonia. This term indicates the great clinical resemblance to, and confusion with, acute lobar pneumonia; but it must be remembered that genuine lobar pneumonia does not caseate, and that this form is not synonymous with tuberculosis complicated by pneumococcic pneumonia, which, in this association, runs its usual course. Acute caseous pneumonia begins in an individual in seemingly perfect or but slightly reduced health. An initial rigor is fairly common, or spitting of blood may be the first symptom. There are often herpes, cough, pain in the side, rapid breathing and sudden fever, which frequently imitate

exactly the onset of genuine pneumonia. The resemblance may persist but often certain variations arouse suspicion. The fever is remittent or intermittent, and more variable than in lobar pneumonia; dyspnea and cyanosis are less frequent than a gradually increasing pallor; sweating is usually copious. In a few days dulness prevails over a lower lobe but much more often over an upper lobe or over an entire lung. The breathing at first indistinct, may remain so, but often becomes bronchial. The sputum is often typically pneumonic, viscid and rusty, or of an olive or grass-green color; elastic fibers are seldom found. Tubercle bacilli are sometimes found, but in this case originate not from the fresh pseudo-pneumonic patches, but from the causal focus; in few cases can the physical signs of the primary cavity in an upper lobe be elicited. The physical findings change but little, though the crepitant rales are replaced by coarse mucous rales; the anticipated crisis does not occur, and most cases are called unresolved pneumonia until the increasing anemia, loss of strength or rapid pulse, suggests tuberculous pneumonia. Albuminuria is uncommon; the diazo reaction is present. Five-sixths of the cases die within six weeks; in some few cases considerable regression may occur and it is barely possible for the course to last three months or more. The *findings at autopsy* are an old focus of tuberculosis, a ruptured cavity or gland; from it caseous material, containing bacilli and tuberculous toxins, is aspirated into many finer alveoli and bronchioles, where it excites a miliary, caseous, lobar pneumonia. The inflammation is due wholly to Koch's bacillus or its toxins, and is (i) partly fibrinous and desquamative, red and white polynuclear and mononuclear cells being poured out; proliferative changes are noted, as the epithelioid and giant cells seen in the typical tubercle; caseation ensues, as shown in the already fused and fusing yellowish-gray areas; (ii) the most rapid exudation is serous, Laennec's "gelatinous or smooth pneumonia," probably due to the tubercle toxins and not to mixed infection; this part of the inflammation may regress without caseation. Cavities rarely form in this type.

(*b*) *Acute Disseminated Form.*—It may occur as a special type, or may complicate the ordinary form of phthisis. Following the rupture of a small area of softening, usually located only at the postmortem, and following dissemination by aspiration, there develop (i) fever, repeated hemoptysis, chills, rapid pulse, increasing dyspnea, diffuse crepitant or bubbling rales, delirium, or even a status typhosus. (ii) The sputum is absent or scanty, and the bacilli are sometimes absent throughout the course. Death may occur acutely in three or four weeks without physical signs having appeared and without breaking down of the foci; or a remission may take place, after which there is (iii) breaking down of the *indistinct* foci which cause slight *dulness* and distant *bronchial breathing*, simulating lobular pneumonia. At autopsy scattered, yellow, partly caseous, peribronchial, nodular foci are seen; in some cases the presence of tuberculosis is proven only by microscopic examination, for, without caseation, the foci may resemble ordinary bronchopneumonic patches; secondary infection with streptococci is said to cause these lesions, but they result from the tubercle bacillus alone.

(*c*) *Acute Disseminated Ulcerative Form.*—This type is often observed in children between two and six years of age, in pregnancy, alcoholism, diabetes and influenza. The foci, which vary in size from a pea to a walnut, are widely disseminated; they fuse, soften early and develop cavities without walls, which are only ulcers or necrotic caseous material. There are present clinically hectic or continuous fever, pronounced anemia, exhausting sweats, rapid emaciation and viscid, purulent and bacilli-laden sputum; diffuse, fine and large rales and bronchial breathing may be heard, but the physical signs are often vague. The galloping course ends fatally in one to four months.

(*d*) *Acute Miliary Form.*—Acute miliary tuberculosis (*q. v.*) develops in 3 per cent. of cases of pulmonary phthisis.

2. Chronic Tuberculosis.—(*a*) *Chronic Ulcerative Form.*—The ordinary phthisis may run a course which is long latent, except for anemia, dyspepsia or other indistinctive symptoms. After the detection of an apical catarrh, the cough, fever, sweats and anorexia may subside; another relapse may occur after years of quiescence, or exacerbations develop, with loss of weight and strength. Recovery may occur, even with pronounced physical signs. New tuberculous manifestations may be reinfections rather than relapses.

(*b*) *Phthisis Fibrosa.*—The fibrosis excited by the tubercle bacillus may begin acutely or insidiously. Auclair thinks the bacillus secretes two toxins, one with a necrosing action which leads to caseation, and another with an indurating action which leads to fibroid phthisis. There may be no cough, sputum, bacilli, fever, emaciation or anemia. Ulceration may result only from mixed infection. Sometimes there is hemoptysis. If left-sided, the spleen rises higher and the retraction of the lung away from the heart makes the cardiac shock more diffuse, and the pulsation of the pulmonary trunk more visible. If right-sided, the liver rises, the right ventricle is apparently enlarged (from lung retraction) and is often actually hypertrophied and dilated from increased tension in the pulmonary circuit. The heart may be drawn to the right or left by the inevitable pleural adhesions, bronchiectases are not uncommon, the sound lung is vicariously emphysematous, and the diseased lung is smaller, flatter and depresses the shoulder, impairing expansion and often presenting bronchial breathing and large rales.

(*c*) *Pleuritic Form.*—The pleuritic form follows small latent foci in the lungs, lymph glands, etc. (See Pleurisy.) Exudates not due to rheumatism, inflammation in contiguous organs or tumor always suggest tuberculous pleurisy. Pleurisy, in fact, more often precedes than succeeds pulmonary tuberculosis and repeated attacks are frequent, with moderate, irregular or continuous fever, perhaps with rales in the apex of the affected side (not the rales frequently heard just above the upper level of the exudate), or with dulness in the contralateral apex. Bowditch, following up 90 cases of pleurisy, found that 33 per cent. developed phthisis.

Variations According to Age.—1. Children.—Tubercle bacilli pass the bronchial mucosa and produce tuberculosis of the bronchial and mediastinal glands; the wide lymph vessels and more active metabolism have

already been noted; the process in the glands attacks secondarily the hilum of the lung and the lower lobes (90 per cent.), which explains the frequency of miliary tuberculosis from involvement of the thoracic duct and bloodvessels.

(*a*) *Chronic Generalized Tuberculosis.*—The younger the child the more general is the tuberculosis. Measles, pertussis and influenza are prone to arouse a latent tuberculosis in the bronchial glands. The course is generally more active than chronic, like that of tumors in children. Emaciation, anemia, enlargement of the spleen and liver, and moderate intumescence of the peripheral lymph glands are the variant features of this type. Hectic fever is seen chiefly in children over three years. The sputum is swallowed, but washing the stomach recovers bacilli-laden sputum.

(*b*) *Acute Miliary Tuberculosis* (*q. v.*).

(*c*) *Localized Lung Tuberculosis.*—(i) The acute caseous type resembles pathologically bronchopneumonias of other causation; the microscope may be necessary to detect its nature. Hemoptysis is rare; Henoch observed over a dozen cases under six years of age. (ii) Chronic forms are very infrequent under six years.

In children, physical findings are often absent except rales between the third and fifth ribs in front or over the lower lobes. Cavities are rare, and if softening develops do not freely communicate with the bronchi. In half the cases the wrong diagnosis is made (Henoch). Percussion is more difficult than in adults, because of the small chest and its thinner walls. (A cracked-pot resonance can be obtained in a normal young chest by strong percussion.) Children breathe irregularly, and puerile respiration is physiological. Induration is rare.

2. The Aged.—Hemoptysis is rare, physical findings are less frequent than in adolescence, because the breathing is weaker, and emphysema is more common. Cough and fever are less conspicuous.

Diagnosis of Pulmonary Tuberculosis.—Early diagnosis is most important. (i) A positive *family history* is of some value, but it does not establish the diagnosis in a doubtful case, nor does its absence exclude tuberculosis. (ii) The *symptoms of onset*, toxemic, digestive, etc., are important. (iii) The *Bacillus tuberculosis* distinguishes pulmonary tuberculosis (*a*) from other catarrhs, such as acute and chronic bronchitis or influenza; (*b*) from other consolidations, *e. g.*, pneumonia, bronchopneumonia, neoplasm or syphilis; (*c*) from other vomicæ, such as abscess, gangrene, actinomycosis, echinococcus, syphilis or bronchiectasis; and (*d*) from indurations other than tuberculous. *Every sputum should be examined. Cultures and inoculations* are made in doubtful cases. (iv) In doubtful cases *repeated physical examinations* are imperative; auscultation is more valuable than percussion; too great reliance should not be placed on negative findings; coincident lesions in a lower lobe and in an upper apex are almost surely tuberculous. Many patients are needlessly alarmed by precocious diagnoses.

(v) *Tuberculin Tests.*—The principle is the same in all; the tuberculous subject develops a hypersensitiveness to the tuberculous poison—known as the allergistic reaction, *i. e.*, a specific reaction of the organism

to poisons with which it is already acquainted; the tuberculin seeks out the point (of disease) in which the receptors are accumulated. (*a*) The *ophthalmo-reaction* of Wolff-Eisner and Calmette (1907) consists of dropping in the eye one drop of 1 per cent. old tuberculin which is followed in 12 to 24 hours by a reactive conjunctival hyperemia. In 6300 tests Calmette found a positive reaction in 90 per cent. of tuberculous patients, in 57 per cent. of suspected tuberculosis and in 17 per cent. of apparently healthy persons; the only complications were keratitis in three instances, conjunctivitis in 20 and a reaction lasting over three weeks in 72 instances. Ulcers may develop if tuberculin is instilled into diseased eyes. The reaction may develop in typhoid, and fails in advanced tuberculosis, whence the poor outlook of an absent reaction in manifest phthisis. (*b*) *Von Pirquet's test* is vaccination of the skin under a drop of 25 per cent. old tuberculin (in salt solution); in 24 hours a small papule appears. This method is absolutely safe, mostly adapted to children (up to the fourth year), but too delicate to be valuable in adults unless it is negative. It is positive in 50 per cent. of apparently healthy adults. It is absent in children in advanced and cachectic tuberculosis, whence its poor prognostic significance, and in many intercurrent, acute infections, as measles, scarlatina, typhoid, etc. (due to insufficient antibodies to react to the tuberculin or to antianaphylaxis). The test may react in 24 hours, resulting in a large papule or dense infiltration; again the reaction may be prolonged, chiefly in adults, developing a papule on the second or third day and lasting a long time. In *Moro's test* equal parts of lanolin and old tuberculin are rubbed into the skin. (*c*) *The intracutaneous test* (Mantoux and Roux). They inject, by a fine very sharp needle, 0.01 mgm. tuberculin, in a dilution of 1 to 5000, not beneath but into the skin itself. A positive reaction consists in the appearance, after a few hours, of a small papule, which on the third day attains its maximum. It is more sensitive than the cutaneous test. (*d*) *Hypodermic injection* of Koch's *tuberculin* produces (i) a local exudative reaction, and after four to ten hours, a (ii) systemic reaction, characterized by chill, fever of even 5 degrees, headache, general pains, malaise, nausea, sometimes erythema nodosum, and (iii) an increase of sputum, rales, or even a slight area of pulmonary dulness; the first injection of Koch's older tuberculin should be small, in adults 0.2, 0.5, 1 or 5 milligrams—in children 0.1 to 1 milligram. It is held that in a positive reaction with less than 5 mgm., the focus is active; if with 5 to 10 mgm., it is inactive. The reaction is specific and indicates that there is a tuberculous focus in the body, but not necessarily in the lungs, unless rales develop at the suspected spot after the injection. Contra-indications to its use are, fever, hemoptysis, weak heart, nephritis or a certain diagnosis. In veterinary medicine it is more valuable, leading to a correct diagnosis in 90 or 97 per cent. The author admits a reluctance to use tuberculin; four cases of miliary tuberculosis apparently resulted from its employment.

Sticker recommended the use of *potassium iodide* in small doses (gr. iij); it congests the lungs at the seat of the lesion, possibly makes the auscultation findings clearer, and carries bacilli into the sputum; its use is dangerous.

Pseudotuberculosis.—The Rhizomucor parasiticus, the ray fungus, streptothrix, blastomycetes, yeasts, the aspergillus and Pfeiffer's pseudotuberculosis bacillus, which is not acid-fast, may cause pseudotuberculosis. The form caused by the Aspergillus fumigatus is described as a primary lung affection, but many writers consider it secondary only. The symptoms of *streptothricosis* are hemoptysis (23 per cent.), fatigue, emaciation, anorexia, dry cough and, later, a greenish, purulent sputum. The physical findings are bronchitis, less often slight consolidation or cirrhosis of the lung. Ashton and Wright (1905) collated 26 cases, most of which resembled phthisis; over 50 per cent. were unmixed infections and 76 per cent. died. Pulmonary thrush is rare, Garin finding only 6 recorded cases (1911); the symptoms resemble tuberculosis and the Endomyces albicans is found in the sputum and lung.

Prognosis of Pulmonary Tuberculosis.—Two issues are possible:

1. Recovery.—Autopsies on hospital cases show 25 to 97 per cent. of healed tuberculosis. Many recoveries are only relative, since Kurlow's inoculations from seemingly latent caseous foci into guinea-pigs were positive in 77 per cent.; such foci may be aroused by intercurrent disease, though some writers regard recurrence as fresh infection. Recovery is effected by *agglutinins* (precipitins) and other specific antibodies (*opsonins*) which aid in phagocytosis. The result is encapsulation, cicatrization and calcification. Liebermeister believes recovery is possible in 50 per cent. of incipient cases. The outlook depends on several factors: (*a*) The *hereditary vital endowment* of the patient; (*b*) his *environment, social situation*, habits and intelligence; (*c*) whether the localization is single or multiple; (*d*) secondary or mixed infection; (*e*) digestion; (*f*) degree of toxemia (fever and emaciation); (*g*) youth, diabetes, pregnancy and lactation are unfavorable; (*h*) 80 per cent. with normal pulse-rate were alive after four years and 97 per cent. with rapid pulse were dead (Schneider); and (*i*) complications (*v. i.*).

2. Death.—The clinical course is variable, even in chronic cases; it averages two years (Bayle, Laennec, Andral), over two and a half years (Pollock), three years (Cornet) and seven years (Williams and Dettweiler, though not including acute or infantile forms); the course may last 30 to 55 years. Death is usually peaceful and painless, though at times it comes with dyspnea, delirium or convulsions. Mechanism of death: (*a*) Heart failure is the common cause, associated with edema, asthenia, anemia and marasmus. Other causes are less frequent, as (*b*) profuse hemoptoë; (*c*) pneumothorax; (*d*) pulmonary embolism (clots or gas); (*e*) uremia; (*f*) meningitis; (*g*) perforative peritonitis; and (*h*) sudden death without adequate pathological findings, in which Arnold, Brissaud and Toupet found in the liver large numbers of bacilli, which probably caused an acute intoxication (the injection even of dead bacilli causes rapid marasmus and death).

Complications.—Tuberculosis in other organs will be considered below under separate headings.

1. Respiratory.—(*a*) Tracheitis and *bronchitis* occur in almost every case. (*b*) *Dysphagia* usually results only from local involvement of the pharynx or larynx; swallowing into the larynx is the result of paralysis

of the epiglottic depressors. *Hoarseness*, dysphonia and aphonia suggest *laryngeal tuberculosis*, but may develop from syphilis, toxemia or catarrh; from paralysis of the recurrent laryngeal nerve from pressure by lymph glands; from inflammation, on the right side by pleural scars, and on the left by large pleural effusions; or from thickening at the hilum. (*c*) Pulmonary *gangrene* may develop in the cavity wall. Lobar pneumonia is not infrequently a cause of death. (*d*) Dry *pleurisy* is an almost constant complication, and is a conservative process, militating against pneumothorax. Exudative pleurisy more often develops previously to lung phthisis than during its course. In the latter instance it is usually serofibrinous, and not extensive. Effusions, by compression, sometimes seem to exert a favorable effect on the lung lesion. Litten thought that their sudden absorption might occasion miliary tuberculosis. Chyliform or adipose effusions may occur (see CHYLOTHORAX AND CHYLOUS ASCITES); purulent forms result from mixed infection, cavities or pneumothorax. (*e*) *Pneumothorax* is observed in 5 per cent. of cases, and results from rupture of superficial rapidly forming cavities without a limiting membrane. Perforation occurs most frequently between the second and fourth interspaces, and between the mammary and axillary lines. Its onset is spontaneous, or results from straining or coughing, whereon sudden symptoms develop, as great dyspnea, orthopnea and cyanosis, and sudden physical signs, as dislocation of the heart, spleen or liver, sudden ectasia, unilateral thoracic immobility, weak vocal fremitus, voice sounds and breathing, a tympanitic note, and succussion when serum or pus forms. The onset is occasionally gradual or even symptomless. Death may result in a few hours, but life may be prolonged weeks or months. Infrequently pneumothorax (*q. v.*) may exert a beneficial effect on the lung trouble.

2. CIRCULATORY.—(*a*) The typical *rapid pulse first* appears only after meals or exertion, but later becomes permanent. It is *chiefly toxemic*, but may be due to compression or inflammation of the vagus from lymphadenitis, hyperthyroidism, mediastinitis, pleurisy or pericarditis. There is a constant and early *lowering* of the *blood tension*, and the vasomotor instability causes the bright red cheeks. (*b*) The *heart* is pale, fatty, flabby and weak, and has been described as atrophied since the time of Laennec, but Potain considers that the small heart is merely accommodative to the malnutrition and anemia. Cardiac hypertrophy and dilatation are due to renal disease, pericardial or pleural adhesions, or pulmonary induration; the second pulmonic tone is accentuated in one-third of the cases. Adhesive or effusive *pericarditis* (in 3 per cent.) may result by extension from pleurisy or caseated lymph glands, or pyogenic infection. Endocarditis tuberculosa is infrequent (1 per cent.); tubercle bacilli may be deposited in cardiac clots, or endocarditic excrescences of non-tuberculous origin; this endocarditis is due to miliary deposits in the intima, and clinically escapes recognition in most cases. Woolley reported 11 cases of tuberculosis of the aorta (1911). (*c*) A functional, systolic murmur is very common. (*d*) *Thrombophlebitis* occurs in less than 1 per cent. of cases.

3. Nervous and Muscular.—(*a*) The mental power may be normal; oftener there is a *slight mental weakness*, irritability, self-concentration, triviality and *lack of endurance*. Consumptives are usually *optimistic*, even on the day of their death (*spes phthisicorum*); occasionally melancholia or mania develops; anemia, toxemia, marasmus and edema of the brain may cause terminal aberration. (*b*) *Neuralgia* and hyperesthesia are not uncommon. Toxemic multiple *neuritis* develops in 1 per cent.; it may cause pain, sensory disturbance or amyotrophy. (*c*) The toxins which reduce the body weight one-half or more, result in marked *muscular degeneration* and atrophy, which cause the flat chest, and possibly the cardiac wasting. Wasting is expressed in terms synonymous with tuberculosis—consumption, *phthisis*, tabes. The muscles are dry, pale, fatty; they show transverse rents, obscure markings, granular degeneration or hyperplasia of the connective tissue, or myositis. *Their wasting and the nervous toxemia explain the asthenia* and incapacitation for work, a matter of national economical importance. The muscles are often irritable, show fibrillary contractions and when tapped or pinched show local lumpy contraction, known as *myoidema*.

4. Digestive.—(*a*) The tongue may be furred, or red and clean. Aphthæ are frequently distressing. Excessive redness of the gums is frequent. (*b*) *Stomach* disorder is present in 50 per cent. of cases; it may be the initial symptom of toxemia, or appear only late in the process. *Anorexia* varies in degree and occurrence, being most marked when the fever is high, and most frequent in those with previous dyspepsia or poor appetite; again it may be due to glands compressing the vagus (Peter, de Mussy). Cornet instances an advanced patient who complained that he could only eat four beefsteaks for dinner. "The patient, with fever, who eats and digests well, is tuberculous" (Lesègue). Brieger found the chemism normal in 50 per cent. in the incipient stage, in 33 per cent. in moderately severe, and in but 16 per cent. in severe advanced cases. After eating there may be a sense of fulness or pain. Nausea, gagging and *vomiting* may result from pharyngitis, forced feeding or coughing (Morton's "emetic cough"). In advanced cases, nausea and vomiting occur from overeating, *toxemic gastric catarrh*, swallowing of sputum, etc.; anatomically, the stomach shows catarrh, fatty degeneration of the glands, interstitial hyperplasia and amyloid infiltration. (*c*) *Intestines:* Diarrhea is usually late; it may be caused by toxemia, swallowed sputum, intestinal catarrh, toxemic obliteration of the lymph and blood-vessels, ulcers or amyloid degeneration of the gut. Tuberculous *ulcers* in the small gut, cecum and ascending colon cause diarrhea in but half the cases and probably only when combined with catarrh or amyloid degeneration; ulcers in the lower colon and rectum almost constantly induce diarrhea, which may therefore occur from causes other than ulceration, and ulcers may exist without it. Blood, pus, hematin crystals and tubercle bacilli are not always present. Tubercle bacilli may pass through with the sputum swallowed, without intestinal tuberculosis. Anal fistula occurs in 3 per cent. of cases. (*d*) The liver is fatty (in 10 per cent.); Jonescu (1902) collected 33 cases of cirrhosis cardiotuberculosa—induration from stasis, with tuberculous granulations and fatty

degeneration. (*e*) Tuberculous *peritonitis* (*v. i.*) occurs in 10 per cent. of pulmonary tuberculosis.

5. Amyloid Degeneration.—Of all amyloid degeneration 80 per cent. is due to tuberculosis, and 69 per cent. to ulcerative phthisis. It is found in 8.8 per cent. of tuberculous autopsies. The spleen is affected in 93 per cent., the kidney in 77, the liver in 62, the intestines in 28 and the stomach in 12 per cent. of cases.

6. Genito-urinary.—*Urinary.*—The loss of weight causes *phosphaturia* and increase of chlorides (0.5 to 1 or even 12 gm.). More protein is excreted than is ingested. *Albuminuria* indicates nephritis, amyloidosis or local tuberculous disease. Acute and chronic parenchymatous nephritis is not infrequent, due to long-standing toxemia. *Amyloid* disease of the kidney produces albuminuria in but 50 per cent. of the cases; nephritis is also present in 75 per cent., and the chronic parenchymatous type in 66 per cent. Flick and Walsh found tubercle bacilli in 84 per cent. by inoculating guinea-pigs with the urine; tubercles were found in 63 per cent. Ehrlich's *diazo reaction* is very frequent and usually of unfavorable import.

Sexual.—The sexual desire may be more intense, though its increase is exaggerated. The *menses often disappear early*, and conception is less apt to occur. Pregnancy often temporarily betters the condition, but a more rapid course is usual after delivery (*v.* page 190). The mammary enlargement described by Bedor, particularly in males, is rather a chronic interstitial mastitis than an actual hypertrophy. Bacilli have been found in the milk of tuberculous mothers.

7. Skin.—*Pityriasis versicolor* occurs very frequently. Piery claims that bacilli exist in the sweat. Cyanosis, chloasma, desquamation, coarseness of the hair, herpes, lichen and purpura are sometimes observed. *Clubbing of the fingers* ("Hippocratic fingers") and toes develops chiefly in chronic cases, is largely due to hypertrophy of the soft tissues and is not pathognomonic, for it occurs also in congenital cardiac disease, aneurysm, bronchiectasis, emphysema, etc. (See Acromegaly.) Emphysema of the skin is very uncommon; it follows interstitial emphysema of the lungs (due to rupture of the alveoli), pneumothorax or cavities. Edema results from anemia, cardiac weakness, phlebitis, nephritis or neuritis.

Treatment of pulmonary tuberculosis is considered later, after description of other localizations of the tubercle bacillus.

IV.—Tuberculosis of the Lymph Glands.—Of glandular adenopathies 68 per cent. are found in the first ten years of life. It occurs fairly often in adolescence and sometimes even in the aged. It is found anatomically in 85 per cent. of tuberculous children, and in 25 per cent. of all autopsies on children. *Scrofula* is glandular tuberculosis, but it presents different characteristics from those of pulmonary infection (decreased virulence of the bacilli in scrofulous glands, infection with the bovine bacillus, or infection with very few bacilli). The skin, mucous membranes and lymph vessels are physiologically more permeable in the very young, and an increase or persistence of this condition is regarded as an embryoism, *i. e.*, a structural tendency toward infection. The

accessory causes are the same as in pulmonary tuberculosis: (*a*) An hereditary tendency; (*b*) poor hygiene, dark damp dwellings and inadequate nutrition; (*c*) infections, as pertussis and measles, to a less extent scarlatina, diphtheria, variola and syphilis.

General Characteristics.—(*a*) It usually runs a chronic course. (*b*) Recurrence is frequent. (*c*) Multiple lesions are usual. (*d*) Glandular involvement is frequently associated with affections of the skin (as eczema, scrofuloderma or lupus), inflammations of the mucous membranes (as conjunctivitis, otitis, rhinitis or bronchitis), and disease of the bones or joints (as coxitis or Pott's disease). Escherich refers the hypersensitiveness of the skin and mucosæ to the presence of the antibody—tuberculous "allergy." This association is frequently known as scrofula.

General Anatomy.—(*a*) In infection with very few bacilli regression is possible. (*b*) If the infection is sharper, conglomerate tubercles form, with the following issues: death of the bacillus in the central caseation, softening and external rupture, sometimes calcification or extension locally, generally by the blood current. (*c*) Hyperplasia without any or with very late caseation results from infection with few bacilli. The lymphadenoid tissue is compressed by epithelioid spindle-shaped cells; the glands may reach the size of an egg, and their cut section may show no gross evidence of tubercle. (*d*) Mixed infection causes rapid caseation, necrosis, softening and early rupture; their cut section appears "like that of a raw potato but not so moist"; the inflammation often causes a periadenitis, whereby the glands fuse, a phlegmon results, and generalization of the process throughout the body ensues.

1. Cervical Tuberculous Adenitis.—Infection occurs through the tonsils, nose, etc.; the bacillus may pass through the mucosæ without producing any lesion in them. The cervical glands constitute 96 per cent. of Wohlgemuth's series. In cervical lymphadenitis, the glands at the angle of the jaw and beneath or anterior to the sternomastoid muscle are most often involved; in bilateral involvement the glands of one side are usually larger. The glandular swelling is slow in onset, intermittent in progression and usually unattended by pain or other symptoms. In some cases the first symptoms are acute, but the trouble subsides in a few weeks. The nodes first are discrete and freely movable; they may blend into large nodular masses, and periadenitis may fuse the glands and cause their adherence to the skin, which becomes edematous from lymph stasis, dough-like and reddened. Softening and fluctuation are then frequent, with burrowing of pus and external rupture through small fistulæ. The fistulæ widen, discharge cheesy and milky material and form intractable ulcers covered with granulation tissue. Cicatrization leaves deep, deforming, depressed scars. Extension may occur to the deeper or subpectoral or axillary glands. Fever, anemia and other toxemic manifestations may develop as in lung tuberculosis, or the adenopathy may be the sole clinical fact.

2. Bronchial Glands.—In autopsies on tuberculous children these were affected in 100 per cent. of Northrup's series, and in adults in 60 per cent. In children tuberculosis of the bronchial glands is primary and tuberculosis of the lung secondary. In adults lung involvement is

usually primary and bronchial adenitis secondary. Biedert, in 1346 autopsies on tuberculous children, found the lungs diseased in 80 per cent., the bronchial glands in 78, the intestines in 32 and the mesenteric glands alone in 40 per cent.

Symptoms.—In most cases no symptoms develop; in others pulmonary lesions overshadow the bronchial involvement; in others miliary tuberculosis develops, promoted by rupture of the bronchial glands into the pulmonary vessels; in still others mediastinal symptoms result; in very few cases are there actual physical findings.

Pressure Symptoms.—Pressure on the vagus, known to Wrisberg, causes the rapid heart, so frequently remarked in tuberculous children; paroxysmal dyspnea, or spasmodic cough which sometimes resembles whooping-cough (*v.* page 109); lessening of the sensitivity of the bronchi, which favors infection; and finally cardiac dilatation. Pressure on the *recurrent laryngeal* nerve is marked by hoarseness, aphonia, dyspnea or laryngospasm. The *air passages* may be compressed, as evidenced by dyspnea, holding the head backward and impaired expansion of one or both sides of the chest (Grancher's sign). The pupils may be unequal from pressure on the sympathetic nerve. Other pressure symptoms are: hypertrophy of the heart, or the paradoxical pulse, from pressure on the aorta; edema and cyanosis of the face and arms, from compression of the superior vena cava; cough, rales or hemoptysis, from stenosis of the pulmonary veins; a murmur over the innominate vein on throwing the head backward; and dysphagia, from esophageal compression.

Signs.—Bronchial breathing is occasionally heard between the second and fourth dorsal vertebræ, more clearly behind than in front; more on the right than on the left side (since more glands are clustered around the right bronchus), and more markedly than the high-pitched breathing heard physiologically over this region. Propagation of the whispered voice is D'Espine's sign. If the bronchus is compressed there is neither bronchial breathing nor increased vocal fremitus. An irregularly outlined dulness is elicited on exceptional occasions either between the clavicle and third rib, on either side of the sternum, or behind, on percussing the third and fifth dorsal vertebræ (de la Camp's sign). Adjacent interscapular tenderness constitutes the spinalgia of Petrusky. A tympanitic note in the upper lobes denotes relaxation of the lung. The symptoms and signs are extremely variable, appearing suddenly and remitting unaccountably.

Issues.—(*a*) Tuberculosis of the lung; (*b*) miliary tuberculosis; (*c*) tuberculous pleurisy or pericarditis; (*d*) rupture into the air passages, causing sudden suffocation; (*e*) fatal hemorrhage into the air tubes, which is less frequent; (*f*) rupture into the esophagus, or traction upon it causing an esophageal diverticulum.

Diagnosis.—The direct diagnosis rests upon tuberculous adenopathies elsewhere, pressure symptoms, physical signs and *x*-ray plates. As Henoch puts it, "A diagnosis can rarely be made (from the physical signs), but if made, is nearly always correct."

3. Mesenteric Glands.—The bacilli pass the intact intestinal mucosa and settle in the mesenteric glands. Carrière found that but 1

per cent. of cases were primary; of the secondary cases, the primary focus was in the peritoneum in 40 per cent., in the lungs in 30, in the intestines in 20, in other glands and bones in 5 per cent. each. Hess holds that in over 60 per cent. of the primary infantile cases the infection is by bovine bacilli; in adults it is by the human type (*v.* page 143). The glands may be palpable as thick, nodular masses through a thin abdominal wall, but are often obscured by the *abdominal distention* which contrasts strikingly with the *general emaciation* (*tabes mesaraica*). Digestive disturbance is frequent, and there is usually *a diarrhea* with thin, stinking stools. Compression is less frequent in the abdomen than in the less yielding thorax, though the veins of the abdomen may stand out and edema of the legs may appear. The glands sometimes heal by induration and calcification, or, by softening, rupture into the intestine or peritoneum.

4. Generalized Lymphadenitis may closely resemble Hodgkin's disease (Paltauf and Sternberg). This form is common in the negro. In two personal instances the clinical and gross pathological findings were typically pseudoleukemic, and only the epithelioid hyperplasia and the Bacillus tuberculosis stamped them as tuberculous.

The *treatment* of tuberculous lymphadenitis is that of tuberculosis in general (*v. i.*). Hyperplastic tonsils and adenoids require removal, being the atrium for cervical infections. In 175 cases of cervical adenitis treated surgically, 58 per cent. were cured after three years, 8 per cent. had recurrence, 11 per cent. developed pulmonary tuberculosis and 23 per cent. died. In another series of 700 cases treated medically, 28 per cent. developed tuberculosis in some other organ (Demme). The writer relies largely upon full feeding, *x*-rays, ample sunlight and fresh air; if possible, a warm sea-side climate, and operation if finally necessary, after puberty. The syrup of the iodide of iron and cod-liver oil are very largely used. Inunctions of green soap are recommended, ℥ss rubbed into the back three times a week. Some 62 instances of tuberculous mesenteric adenopathy have been operated upon (Floderus).

V. Tuberculosis of the Serous Membranes.—These membranes may be involved separately or simultaneously; multiple serositis or polyorrhomenitis may be acute, subchronic or chronic, serous, fibrinous or purulent, solitary or with visceral tuberculosis.

1. **Pleura.**—Tuberculous pleurisy has been touched upon as a complication of phthisis, and is separately considered under Pleurisy (*q. v.*). It may appear as an apparently primary infection, as secondary to unmistakable phthisis, or as a terminal infection. Miliary tubercles in the pleura may occur without pleurisy. Pleurisy results from direct bacillary invasion, or possibly from simple toxic action. In some cases the pleura proliferates and forms a massive, adhesive, obliterative membrane. Sometimes the interstitial tissue of the lung is invaded (pleurogenous cirrhosis).

2. **Pericardium.**—Accidental miliary deposits are distinguished from tuberculous pericarditis wherein inflammation predominates. It is less common than pleural and peritoneal involvement (in 3 per cent. of phthisis autopsies). Tubercles may be detected beneath the fibrin

deposits or adhesions. It may be symptomatically latent; it may form an unessential part of a miliary tuberculosis; or it presents the ordinary symptoms of pericardial effusion or concretion (*q. v.*). Only eight primary cases are recorded (Scagliosi, 1904).

3. **Peritoneum.**—Tuberculosis of the peritoneum became a clinical entity when Louis, in 1825, declared that chronic peritonitis was tuberculous. It complicates 10 per cent. of phthisis, is found in 2 per cent. of autopsies and constitutes one-quarter of all peritonitides.

Age.—It is almost equally frequent in the second, third, fourth and fifth decades (20 per cent. in each); 66 per cent. of cases occur between ten and forty years of age.

Sex.—Ninety per cent. occur in women. More cases are found in autopsies on males, though more women are operated on. In America it is common among the negroes. It is very seldom primary (0.7 per cent.), but is *secondary to* (*a*) *lung, bronchial gland* or *pleural tuberculosis* (in 80 per cent.); (*b*) intestinal ulcers or mesenteric and retroperitoneal adenitis, especially in children (tubercle bacilli may pass the intestinal wall without localizing in it); (*c*) tuberculosis of the Fallopian tubes (which in turn, is secondary to bronchial adenopathy). *Tuberculous peritonitis is hematogenous.*

It frequently complicates liver cirrhosis (though it is strange that tuberculosis should develop in any stasis), ovarian tumors or trauma, which disseminates infection from some quiescent focus. Sixty-two cases are reported in hernial sacs.

Types.—A clinical description is difficult, for the disease may be acute or chronic, latent or stormy, circumscribed or diffuse; it may be marked by adhesions, free or encapsulated ascites or by large tumor-like masses; it may be obscured by coincident miliary tuberculosis, liver cirrhosis, exudative pleurisy or ulcerative phthisis, or may appear as a seemingly primary clinical fact. The unessential miliary tubercle, and the small, non-inflammatory tubercles over intestinal ulcers are not considered. Certain types may be described:

1. The *acute form* begins with severe symptoms, fever, rapid pulse, vomiting, abdominal tenderness and pain, free peritoneal fluid, which shifts with change of position, and meteorism from paresis of the gut. This type may resemble *typhoid* when the intoxication is more marked than the local signs. The *peritonitic* local signs may be sharply marked, but always less so than in suppurative peritonitis. Certain cases simulate appendicitis, ileus from tuberculous adhesions, or incarceration beneath bands; or in three cases precipitated by delivery, puerperal infection. Indicanuria, so frequent in other acute peritonitides, is usually absent. The ascitic fluid is usually lemon-colored, serous or slightly flocculent or hemorrhagic, and of a specific gravity of over 1.014; the leukocytes are usually mononuclear. In rare instances the acute *peritonitis is suppurative;* 26 cases are on record due to perforation of a tuberculous ulcer in the gut. In some cases the acute type is a *terminal infection.* Though commonly diffuse, there is sometimes acute local pain or circumscribed effusion. Plastic adhesions occasionally modify the freedom with which this ascitic form shifts with alteration of posture. Acute

tuberculous peritonitis comes more often under the care of the physician than of the gynecologist or surgeon.

2. *Subacute or chronic forms* include: (*a*) The *fibrinoplastic form*, causing adhesions, may result from the acute form or may develop insidiously; symptoms and signs are usually lacking, or not distinctive. (*b*) The *fibrinocaseous form* is characterized by large solid masses, by fusion of tubercles. Between the recesses formed by the adhesions, pockets of exudate frequently occur, containing blood-stained serum, cold abscesses or cheesy material; the intestine is thickened and contracted, the mesentery deformed and the omentum shrunken. (*c*) The *effusive form* is marked by serous, serofibrinous, serohemorrhagic and less often by puriform or adipose exudate.

These subtypes vary so much that the individual symptoms will be considered separately:

Individual Symptoms.—1. Adhesions and Retraction.—The infiltrated *omentum* gradually *indurates and retracts* above the navel as a fibrocaseous tumor-like mass; it may often be palpated, even when there is much exudate, but becomes clearer after paracentesis; it has been mistaken for gastric carcinoma, enlarged liver or distended gall-bladder, especially in chronic forms with little fever; it is more suggestive of tuberculous than carcinomatous peritonitis. The *walls of the gut* are frequently so *thickened* that intestinal stenosis results; the gut may measure but one-half or one-third of its original length; it may be thickened, adherent or tumor-like. The entire peritoneal sac may be distorted (*peritonitis deformans*) and intestinal stenosis ensues. The mesenteric retraction (*mesenteritis retrahens*) modifies the physical findings, tympany and dulness; the mesentery may measure one inch in thickness.

2. Ascites.—The abdomen is moderately enlarged by exudation and meteorism. Large exudates may cause widening of the abdominal veins because the cava is compressed, as in ascites (*q. v.*). The abdomen protrudes but its centre is not usually flattened nor its sides bulging as in the ascites of liver or cardiac disease, but it is often prominent in the median line, as in ovarian cyst; most of Löhlein's cases were sent to him with a diagnosis of ovarian cyst. Without adhesions, the dulness shifts with change of posture, with tympany over the supernatant gut and dulness over the settling fluid. The physical signs differ from common ascites in that the tympany is often on the right side, the *mesenteritis retrahens* pulling the small gut to the right. A tuberculous mass may dislocate the tympanitic intestines to the left, the median line or pelvis. In very rare cases the fluid may rupture into the bowel or externally through the navel. Dulness may indicate adhesions, thickening of the gut, caseous masses or enlarged glands.

3. Meteorism.—Sudden paresis of the gut in the acute type or tuberculous tumors impeding peristalsis cause gaseous accumulations. Meteorism is one of the stigmata of tuberculosis of the mesenteric glands and then is often due to coincident peritonitis; in both conditions, but most often in children, the stools are fatty.

4. Palpation.—A *doughy sensation* on palpation is usually referred to peritoneal adhesions or constricted intestine, but in some instances is due to early infiltration of the preperitoneal tissues.

5. Other Symptoms.—In some cases the *Beatty-Bright friction-rub* can be elicited. *Pelvic effusion* is common in women, who also suffer from painful and disturbed menstruation. *Fever* is frequently irregular, sometimes continuous, remittent or hectic; it is most frequent in acute types or in acute exacerbations of chronic cases; the disease cannot be excluded by the absence of fever. Fever, pain and tenderness distinguish tuberculous encapsulation from simple ascites. *Pain and tenderness* are generally conspicuous, though less than in suppurative peritonitis and by no means constant. Emaciation is frequent, but often patients with tuberculous peritonitis present a good color and robust appearance. *Gastro-intestinal disturbances* are frequent; the usual constipation is occasioned by impaired peristalsis; in some cases diarrhea is caused by toxemia. In rare instances there are icterus, splenic tumor, and other complications due to special localizations of the process.

Diagnosis.—Sometimes there are no just grounds for suspecting the disease. Importance attaches to tuberculous lesions in the lungs, pleuræ, glands or Fallopian tubes. Multiple serositis is extremely suggestive. If another tuberculous lesion cannot be established, tuberculous peritonitis, which constitutes nearly all chronic peritonitides, is very probably present; malignancy must be considered. A positive tuberculin reaction does not prove that the tuberculous lesion is peritoneal. Inoculation may be made with fluid withdrawn by paracentesis. (See Pleurisy.) Exploratory laparotomy is justifiable, as simple abdominal section is the foremost therapeutic measure.

Course.—The onset is usually insidious and the course, independently of therapeutic measures, shows spontaneous remissions.

Treatment.—*Rest in bed,* good hygiene and *full feeding* very often arrest the process (in 25 per cent. of cases). The tumors, exudate and adhesions may recede and no symptom reappear for months or years. Spontaneous recovery, first described by Bamberger was ignored until recent times. In 1864 Spencer Wells observed that healing may follow laparotomy. Experiment has shown that the tubercles heal by degeneration, vascularization and development of connective tissue.

Operation gives various results, recovery occurring in 75 per cent. of 1300 cases. Operation in the first four months is contra-indicated, as the early exudate has strong bactericidal properties. Simple paracentesis is inferior to laparotomy. Lavage is superfluous. It is thought that the access of air or sunlight or manipulation is the potent curative factor in operative treatment; the withdrawal of fluid allows the access of fresh serum with increased antitoxic properties. Operation, in the author's opinion, is clearly of great value; the free fluid is evacuated, detritus or pus removed and the (local) cause, as pyosalpinx, is excised, the percentage of recoveries rising to 71 to 92 per cent. Some writers believe that the tubercles which heal after operation were already in process of healing spontaneously. (See Pleurisy for autoserotherapy.)

VI. Tuberculosis of the Brain and Meninges.—Involvement may result from (*a*) the miliary tubercle (*v. s.*), (*b*) the solitary or conglomerate tubercle, which produces symptoms identical with those of brain tumor (*q. v.*), (*c*) the tuberculous abscess and (*d*) meningitis (in 4.7 per cent. of adults dying with pulmonary phthisis).

Primary cerebral tuberculosis is extremely rare, and in reported cases it is probable that microscopic foci elsewhere escaped detection.

VII. Tuberculosis of the Eye.—Only 50 cases occurred in 141,000 eye patients. The number of reported cases of conjunctival tuberculosis is 150, of which but 100 were proved tuberculous.

VIII. Tuberculosis of the Alimentary Tract.—1. **Lips.**—The lips are very seldom affected; lupus may invade them. Tuberculous ulcers are very painful and may be confused with cancer or chancre.

2. **Gums.**—The gums are seldom involved, though tuberculosis has followed caries and extraction of the teeth.

3. **Tongue.**—This is involved in 0.6 per cent. of cases. Tuberculous tumors sometimes break down into cold abscesses. Tuberculous ulcers develop on the dorsum or edges; they have rough caseous bases with uneven or undermined edges, and are almost never primary; syphilitic ulcers are differentiated by the therapy and Wassermann; cancer by adenopathy; and both, by the absence of bacilli and the results of inoculation.

4. **Pharynx.**—Primary disease is most infrequent, and involvement, secondary to pulmonary or laryngeal phthisis, occurs in 0.3 per cent. only. The process begins in the lymphoid structures, which are infected by tuberculous sputum or food, or it extends by the lymph vessels from the larynx. Three-fifths of the cases are associated with tuberculous intestines, and one-seventh with oral tuberculosis. The cervical glands may be affected in acute, but rarely in chronic, cases. The ulcerations are usually superficial; when propagated from the larynx the most distressing dysphagia results. *Retropharyngeal abscess* usually results from suppurative lymphadenitis and osseous disease.

5. **Palate.**—Ulceration occurs in 1 per cent. of phthisis cases; in coughing, bacilli come in forcible contact with the palate. Lubinski observed three instances among 16,000 throat cases.

6. **Tonsils.**—The tonsils are diseased in 4 per cent. of phthisis cases; the process may stop at the tonsils, for they act as barriers to infection, or tuberculous cervical adenitis may develop. Their infection may be primary, and Dieulafoy holds that tonsillar infection may be followed by a descending tuberculous adenitis, which in turn infects the lungs. The lesions are more often miliary than caseous.

7. **Salivary Glands.**—Of parotid disease 14 records exist.

8. **Esophagus.**—No clear primary case is recorded, and Cone could collect only 48 secondary cases. Infection by contiguity, from the peribronchial glands, may occasion esophageal perforation, hemorrhage, diverticula or stenosis. Extension from the larynx or pharynx along the lymph tracts or from the spine or lung cavities is possible.

9. **Stomach.**—There are only 4 primary cases on record. Secondary disease is estimated at 0.4 per cent. Garrè collected about 100 cases of

tuberculous ulcer, of which 25 were operated upon for pyloric stenosis. The HCl is inimical to gastric tubercle. Tuberculous ulcer of the stomach occurs from the sputum, invasion by contiguity or in few cases from hematogenous infection.

10. **Intestinal Tuberculosis.**—It was first observed by Bayle (1810). (*a*) It is usually *secondary* to ulcerative lung tuberculosis from infective sputum; its frequency is 30 to 50 per cent. Tubercle bacilli may be swallowed without causing intestinal tuberculosis. Of all intestinal ulcerations, tuberculous are the most frequent. Infection from the peritoneum, mesenteric glands or hematogenous infection is barely possible. (*b*) *Primary* intestinal tuberculosis is much more rare, occurring in only 0.1 per cent. of adult cases. The intestinal mucosa in children often allows of the passage of tubercle bacilli to the intestinal lymph glands without disease of the intestine itself. Koch denies infection by milk, and Behring affirms that it is the chief cause of tuberculosis.

The *ulcers* are most frequent in (i) the *ileocecal* region and next in the colon. (ii) They always originate in the *lymphadenoid follicles* or Peyer's patches. Small tubercles fuse into large ones, which caseate and ulcerate; in exceptional cases the caseous nodes do not rupture but cause the peritoneum and mucosa to bulge out. (iii) The ulcers are *irregular* in shape and (iv) their disposition is *ring-like* around the gut (extension by the bead-like lymphangitis tuberculosa); rarely they lie in the long axis of the gut and are ovoid, incompletely corresponding to Peyer's patches. (v) They are single or multiple, and sometimes cover most of the large and small intestines. (vi) Their bases are rough or caseous and their edges infiltrated, which also invades the contiguous mucosa and submucosa. (vii) Localized adhesive peritonitis is a common issue when the process reaches the serosa; perforation occurs in 5 per cent. of cases, but is resisted by the muscular coat and by adhesions; it results in pericecal abscess and suppurative peritonitis. *Stenosis of the intestine* may also result, single, or less often multiple (129 cases collected by Arbuson, 1904). Eisenhardt saw 10 complete and 26 incomplete recoveries in 567 tuberculous ulcerations. Hepatic suppuration or steatosis may result.

Symptoms.—The symptoms usually commence with catarrhal enteritis, pain and fever; diarrhea occurs in but half the cases of ulceration. They may resemble typhoid. The *only pathognomonic finding is the tubercle bacillus*, but this finding may result also from swallowed sputum. Rosenblatt's method of detecting the *bacilli* in the feces is to give laudanum until the stools become hard; in the mucus on their surface, the bacilli are usually found on the first examination, for the hard feces apparently scrape the bacilli from the ulcers. *Pus* is often found. Girode called attention to the black stools, which are ominous, as they indicate malnutrition; the picture of dysentery is rarely observed; fatal hemorrhage is infrequent. An *appendicitic form* may be noted, with recurrence and fistulæ. In *typhlitis tuberculosa* there is great thickening in the cecal region, with a hard, slightly movable or totally adherent, vertical, tender tumor. The onset is usually insidious with recurrent pain, increasing intestinal stenosis and constipation alternating with diarrhea. Hemor-

rhage is infrequent, fever is usually absent and emaciation is sometimes pronounced. Duguet described this condition in 1869, and, since it so fully resembles cancer or sarcoma, it attracted the especial attention of surgeons, Bassini, Bouilly and Billroth. Campiche collected 379 cases, of which 25 per cent. died from the operation; 35 per cent. were well for months or years; and 40 per cent. ultimately died of tuberculosis. The process usually begins in the mucosa (rarely in the serosa) and is marked by ulceration, villosities in the mucosa, great local infiltration and hypertrophy of the bowel and destruction of the ileocecal valve. Patients may die of acute obstruction, or only after a chronic course of a decade or more; recovery by fibrosis is possible.

In the *rectum* ulcers, lupus, tuberculosis cutis and periproctal abscess are sometimes seen. *Fistula in ano* occurs in 3 per cent. of phthisic subjects and rarely in other conditions; the idea that its excision excites phthisis is confusion of cause and effect.

11. **Liver.**—The liver is involved less in adults than in children (38 per cent.), but at all ages it is of pathological interest chiefly. (i) Large tubercles may develop in the liver, sometimes with peritonitis and perihepatitis. (ii) Miliary tubercles (in 80 per cent.) are of no clinical significance. (iii) Hanot maintains that there is a primary cirrhosis of the liver, associated with tubercles, fatty degeneration, tuberculous peritonitis and perihepatitis (*v.* page 167); at the most it is extremely rare. (iv) Tuberculous cholangitis begins in the bile vessels as small nodes; breaking down, they resemble cysts or abscesses.

12. **Spleen.**—The spleen is involved in 45 per cent. of tuberculous children. Franke collected 29 cases of primary tuberculosis; 10 were operated on, with 7 recoveries. (See DISEASES OF SPLEEN.)

IX. Tuberculosis of the Genito-urinary Tract.—Genito-urinary tuberculosis was described by Morgagni and by Bayle, but first fully by Lichtheim, in the cadaver, and by Rosenstein, Babes and Smith, clinically. It is most frequent in the period of greatest sexual activity (twentieth to fortieth year), and 72 per cent. occur in males.

Modes of Infection.—1. HEMATOGENIC INFECTION.—*This is the most frequent variety.* It occurs in 5 per cent. of cases of chronic pulmonary tuberculosis; 76 per cent. of cases are *secondary to some pulmonary or peribronchial glandular focus,* which very often is latent, so that most, seemingly primary, foci are secondary. Jani found tubercle bacilli in sound testes and prostates (an invasion in the death agony).

2. UROGENIC INFECTION.—Contrary to the old doctrine, hematogenous *infection of the kidney descends* to the lower urinary tract, not *vice versa.* Transmission is very rare by infected fingers, instruments, catheters or sexual intercourse.

3. INFECTION BY CONTIGUITY.—The *Fallopian tubes* are diseased in a third to a half of the cases of tuberculous peritonitis. Jani found tubercle bacilli in the tubes in women dying of phthisis. The bladder, seminal vesicles and vasa deferentia are rarely invaded *per contiguitatem* from peritoneal, vertebral or rectal foci.

1. **Renal Tuberculosis.**—The kidney in *miliary* tuberculosis is involved in 90 per cent., but without distinguishing symptoms. The common

clinical form is the caseous-ulcerative. *Caseous nodes* develop in the cortex, infection reaching it from the blood stream (in 90 per cent.); they often break down into cavities, form cold abscesses, undergo secondary pyogenic infection or remain localized in one pole of the kidney. Tubercles also develop in the apices of the pyramids and pelvis of the kidney and form caseous infiltration and ulcers; Caspar, Walker and Israel proved that the process in 41 per cent. of cases descends from the kidney to the bladder. When the process is developed there is a tuberculous pyonephrosis. The entire kidney may become a large sac of caseous material, detritus and lime salts—*dégénérescence massive*. The kidney is often adherent to adjacent structures; its capsule is thickened, its removal strips off particles of renal substance and it is sometimes perforated, causing tuberculous paranephritis. The surface of the kidney may be smooth and normal, lumpy from caseous deposits or softened from diffuse ulceration. In exceptional cases spontaneous healing is possible. Whether one or both kidneys are diseased depends on the stage of the process, and is a most important matter when surgical interference is contemplated. Israel, in his celebrated observations, found one kidney involved in 90 per cent., and Pallet and Albarran in 85 per cent. (Naturally, in autopsy figures, bilateral disease is greater.) Compensatory hypertrophy of the sound kidney is frequent.

Symptoms.—The symptoms are (*a*) urinary, (*b*) local and (*c*) general. These may be absent if the renal pelvis is intact.

(*a*) *Urinary.*—The urinary symptoms are chiefly those of pyelitis (*q. v.*). The urine is acid. Urination is often difficult (dysuria) or frequent (pollakiuria), so that cystitis is at first suspected; it is a safe rule always to *consider the possibility of renal phthisis in every cystitis*, in which the origin is not clear and the treatment unsuccessful, and in every acid, sterile, purulent urine. In 62 per cent. bladder symptoms are the first to appear; they are reflex from the kidney lesion, or are due to complicating cystitis, simple or tuberculous. Guyon describes an early *polyuria* in which the urine is clear, and a later polyuria in which it is turbid. *Hematuria* (*q. v.*) occurs in 66 per cent.; it may be severe or slight and its occurrence is more often early than late; its recurrence distinguishes the "hemorrhagic type"; it may last one month to four years. *Pyuria* occurs when the pelvis is invaded, but is absent if cortical foci do not communicate with the pelvis, or the ureter is occluded; the amount of albumin corresponds to the glistening, shrunken and poorly nucleated pus cells. Epithelial cells, sometimes caseous matter, elastic fibers and necrotic renal tissue are found. The sediment is often a crumbly, rapidly settling detritus; *tubercle bacilli* are found in 80 per cent., but must not be confused with the smegma bacillus (*v.* page 167); the antiformin method is most valuable (page 167), and inoculations settle the diagnosis. Sometimes bacilli in the urine are found in pulmonary tuberculosis without disease of the kidneys. Casts are very infrequent. Ureteral catheterization determines which kidney is involved.

(*b*) *Local.*—*Lumbar pain* sometimes results from capsular tension; it is dull, or colicky, and reflected to the groins and testes, which may be retracted, as in renal colic; colic is due to the passage of caseous particles

or blood clots; differentiation from calculous disease (*q. v.*) may be difficult until tubercle bacilli are found. *Tumor*, in tuberculous pyonephrosis or hydronephrosis, is a small, tender enlargement, seldom of the size attained in calculous or other obstructive disease. The kidney is palpable in one out of five cases. X-ray examination frequently shows caseous foci or calcification.

(*c*) *General.*—General symptoms are hectic fever, malnutrition and other general toxemic symptoms (see page 155), tuberculosis in the lungs and foci in the testes. The heart almost never hypertrophies. In 60 per cent. of the cases operated on, the diagnosis was found correct. The course is chronic and remittent, two, three or even five years being the usual duration; frequently there is prolonged clinical latency; recovery from circumscribed lesions is possible; death results from tuberculous generalization, less often from sepsis, perinephritis or amyloid degeneration, and rarely from nephritis or uremia; in Vienna tuberculous meningitis was present in 17 per cent. as against 6 per cent. in phthisis. The *treatment* is surgical, the first operation being performed by Simon (1871). The operative mortality of nephrectomy is 3 to 5 per cent., and the end result, in Legueu's and Chevassu's 1539 collected cases, was 75 per cent. of recoveries.

2. **Ureter and Bladder.**—The ureter is thickened, infiltrated, caseous, ulcerated and often stenosed; it is involved in 80 per cent. of cases of renal tuberculosis. Secondary involvement from the kidney may be limited to the ureter at its vesical ending. Bladder disease is almost invariably secondary *per contiguitatem* or from hematogenous deposits, as seen by means of the cystoscope. Israel noted tuberculous cystitis in 41 per cent. of cases of renal tuberculosis (*v. s.*), and Vignernon in 50 per cent. Cystitis of long standing always suggests renal tuberculosis, even with gonorrheal or other antecedents. Diagnosis is based on the bacteriology and cystoscope. Rovsing cauterizes with carbolic acid and Caspar irrigates with bichloride. Surgical intervention is indicated.

3. **Male Genitalia.**—The *epididymis* is more often involved than the testis, except in youth. (In early life testicular disease results from generalized tuberculosis; one-half of Jullien's cases occurred under two years of age.) Sometimes there is no caseation, as in hyperplastic lymphadenitis, and even microscopic confusion with sarcoma is possible unless the bacilli are particularly sought. The process is bilateral in 50 to 75 per cent. of cases. Testicular tuberculosis is most often confused with syphilis (*q. v.*), though in the latter there is less pain and fever and more nodular enlargement. The seminal vesicles, vasa deferentia and urethra may be invaded and a rectal examination should never be neglected. Recently the prostate, "the cross road of the urinary and genital tracts," has attracted especial attention; its primary involvement is disputed by some, but the gland is affected in 65 per cent. of genital tuberculosis in the male. The lungs are invaded in primary genital tuberculosis in 70 per cent. and the urinary tract in 56 per cent. *Treatment* is surgical. Carl Rose found but 8 records of tuberculosis of the penis (1911), aside from infection by ritual circumcision of which 41 instances exist (Holt, 1913).

4. **Female Genitalia.**—Local tuberculosis occurs in 2 per cent. of pulmonary tuberculosis. *Tuberculous tubes* are found in 1.5 per cent. of abdominal operations (according to W. Williams, 8 per cent.). Nearly all lesions are due to small foci in the *bronchial glands*. The tubes show nodules, infiltration, caseation and ulceration; bilateral involvement is usual and its true nature may be apparent only under the microscope; tuberculous salpingitis may simulate tuberculous peritonitis; tubal disease may be found in very young children. The uterus ranks next and is involved secondarily. Ovarian phthisis is always secondary. Tuberculosis of the vagina, cervix, vulva and female urethra is most exceptional.

Mammæ.—Schley (1903) collected 65 cases of which 12 were primary; tuberculous lesions often cause nipple retraction, tumor formation or axillary adenopathy (66 per cent.). When cold abscesses, fistulæ or ulcers form, the diagnosis is more definite. A chronic interstitial non-tuberculous mastitis was described by Bedor.

X. Tuberculosis of the Upper Respiratory Tract.—1. **Nose.**—The nose is seldom affected, because its irritation excites sneezing and increased secretion, inimical to the tubercle bacilli. Thrasher (1905) collected 125 cases. In the rare primary form there are very few bacilli and an exuberant non-caseous growth, which may resemble sarcoma; there is little pain and the chief symptom is a chronic coryza. In the secondary form the bacilli are abundant; multiple caseation and ulceration occur as in other localizations; suppressed coughing, with the mouth closed, is thought to force germs into the nose. Nasal infection may occur by picking the nose, and is promoted by wounds, ulcers and various obstructive processes. Infection may spread by the lymph vessels, especially in children, to the cervical glands or even to the meninges. In 33 per cent. lupus affects the nose (Bender).

2. **Nasopharynx.**—Primary involvement is infrequent. Secondary disease, late in phthisis, occurs in 20 per cent.; it is promoted by coughing with the lips closed. The third tonsil may be affected; adenoids are tuberculous in 20 per cent. (Dieulafoy).

3. **Ear.**—Tuberculous *otitis media* occurs chiefly in late phthisis (32 per cent.). It is furthered by measles and scarlatina, is rarely primary, and often causes cervical lymphadenitis, and rarely, mastoiditis.

4. **Larynx.**—Laryngeal phthisis is very rarely primary. Louis, in 1825, asserted that it was secondary to lung disease, resulting from infection by sputum, which view still holds; hematogenous and lymphogenous infection is rare. Laryngeal tuberculosis develops in 25 per cent. of pulmonary tuberculosis in adults, and in 3 per cent. in children. Adjuvant factors are inflammation, fissure and syphilis. In 700 cases Krieg observed unilateral involvement in 39 per cent., of which 92 per cent. occurred on the same side as the lung lesion, not from lymphatic extension but from paresis of the vocal cord due to neuritis of the recurrent laryngeal nerve, and to stagnation of sputum on the paralyzed and anesthetic side. Unilateral disease usually becomes bilateral from contact.

Symptoms.—*In those rare and unfavorable cases* in which the trouble *begins with laryngeal symptoms*, the cough is dry or brassy; it may

develop under the guise of simple laryngitis. *In cases following phthisis,* the bronchitic hack becomes brassy; in the last stages it becomes incomplete, ineffectual or eructative, as the vocal cords cannot be apposed. *Pain and hoarseness* are followed by *dysphonia, aphonia, dyspnea* and distressing *dysphagia.*

FORMS.—(*a*) Most commonly the *ulcers* are found in the interarytenoid region, on the vocal cords, posterior wall of the larynx and arytenoids; they are not deep but are broad; their edges are "nibbled," and their bases pale and sometimes caseous. Particles aspirated into the lungs, cause tuberculous or suppurative foci. In diagnosis, syphilitic ulcers (*q. v.*) are more often single and redder, have more "cut out" edges, frequently show cicatrization, oftener involve the epiglottis and posterior surface of the arytenoids, and less often the vocal cords; show no tubercle bacilli, show no local reaction on tuberculin injection and respond to mercury, iodides and Wassermann test. Ninety per cent. of all laryngeal necrosis is tuberculous. Perichondritis may occasion sudden edema glottidis, urgent dyspnea and early death unless timely tracheotomy is performed. (*b*) In other cases the *submucous tissues are infiltrated* and the mucosa is hardened (pachydermous form); the true cords are less involved than other parts; if ulceration develops it is inconspicuous. (*c*) *Caseous tumors* are not frequent; they may precede the ulcerative form or occur independently of it. (*d*) The *vegetative type* exhibits small excrescences on the ulcers or on the intact mucosa.

TREATMENT.—Pronounced lung changes exist and dysphagia occasions rapid emaciation. Besides general treatment (*v. i.*), local measures, as a rule, do not modify the process. Insufflations of boric acid, iodol and painting with 10 per cent. menthol, 10 per cent. carbolic acid or 20 per cent. lactic acid solution (gradually increased to 80 per cent.), may relieve the local pain; gr. $\frac{1}{4}$ of morphine, hypodermically, and 10 per cent. cocaine, locally, for a time allow the patient to eat but finally fail to anesthetize the wide-spread ulceration. The patient may be able to draw up milk and water by a tube, when the head is turned over the edge of the bed. Caustics are injurious. In Casselberry's practice, 10 recovered.

XI. Tuberculosis of the Heart and Vessels.—(*a*) Pericardium: (*v.* SEROUS MEMBRANES). (*b*) Myocardium: Raviart (1906) collected 185 cases of myocardial tubercle; miliary granulations 41 cases, conglomerate tubercles 83, tuberculous infiltration 12 and tuberculous sclerosis 6 cases. (*c*) Endocardium: Norris found endocarditis in 1.4 per cent. of over 11,000 phthisical necropsies; tuberculous endocarditis is vegetative, very rarely ulcerative or caseous; secondary infection causes some cases complicating tuberculosis. Pellegrini (1912) cites 120 cases of endocardial tuberculosis, of which 53 showed tubercle bacilli. (*d*) Few cases of primary disease of the vessels are reported; 18 cases of tuberculosis of the aorta were collected by Simmitsky (*v.* miliary tuberculosis).

XII. Tuberculosis of the Bones and Joints.—This topic properly belongs to surgery, but its relations demand consideration. Koch first demonstrated the tuberculous nature of caries, fungus joint lesions and tumor albus, and Damsch determined this relation by inoculations.

König and Orth found that 80 per cent. of bone and joint tuberculosis was clearly secondary to pulmonary, glandular, genito-urinary and other tuberculosis. Probably *all cases are secondary* to tuberculosis elsewhere. Infection is *hematogenic* in the majority of cases, far less often lymphogenic. Experimentation shows that *trauma* may determine localization in animals already infected with the Bacillus tuberculosis. Thirty-three per cent. of the cases occur in the first decade of life, and 50 per cent. in the first two decades. Tuberculosis in children involves the bones and joints in 22 per cent. of cases (versus 3 per cent. of adult phthisis). Bovine bacilli cause a certain proportion of tuberculous osteopathies.

Localization.—Watson Cheyne estimates that the knee is affected in 16.5 per cent. of cases, the hip in 14.6, tarsus and ankle in 14.4, elbow in 6.3, skull and face in 5.5, sternum, clavicle and ribs in 5.2, pelvic bones in 3.5, femur, fibula and tibia in 3.5, spine in 2.3, shoulder in 1.5 and scapula, ulna and radius in 1 per cent. Jaffe finds involvement of the spine in 20, foot in 21, hip in 13, knee in 10, hand in 9 and elbow in 4 per cent.

1. **Bones.**—The spongy epiphyses of the long bones are the particular seat of tuberculous osteomyelitis and periostitis, whence infection easily reaches the joints; the diaphyses and flat bones are seldom involved. Involvement of the fingers and toes, less often of the ulna, may cause bulging—the *spina ventosa,* seen oftenest at about five years of age.

In the bone-marrow there develops a grayish-red granuloma which dissolves the bone (*caries sicca*); the trabeculæ necrose and are discharged through fistulæ as sequesters or "bone sand," in a caseous, pus-like fluid. Even large foci may heal by granulating osteitis. Infection may remain semiquiescent until aroused by mixed infection or trauma. *Cold abscesses* are lined by granulation tissue. There may be local tenderness, stiffness, fever and nocturnal pain. Bone lesions are not incompatible with otherwise robust health. Landouzy's camptodactyly is a bending of the fourth and fifth fingers which can be flexed but not extended.

2. **Joints.**—Involvement generally follows contiguous osseous disease. Very often there is synovial inflammation and proliferation; far less frequently there are isolated tubercles in a non-inflamed synovial sac. Serous or serofibrinous synovitis (or the rice bodies, *corpora orzyoidea*) may result from toxins in a neighboring osseous focus, without the actual presence of tubercle bacilli. The *symptoms* of onset may be acute or insidious. Pain, especially at night, contractures and fever may be noted. In the later stages of joint disease granulation tissue forms with large caseous deposits, hydrops, empyema articulorum or the tumor albus (so named because of the thickening of the connective tissue, the edema and glistening appearance of the periarticular structures). The joint is sometimes disorganized. Twenty-five per cent. recover under immobilization, iodoform injections, *x*-rays, Bier's stasis, heat and sea air.

Poncet first called attention to a *tuberculous pseudorheumatism,* which closely resembles rheumatism (*rheumatisme tuberculeuse ankylosante*). Poncet maintains that 5 to 17 per cent. of active, and 20 per cent. of chronic phthisics have pseudorheumatism and 40 to 50 per cent. of

patients with chronic deforming rheumatism are tuberculous. It is affirmed that the exuded leukocytes are of the lymphocyte type; inoculations have proven positive in a number of cases, but *toxemia* is the most probable cause. It is most frequent in children. Lejars found one case of primary tuberculosis of the muscles.

XIII. Tuberculosis of the Skin.—1. **Lupus.**—Lupus, the most frequent form, in 66 per cent. of cases is located on the nose, lips and angle of the eye. It occurs with pulmonary tuberculosis in 21 per cent. and other tuberculosis in 62 per cent. It is typical granulation tissue with true tuberculous tumors, which contain few bacilli and often advances on one border while it cicatrizes on another.

2. **Scrofuloderma.**—This granuloma appears as movable, painless, subcutaneous nodes, which later become cold abscesses (the so-called scrofulous gumma, *gomme scrofuleuse*); they discharge a milky fluid and leave a sharply marked granulating ulcer.

3. **Lichen.**—Lichen scrofulosorum is often tuberculous; the eruption originates from the follicles and is constituted of thick, oily, epidermal cells removable without bleeding, and distributed in sharply marked groups of yellow-brown nodules as large as a pin-head.

4. **Tuberculosis Cutis.**—Tuberculosis cutis verrucosa of Riehl and Paltauf is observed on the backs of the hands and fingers and in the interdigital folds, chiefly in butchers, cooks and horsemen.

5. **Postmortem Tubercles.**—Laennec referred his pulmonary phthisis, though probably incorrectly, to a postmortem tubercle. Gerber suffered from tuberculosis of the axillary glands following a tubercle. They are observed in butchers, veterinary surgeons and pathologists, especially in those beyond middle life.

6. **Ulcers.**—Tuberculous ulcers may develop from cuts by broken sputum cups, bites of tuberculous subjects, ants or flies, burns, tattooing, skin grafting, circumcision or vaccination.

Treatment of Tuberculosis. — (*A*) **Prophylaxis.** — 1. ANTIBACILLARY PREVENTION concerns the etiological factors in the dissemination of the bacillus. Cleanliness of the skin; disinfection of contaminated feces, urine or pus; confiscation of diseased meat; and regulation of dairies and inspection of cows are important; but the *destruction of infective sputum* is the prime indication. Tuberculous, pneumonic, grippal and other sputa must be destroyed. An appeal is made to the conscience or selfishness of tuberculous patients, setting forth the risks of autoreinfection. Of all educators, Biggs, of New York, and Flick, of Philadelphia, have rendered the greatest service. Patients should carry small spit cups, of which Dettweiler's and Major Appel's are the best; the sputum should be burned or boiled before it dries. Spittoons are dangerous. The patient should sleep alone, and kissing must be interdicted. The bedclothes, linen and eating utensils should be steamed or boiled. Rooms should be cleaned with moist cloths and swept with the windows and doors open. The danger of swallowing sputum must be set forth, and the mouth washed out with some antiseptic before eating, though a few bacilli with the food are probably destroyed by the gastric juice.

In Germany the reduction of mortality by 33 per cent. is clearly due to an awakened public intelligence. The dangers of overcrowding, public conveyances and foul air, are already impressed upon the public, yet public school ventilation remains a scandal.

2. Governmental Prophylaxis, less promising in republican than in autocratic states, is important. It comprises: Education of the public by publication of the dangers and preventive measures in tuberculosis; the establishment of sanatoria for the poor, among whom the mortality is four times, and the morbidity many times greater than in the rich; the building of separate wards for the hopelessly diseased; the reporting of the disease; and the regulation of the air capacity of dwelling rooms, in tenements, factories and stores. Aside from great suffering and sorrow of thousands in every large community, there is an enormous economical waste in work and wages, an enormous cost in hospital care and a great loss in live stock. The sociological damage is apparent, as the ravages of tuberculosis involve the working years of life (15 to 60), while 94 per cent. of deaths from diphtheria occur under ten years.

3. Individual Prophylaxis concerns increasing physiological resistance and promoting sound development in children. Weakly children should be brought up and taught in the open air, judiciously fed, watched during acute infections, sent into the country during vacation, taught moderation and later, should be informed as to the danger of alcoholism, sexual excesses and infections. Milk and cream should be pasteurized or better, boiled.

(*B*) **Hygienic Treatment.**—The *three great factors are fresh air, proper food* and *rest.* Bodington (1839), in England, treated cases by fresh air, but to Brehmer (1850), in Germany, is due the credit of soundly establishing the fresh-air treatment and the curability of phthisis. A few of his views are untenable, but many of them are the basis of the modern methods practised by McCormack, Driver, Dettweiler, Trudeau, Knopf and others. Patients were treated in sanatoria, and those at Nordrach and Saranac and many others, have given such excellent results that institutions of this class in America number almost three hundred. Results depend on (*a*) the extent of the disease; (*b*) the condition of organs other than the lungs, as the pleura, intestines, larynx and heart; and (*c*) the social status of the case. Trudeau brought about recovery in 31 per cent. of all, and in 66 per cent. of incipient, cases; Rumpf followed 97 per cent. of his cases: 70 per cent. of incipient cases were able to work—"the best test of a cure"; 55 per cent. of those in the second stage and 23 per cent. of those in the third, recovered. The chief value of sanatorium treatment is that the patient learns the lesson of living properly.

1. Fresh Air.—This is the chief hygienic factor in treatment, at home, in sanatoria or in change of climate. (*a*) *Treatment at home* is especially important, as but 5 per cent. of tuberculous people are able to leave home. The patient should be carefully clothed and recline in the sun with the windows of his room open, or lie on a veranda, one-quarter to one-half the sunny hours, according to the season. At night the patient's bed can be brought to an open window, from which "a window

tent" of canvas encloses the head and leaves him out of doors. Rain, snow, dampness and extreme cold are no contra-indications, nor are fever, cough and hemoptysis; but wind, dust and sudden variations in temperature are to be avoided. Very thick clothing, so frequently observed among the poor, is unhygienic. Acute caseous and advanced types generally should be kept at home. Extreme care in disinfection is imperative lest the home become a menace to the family. For those unable to obtain sanatorium treatment, "classes" have proved of the greatest value (Pratt). (*b*) *Sanatorium treatment* offers the advantage of strict discipline, systematic living and the constant regulation of the details of everyday life by a physician. Sanatoria should be located well outside of every large centre of population. (*c*) Change of *climate* was once considered absolutely indispensable; now it is said "to be not without influence." No climate is specific but many offer advantages over the northern climate. *High altitudes* give purer air, stimulation of breathing, increase of the chest dimensions, growth of the muscles of inspiration, increase of metabolism and stimulation of the appetite and blood-making organs. Colorado, Arizona and New Mexico (4000 to 7000 feet elevation) are excellent for opportunities to secure permanent occupation after recovery; because of the emphysema induced by altitudes, a return to the sea level should be avoided. St. Moritz, Les Avants and Davos (5200 feet elevation) offer a dry, clear, cold, still atmosphere, where patients may reside summer and winter; it resembles the climate of Colorado, Montana and northern Wyoming. There are also the Peruvian Andes and numerous other altitudes (see *Solly's Climatology*). High altitudes are best adapted to those with incipient lesions, slight cavity formation and little emaciation, or slowly progressing or torpid cases. Slight fever and slight hemoptysis are not contra-indications; emphysema, weak heart, constant fever, great nervousness or repeated hemoptysis are contra-indications. *Moderate altitudes* (2000 to 2500 feet) best serve the majority of cases, as Asheville (2250 feet), the Adirondacks (2000 feet), Aikin (2250 feet), Summerville, Thomasville, etc. The climate of Minnesota, Nebraska and Dakota (1000 feet elevation) is cool and dry; dryness is more desirable than lack of variability. Moderate elevations do not induce emphysema, whence return to the sea level is easier. The *sea level* is best for the tuberculous aged, quiescent cases with great cicatrization or extensive damage, those with diabetes, albuminuria, nephritis, bone disease, marked anemia, repeated hemoptysis, emphysema or rapid softening, incurable forms, thin, nervous and exophthalmic subjects. Sea voyages are experimental; those with incipient forms, genito-urinary, glandular or osseous disease or open wounds are often helped. Moist, warm climates at the sea level are especially adapted to laryngeal disease—as the Bermudas, Florida, the Madeira or Canary Islands; patients with dyspepsia and diarrhea do well in Corsica, Palermo and Capri. Warm, dry climates, as southern California, Egypt, Algiers or the Riviera are good for catarrhal types. Climate alone, without reasonable hygiene, rarely benefits. Direct exposure of the body to the sun (heliotherapy) is advocated, especially in surgical tuberculosis.

2. Nutrition.—Most physicians recommend 5 or 6 meals daily, but better results are often achieved by 3 meals, whereby the stomach is allowed rest. Feeding requires individualization. The *staple diet* is *meat, eggs, milk, cream* and *butter,* fish, well-cooked leguminous vegetables (advantageously given in soups), oil on salads, bread, pastries and cakes. If anorexia is of the nervous type, patients must be compelled to eat, but long hours in the fresh air usually stimulate the appetite. With habitually light eaters caution is necessary; beef-juice, milk and koumyss should be given; the amount should be gradually increased and other foods added. Wine, vermouth, fluidextract of condurango ℿxv–xxx or strychnine, gr. $\frac{1}{30}$ in solution, half an hour before meals, are valuable appetizers. Raw beef (zomotherapy) was recommended by Richet and Hericourt; fresh lean meat is cut, soaked in one-fifth its weight of water, pressed to extract the blood and fluid, and administered; it must be freshly prepared, as it soon becomes tainted. Raw eggs are often given between or just before meals, beginning with 1 and increasing to 4, three times daily, the taste being disguised by orange-juice. Fever does not contra-indicate full feeding. More than $1\frac{1}{2}$ quarts of milk induces gastric atony; it is given to advantage with bread, crackers or toast, in order to divide the curds; it may be peptonized, disguised with cocoa or cognac or given by the rectum (*v.* Gastric Ulcer); cream and butter are excellent substitutes for cod-liver oil, and buttermilk is indicated when the stomach is weak. Fats and carbohydrates are necessary to offset nitrogenous waste; the dark form of cod-liver oil (*v. i.*), lipanin (94 parts olive oil and 6 parts oleic acid), malt, honey and cocoa are valuable. In overfeeding (*suralimentation*) the *tolerance of the stomach must be considered;* Debove's method consists of gastric lavage followed by the introduction through the tube of three ounces of powdered meat, a quart of milk and three eggs. Alcohol, administered less often and in smaller amounts than formerly, is frequently indicated symptomatically (*v. i.*); small amounts of red wine or cognac and whisky in egg-nogs are very valuable as a food and tonic. Tobacco should be interdicted; in special cases a cigar or two daily may be allowed, but the smoke must never be inhaled.

3. Rest.—Rest is one essential in Dettweiler's therapy. Patients should lie in the bright sunshine and fresh air, and active mental occupation should be carefully avoided. Exercise destroys tissue, induces anemia and irritates the heart; it is distinctly contra-indicated by fever, emaciation, hemoptysis, rapid pulse, nausea and coughing. "Too many cases of phthisis *walk* into their graves." Late in the treatment *graduated exercise* may be taken. Pulmonary gymnastics and pneumatic differentiation are clearly injurious. Hardening and exercise should be obtained by quick, dry rubs with a large, coarse towel, after which rapid partial, and then general, wet rubs may be instituted; hydrotherapy is shunned when there is any tendency to hemoptysis.

(*C*) **Specifics.**—As yet there is no specific for tuberculosis.

Tuberculin Therapy.—(i) *Theory of Action.*—Tuberculin is an endotoxin and not a toxin. Its injection causes an increase in antibodies (opsonins, agglutinins and precipitins) and an increased tolerance of the

tuberculin; unhappily, however, increased tolerance to tuberculin is not identical with immunity, as is demonstrated clinically at autopsy and in animals. The immune substances may be antitoxic but possibly not bacteriolytic. In treatment it is not specific but merely accessory. (ii) The *indications* are: Incipient subjects who are well nourished, without fever and come to a stand-still after some improvement. (iii) The *contra-indications* embrace malnutrition, rapid wasting, profound toxemia, pronounced fever, acute phthisis, rapid heart action, nephritis and great nervousness. (iv) *Dosage* is solely empirical, since there is no scientific gauge of tolerance (Wright's opsonic index and other methods are open to error). Carelessly used, tuberculin is dangerous. Of the "old tuberculin" (O. T.); the initial dose should be 0.0000001 gm.; the initial dosage for the "bacillus emulsion" (B. E.) is 0.00000001 gm., increasing gradually toward the decimal point, every three days at first, and later once a week between doses. The maximum dose of O. T. is 0.0001 and B. E. 0.0005; the course is 5 months, interrupted when coryza, cough, indigestion, fever, emaciation or hemoptysis intervenes. (v) *Results:* Trudeau conservatively states that "tuberculin immunizes, prolongs life, causes the bacilli to disappear from the sputum, aborts commencing infection and also the smouldering fires of chronic infection." The subject is still *sub judice* and as one writer puts it, "a chaos of personal impressions."

(*D*) **Expectant or Symptomatic Treatment.**—1. FEVER.—Rest, digestible food, fresh air and change of climate are indicated in great preference to drugs. Strong wine may be administered as the fever begins to rise; arsenic is recommended, but no remedy in safe doses controls fever. Quinine disturbs the digestion. Sponging is efficacious.

2. COUGH.—A slight morning cough, removing secretion, needs no restraint, but, if excessive, it begets coughing by congesting the lungs, and its consecutive exhaustion, insomnia, aspiration and vomiting necessitate interference. Fresh air and avoidance of dirt, dust and smoke are most beneficial. Rest, especially after eating, should be enforced. The cough is somewhat under the control of the will, as Galen knew; the comment on its absence, especially at the table, is universal among observers of sanatorium methods; slow breathing, with the head slightly thrown back, or the sipping of warm water, with 10 grains of salt, before meals often aids in suppressing the emetic cough. *Creosote* discovered by Reichenbach, in 1832, is not specific, but often increases the appetite, modifies abundant secretion from the bronchi (by which the drug is in part eliminated), and relieves the cough. Administration in the fluid form is best, beginning with one drop, well diluted, after meals, and increasing to ten drops; full dosage often irritates the stomach and kidneys; it should not be given in febrile cases or in hemoptysis. Creosote may be administered in milk, water, gentian or sherry wine:

℞—Creosoti . . . . . ℥iss
Glycerini . . . . . ℥ss
Tr. gentianæ co. . . . . ℥iv
M. et S.—One teaspoonful after meals, well diluted.

℞—Creosoti . . . . . ℥iss
Mucilag. acaciæ . . . . . ℥iij
Pulv. althææ . . . . . q. s.
M. et fac pilulas 10.
S.—One after meals.

Guaiacol carbonate is better tolerated and is cheaper:

℞—Guaiacolis carbonatis . . . . . ʒiij
Strychninæ nitratis,
Arseni trioxidi . . . . . āā gr. iss
Sparteinæ sulphatis . . . . . gr. xlv
M. et fac chartulas 40.
Sig.—One powder after meals.

Cod-liver oil, first used by Hughes-Bennett, is usually given in full doses which disturb digestion; the first dose should be 10 drops and the amount gradually increased. It is most valuable in tuberculosis of the glands and bones. Eructations can be avoided by administration one hour after meals, when the food leaves the stomach. The oil is readily absorbed because of the bile contained; it is possibly alterative but is not superior to butter or cream. It may be floated on whisky, beer or milk, and gulped down. Its taste may be partly disguised by using syrup of orange, capsules, or a "chaser" of whisky. *Opiates* may be used, such as paregoric; codeine gr. $\frac{1}{4}$ with aq. laurocerasi and glycerin āā ♏xv and syr. tolutani ʒj q. i. d.; heroine gr. $\frac{1}{12}$ q. i. d.; even morphine sulp. gr. $\frac{1}{4}$ with ac. hydrocyanici dil. ♏ij; myrtol gr. ij, A.M. and P.M.; terpin hydrate gr. ij–v, t. i. d.; and ol. terebinthinæ ♏ij. (See formulæ under BRONCHITIS.)

℞—Tr. belladonnæ . . . . . ʒss
Spts. chloroformi . . . . . ℥iss
Mucilag. acaciæ . . . . . ʒj
Aquæ . . . . . q. s. ad. ʒiij
M. et S.—One teaspoonful after meals, or as indicated.

Expectorants are to be avoided because of their nauseating tendencies. Tr. benzoin co., 1 per cent. phenol, and creosote may be vaporized. The nasopharyngeal mucosa may be painted with iodine, potas. iodide and glycerin in proportions of 1, 10 and 100; or 20 per cent. menthol applied locally (*v.* LARYNGEAL TUBERCULOSIS).

3. SWEATS.—The room should be cool at night, the bed not too heavily covered, and the body protected by a flannel night-gown. A glass of cold milk, with ʒij of cognac, given at bedtime, is often beneficial or morphine when sweats are associated with cough and insufficient oxygenation. Atropine gr. $\frac{1}{100}$ to $\frac{1}{80}$, and camphoric acid gr. xv to xxx, at bedtime, are superior to aromatic sulphuric acid ♏x to xx, agaricin gr. $\frac{1}{16}$ to $\frac{1}{12}$, tr. nucis vomicæ ♏xxx, picrotoxin gr. $\frac{1}{60}$ and muscarin ♏v of a 1 per cent. solution.

℞—Quininæ sulph. . . . . ʒss
Calcii hypophosphitis . . . . . ʒij
Extr. nucis vomicæ . . . . . gr. v
M. et fac pilulas 20.
S.—One after meals.

4. HEMOPTYSIS.—Exertion, loud talking or singing and excesses in alcohol, venery, coffee or tobacco, must be shunned in all cases. It is well to warn patients of the possibility of hemoptysis, in order to prevent undue excitement, if it appears. Effusive pleurisy should not

be tapped in recent bleeding from the lungs, as thoracocentesis has produced fatal hemoptysis, The indications are absolute physical and mental rest, the semirecumbent posture and silence; massage and stimulants are contra-indicated; no physical examination should be made except by auscultation, as percussion is obviously dangerous; and a hypodermic of *morphine* should be given to lessen cough, quiet the heart and allay the excitement attending most hemorrhages. As the tonus of the pulmonary vessels is beyond regulation, ergotin, tannic acid and other hemostatics are useless, though digitalis and fresh suprarenal extract gr. ij–v are sometimes efficacious. Salt ʒj is a popular remedy and is supposed to act by stimulating the vagus. An ice-bag should be placed over the heart and one over the lung, if rales are heard. Deep breathing seems to lessen bleeding. Calcium lactate gr. xv t. i. d. for two days is valuable. *Amyl nitrite,* recommended by F. Hare, is given at midnight, when the vasomotor centre is regaining its tone, lost during sleep. Ol. terebinthinæ ♏ij–iij is considered the best drug by Fräntzel. In severe or recurrent hemorrhage, bandaging of the extremities (Hippocrates), to lessen the return flow of blood, or injection of *horse serum* may be indicated. As a last resort in hemoptysis, or in cavity formation, *artificial pneumothorax* has been advocated (Morton, 1833).[1]

5. Pain.—Pleuritic pain is treated by adhesive straps; if muscular it is treated by codeine, fomentations or phenacetin.

6. Dyspnea.—If due to stagnant secretion, change of posture aids in its evacuation; if asthmatic, iodide gr. iij is indicated, but with care lest it congest the lungs; if cardiac, digitalis or strychnine is indicated.

7. Cardiac Weakness.—Rest, an ice-bag over the precordia, champagne, camphor, strychnine or digitalis are in order.

8. Insomnia.—The patient should have fresh air night and day, a light, early evening meal, a glass of beer, porter or some whisky on retiring. Bromides, codeine and morphine are beneficial.

9. Gastro-intestinal Symptoms.—Nervous anorexia is relieved by fresh air, forced feeding and guaiacol (*v. s.*). Dyspeptic symptoms necessitate a scant diet and feeding by rectum. In gastric atony, frequent meals of small volume, limitation of fluids and strychnine are indicated. For gas formation or pain the following are indicated; phenol ♏j; resorcin gr. iij; bismuth gr. x; extr. belladonnæ or spts. chloroformi ♏xx. Pyrosis may be corrected by sod. bicarb. and magnesia carb. āā gr. xxx. Hyperacidity demands a milk diet, belladonna, Carlsbad water and alkalies; anacidity is relieved by easily digested foods and hydrochloric acid. *Vomiting* is caused by the cough, overfeeding, overmedication or irritation of the pharynx (Lugol's solution locally, or gargles 1 to 10 of pot. bromide); small doses of carbolic acid are most successful (*v.* Typhoid). *Constipation* (*q. v.*) is treated by the usual measures. Diarrhea may be dyspeptic, toxemic, ulcerative or amyloid; the following are indicated:

[1] Nitrogen gas is injected, under manometric control, no gas being injected till the column oscillates several centimeters with each inspiration (whereby it is claimed that gas embolism, subcutaneous emphysema and pulmonary perforation are avoided); 300–700 c.c. may be injected. Contra-indications are bilateral involvement, dry or effusive pleurisy, cardiorenal disease or great debility. In all probability, the revival of this method will result in a repetition of the former fiasco. Some 3000 cases have been treated, with some fatalities.

tannic acid gr. v; opium gr. j; lead acetate gr. ij; camphor gr. ij; tannigen gr. vij–xv, or bismuth ℥j (see TYPHOID).

10. ANEMIA.—This is benefited by fresh air, change to a high altitude, full diet, iron in small doses and arsenic.

11. SEXUAL SYMPTOMS.—Intercourse must be forbidden in incipient as well as in advanced cases. Three years after recovery marriage may sometimes be allowed; it is generally less injurious to men than to women. Tuberculous parents may rear healthy children, but tuberculosis ("scrofula") is common. In 66 per cent. of pregnant tuberculous women the prognosis is bad, and laryngeal tuberculosis is almost invariably fatal within a short time. In severe cases abortion may be contemplated; Martin and Schauta advise abortion, but in 70 per cent. of cases abortion does not alter the clinical course.

12. TREATMENT OF OTHER COMPLICATIONS.—(*v. s.*)

LEPROSY.

Definition.—A chronic incurable contagion, caused by the Bacillus lepræ and characterized clinically by nodules, which are observed externally and internally in many organs; or by perineuritis leprosa, which causes sensory, motor and other symptoms.

Distribution.—The home of leprosy is Egypt, where it existed 2400 or even 4620 B.C. The leprosy of Leviticus probably includes other diseases. It prevailed in India 700, and in China 400 years B.C. Just before the Christian era, Pompey's soldiers brought leprosy to Italy, whence it spread over Europe and the Crusaders at the end of the thirteenth century further disseminated the disease. In Europe there were 20,000 asylums, but many cases were probably syphilis; the disease has steadily decreased since the sixteenth century. It is estimated that at present there are 3,000,000 cases in the world. Cases are found in Europe, Asia, Africa and America (New Brunswick, Nova Scotia and British Columbia); in the United States (California, Louisiana, Minnesota, North Dakota, Florida; in 1912 there were 146 lepers in this country, of whom half were native born).

Bacteriology.—The *Bacillus lepræ* was found by Hansen (1871) and fully described by Neisser (1879). It is a fine, narrow rod, measuring ½ to ¾ of a red cell, and closely resembles the tubercle bacillus morphologically and in its staining properties; both bacilli contain fat. Inoculation is the surest differential criterion. In the internal viscera it may exceed the dimensions attained in the skin and mucosæ five or six times. It is motile and has been cultivated. It is found in the typical nodes and infiltrations of the skin and upper mucosæ, fresh maculæ and recent nerve disease, sebaceous and hair glands, lymph glands, bloodvessels, blood during fever (metastatic distribution), and in practically all the viscera, secretions and excretions. Strickler found it in the nasal secretion in 96 per cent. of the nodular, 96 per cent. of the mixed, and in 66 per cent. of the nervous type. It corresponds to all of Koch's criteria; *i. e.*, it is always present in leprosy; it can be

cultivated, and has been inoculated in mice, guinea-pigs and monkeys; the value of successful inoculations on criminals under sentence of death has been justly criticized, for these cases developed in a leprous community. *Leprosy is contagious* only by long and intimate contact with lepers. The nodular is far more dangerous than the anesthetic type. Infection may be direct or indirect and chiefly through the skin and nose, and possibly the genitalia. Extension occurs more by the lymphatics than the bloodvessels. Only 4 per cent. of children of leprous parents acquire the disease. More cases occur in colored than in white races, more in men than in women, more in the poorer than in the upper and middle classes and largely between the ages of fifteen and thirty.

The *incubation* is at least three to five, sometimes twenty years.

Symptoms.—Visceral involvement is seen in every type; the main types are nearly always somewhat blended.

1. Nodular Type (*Lepra tuberculosa*).—The prodromal toxemic symptoms indicate an infection already established, but escaping recognition; they are depression, formication, sweats, pains, vertigo, digestive disturbance and temperature. After months or years, *maculæ* appear on the face and extremities; they may disappear, leave pigmentation or metamorphose into *nodules*, which are present in 75 per cent. of all cases. The nodule or *leproma*, is a granuloma similar to the tubercle or gumma, highly vascular and formed of a scanty reticulum, in which lie emigrated leukocytes, small epithelioid cells and the larger multinucleated lepra cells of Virchow, which contain close clumps of bacilli. The nodes by fusion form gelatinous, glistening and yellowish-red *infiltrations*. The nodes vary from the size of a millet seed to a hazel-nut or walnut; in the skin they are dark red and later yellowish-brown, glistening, vascular, slightly desquamating and at first soft but later harder. *Sensation* is always *disturbed* in their vicinity. They are most conspicuous on the face and hands, but also appear on the extensor surfaces and very seldom on the soles, palms, scalp or penis. The forehead, nose, cheeks, chin, lips and ears are thickened, the nose becomes flatter, the eyebrows, lashes and beard are shed. The eyes are involved in over 90 per cent. of the cases; the expression is leonine (*leontiasis*), or, as the ancients called it, faun-like (*satyriasis*). The nodes grow and extend slowly, and the lymph vessels and glands become enlarged and tender. Sometimes the progression is intermittently acute, with fever and an eruption resembling erysipelas. They often *necrose* and ulcerate. At the same time or later, nodules which are prone to early ulceration and infiltration occlude the *nose*, roughen the voice, distort the larynx and throat, and impede mastication, swallowing or respiration. Cicatrization of the face causes added *deformity*.

2. Nervous Type (*Lepra nervorum, anesthetica, mutilans*).—The most common prodromes are fever, neuralgia, paresthesia, hyperesthesia and cerebral congestion. *Maculæ* develop on the face, trunk or limbs; they are pale or dark red, slightly raised, growing peripherally and paling centrally and present a glistening or powdered aspect. Histologically they are identical with the nodules, but contain fewer bacilli, contain no large cells and tend more toward fibrous change. Their

distribution is asymmetrical, like that of the underlying nerve lesions; they are often oversensitive, sometimes pigmented (*lepra nigra*) or leukoderma-like (*lepra alba*).

Sensory disturbances are due to *lesions* in the *nerve trunks,* which are the seat of a perineuritis and interstitial neuritis which slowly compress the nerve fibers. The connective tissue and the bacilli differentiate this from the parenchymatous neuritides. Unlike ordinary neuritis, leprous neuritis affects the *sensory more than the motor filaments.* There is an irregular dissociated anesthesia in which the perception of pain and temperature is usually more dulled than other forms of sensation, mostly in the peripheral branches in the arms and legs. Sensation may be perverted or retarded and sudden anesthesia is considered rather characteristic. Pain may be slight or agonizing, as in a personal case of ten years' duration. The *nerve trunks are palpable* and tender in many cases; in the case cited the ulnar nerve was thicker than a lead-pencil; the auricular nerve is felt in 90 per cent. Motor weakness and atrophy are less common and rarely extreme; the oral, sometimes the masticatory and ocular muscles are paretic; ulnar and peroneal implication causes the "claw-hand" and "claw-foot"; facial paralysis occurs in 9 per cent. of cases. The tendon reflexes are often increased, sometimes normal or decreased; there is often fibrillation and increased myotatic irritability. *Trophic* changes include edema, glossy skin, "*mal perforant*" (26 per cent.), loss of hair or nails, bone resorption, rheumatoid swellings, ankylosis and pigmentation. Great trophic changes mark the *lepra mutilans.*

3. Mixed Type.—This is a combination of the nodose and anesthetic types. *In all developed forms the internal tissues are invaded:* the liver, spleen and lymph glands are enlarged; the lungs show peribronchial foci, cavities or induration; and the intestine is infiltrated. The sexual functions are decreased or lost. Infantilism results in cases developing before puberty. Cachexia, sensitiveness to cold, low temperature and heart weakness develop.

Course and Prognosis.—The course is slow and progressive, the outcome invariably fatal. The nodular type covers eight to twelve years, the anesthetic form fifteen to twenty years, but either variety may endure seven decades; 38 per cent. of cases die of leprosy (exhaustion, diarrhea, cardiac weakness), 22 of chronic nephritis, 17 of lung disease (oftener leprous than tuberculous), 10 of diarrhea (leprous infiltration of the gut), and the remaining 13 per cent. die of erysipelas, sepsis, amyloidosis and intercurrent disease.

Diagnosis.—The diagnosis is based on the history of residence and exposure which is often suppressed by the patient, on the macules or leukoderma found in 90 per cent., on the anesthesia, amyotrophy and multilation, and on the finding of the bacillus in the nasal secretion, nodes and macules. In some reports, a positive Wassermann is given.

Treatment.—1. Prophylaxis.—All suspicious cases should be registered and all clear cases sequestrated.

2. Therapy.—Few drugs are helpful. (*a*) Gurjun oil ℥ss–ij in water or lime-water; (*b*) chaulmoogra oil, in milk, beginning with a few

minims and increasing to ℥j–iij; (*c*) salves of 5 per cent. pyrogallol or 10 per cent. chrysarobin; (*d*) Calamette's antivenin, 20 to 30 c.c.; (*e*) arsenic, nastin, salvarsan, carbon dioxide snow and *x*-rays are recommended.

NON-BACTERIAL FUNGUS INFECTIONS.

ACTINOMYCOSIS.

Definition.—A chronic infection caused by the actinomyces (literally "ray fungus"); mostly sporadic in cattle and occasionally in man; and characterized by chronic inflammation in the mouth, head or neck, lungs or intestines or abdomen.

History.—Von Langenbeck (1845) and Davaine (1850), observed the peculiar sulphur granules in the pus. Bollinger (1877) first scientifically described the fungus in cattle, and J. Israel (1878) found the parasite in human cases, but to Ponfick (1879) is due the credit of identifying the bovine and the human types.

Etiology.—Statistics in Berlin, Breslau and Vienna show large numbers of cases; there are more in Scotland, Denmark, Italy and Russia than in America, where up to 1902, 100 cases were observed. Seventy per cent. of the cases occur in males and 33 per cent. between twenty and thirty; 82 per cent. between ten and fifty. The fungus enters chiefly by the mouth, in which ragged teeth, abrasions, etc., favor its access. Israel and Lord believe it inhabits the tonsils and carious teeth. Though little is known of it outside of the body, it probably exists in the intestines of herbivora. Most cases occur in young cattle at dentition and 89 per cent. in the last half of the year, when the ingested grain is dry.

Bacteriology.—The *parasite*, classified as a streptothrix is found in small granules which measure 0.15 to 0.75 millimeters, usually ovoid or "radiate" when squeezed under the cover-glass. The younger forms are transparent, sago-like, whitish-gray bodies which may be easily overlooked; the older *sulphur-like granules* consist of threads and coccoid bodies which some consider spores. Clubbing is considered a degenerative change. The threads divide and branch. The fungus is therefore pleomorphic. It stains with the usual dyes and Gram's method. Cultures are successful in 50 per cent. of the cases, by mixing the granules with softened gelatin and bouillon. The colonies are thick, folded, membranous, resistant and at first have a mucoid or crystalline appearance. Infection is very rarely direct either from animals to man or from one person to another. The virulence is therefore of low grade.

Forms.—Incubation averages four weeks. Clinically there are *three main forms:* (1) That of the head and neck; (2) the lower digestive tract; and (3) the respiratory tract.

1. Head and Neck.—This form constitutes 50 per cent. of actinomycosis. The atrium is the mouth and throat. The fungus is surrounded by a massive accumulation of round cells which soon degenerate centrally.

The connective-tissue reaction is marked and the proliferation shows numerous round or polygonal cells with large nuclei—the epithelioid cells. Giant cells also form. The process is considered chronic inflammation by Bostroem, and by Israel an *infective granuloma*, resembling the tuberculous or syphilitic process. The destiny of the granulations is modified by the resistance of the tissues and by the animal diseased: (*a*) *In man* the granulation wall melts into a mucoid, creamy fluid, in which there are oil, fibrin, blood, pigment and actinomycotic cocci and threads; the "cold" abscess is formed by the fungus itself which, unaided by pyogenic organisms, burrows to the surface by circuitous *fistulæ;* it discharges a thin, puriform secretion like that of tuberculous glands but contains the *sulphur-like grains.* (*b*) *In cattle and horses* the granulation tissue is more effectually limitative and develops hard, board-like or ligneous tumors ("lumpy jaw," "wooden tongue"); suppuration and miliary dissemination are less, and calcification is more frequent than in man. In human subjects the infiltration may involve the neck, face, tongue, pharynx, tonsils, larynx and thyroid gland. There is frequent inflammation of the soft tissues, especially submaxillary or submental infiltration and superficial invasion of the bones. In a few cases the maxillæ may be primarily invaded. *Sarcoma* and *caries* may be simulated. It may *spread regionally* to the base of the brain and meninges; along the spine causing vertebral erosion; and behind the sternum with mediastinal infiltration.

Symptoms.—The fever is irregular. Pain and tenderness are not marked, for the process is indolent or chronic. In 26 cases of involvement of the tongue, its forms were a hard tumor, localized abscess or diffuse suppuration; it causes less pain and adenitis than does cancer. Dysphagia accompanies retropharyngeal involvement, edema occurs in laryngeal localization and other local interference such as venous obstruction is frequent. The writer observed trismus and severe unilateral trifacial neuralgia from a primary focus at the base of the skull, recovering after operation. Besides the above regional invasion, *hematogenous extension* by metastasis may occur in this type as well as in the others to be described later. Trauma may initiate generalization. Unlike tuberculosis, extension to the lymphatics, or general dissemination by them, is unusual. In the head-and-neck form the mortality is 10 per cent.

2. Digestive Tract.—The digestive tract ranks next in frequency of infection (25 per cent.); 50 per cent. occurs in the region of the ileum, cecum and appendix (*perityphlitis actinomycotica*); of 200 instances of inflamed cecum, 4 per cent. were actinomycotic. Local pain, tenderness, induration and adhesions follow the primary lesion in the intestine, which in rare instances remains superficial (enteritis actinomycotica superficialis). Small nodes develop in the submucosa, followed by softening and the development of small ulcers, the undermined edges and uneven bases of which resemble tuberculous ulcers. Secondary pyogenic infections are frequent. The ulcers may cicatrize or fuse into larger ulcerations.

Symptoms.—The signs were those of appendicitis in 150 cases on record. Colic and vomiting are often present and constipation is more frequent than diarrhea. *Extension* of the process downward involves the iliac

bone and occasionally the hip, bladder, ovaries, vagina or rectum; the ray fungus is voided in the urine and feces; and anteriorly, preperitoneal, suprapubic or muscular infiltrations occur, with external fistulæ. The process then extends backward into the retroperitoneal cellular tissue, in which it may burrow around the kidneys or into the chest—perinephritis, peripsoitis, parametritis or peripleuritis. In rare cases, freely movable operable tumors may develop. Vascular metastases may occur; liver abscess is the most frequent development, and is secondary, though sometimes apparently primary (40 cases reported). In 70 per cent. death results from exhaustion, mixed infection or amyloidosis. Diagnosis is made from the yellow granules or microscopic recognition of the fungus.

3. Respiratory Tract.—Respiratory involvement is less frequent (20 per cent.). It is (*a*) secondary to regional extension from the neck or abdomen, or to infarcts, or, still less frequently, (*b*) primary in the lungs (very rarely as a fibrinous or actinomycotic bronchitis). Most often it invades the alveoli. The lower lobes, its usual seat, show a gray hepatization with the ordinary actinomycotic changes. Fusion of the usually small foci may involve large portions of tissue, but demarcation is usual in the lung. The clinical picture varies: (i) In some instances *cavity formation* is obvious, with hectic fever, a harassing cough and malnutrition; the sputum is puriform, sometimes fetid and stained with altered blood like raspberry jelly and often reveals the actinomycosis rays. (ii) *Induration* may occur from cicatricial development. (iii) *Pleurisy, peripleuritis*, pericarditis or mediastinitis may be the dominant finding. There may be much luxation of the intrathoracic contents, with dyspnea, dilatation and hydrops. Penetration may occur through the chest wall with characteristic discharge from the sinuses which run along the spine and iliopsoas muscle, or into the abdomen. Hematogenous generalization occurs as in other forms. In 25 per cent. of cases the ray fungus is seen in the sputum and *tuberculosis* is excluded by absence of tubercle bacilli and elastic fibers; differentiation may be impossible. The *course* is usually subchronic (averaging under a year), less often chronic (two or three years) and very rarely acute. Only 6 recoveries are reported.

4. Other Localizations are rare; 23 primary skin cases are reported. They may resemble lupus or tubercle. Primary brain lesions may simulate brain tumor or cause cortical epilepsy. Very few cases of primary genito-urinary infection are recorded. The lachrymal duct or middle ear is very rarely the primary focus.

Diagnosis.—Similar fungi may cause difficulty in diagnosis (*v. i.*).

Treatment.—1. Prophylaxis.—Care is necessary in regard to picking the teeth with straws. Thorough cooking of cereals is essential.

2. Surgical Extirpation.—A sharp spoon is used to clean accessible foci and fistulæ, which are then packed with iodoform or sublimate gauze. Injections of bichloride of mercury (0.5 per cent.) are recommended. Recurrence is frequent in abdominal types.

3. Potassium Iodide.—In cases inaccessible to the knife, potassium iodide is excellent. It is not parasiticidal, but large doses soften the

tissues, prepare operable cases for surgical measures, inhibit growth and minimize metastasis; 53 per cent. of bovine cases recover under its use. Rydygier injects large quantities of a 1 per cent. solution into the affected tissues. Tuberculin, vaccine therapy and the internal use of copper sulphate, $\frac{1}{4}$ to 1 grain are recommended.

NOCARDIOSIS.

J. H. Wright describes nocardia as an acid-fast, branching streptothrix, differing from actinomycosis in that it does not occur in compact structures, does not radiate or form club-bearing granules and never is localized about the mouth and jaw. Wright collected a dozen cases in which the lesions and symptoms resembled pulmonary tuberculosis or septicopyemia (abscesses in the skin, brain, etc.). The prognosis is unfavorable, although iodides sometimes help.

SPOROTRICHOSIS.

Schenk (1896) discovered the Sporotrichum Schenkii, a fungus with branched mycelia and numerous spores. Over 200 cases are reported, largely in farmers, in whom trauma probably opens the way for cutaneous infection. Its toxin produces little fever. The serum exhibits specific agglutination. Infection follows the lymphatics. The hard nodules may resemble a chancre; other hard nodes produce subcutaneous abscesses; still others resemble cutaneous gumma or tuberculoma histologically and clinically. Lesions may appear in the mucosæ or generalization may ensue with lesions in the bones, joints, muscles, lungs, etc., by way of the blood stream. Surgery and iodides are indicated.

OIDIOMYCOSIS.

Coccidioidal granuloma is a specific, subacute or chronic infectious disease. Twenty-four cases have been recognized in various parts of the western hemisphere, most of them in California. The histological changes in the lesions closely resemble those of tuberculosis. Differential diagnosis depends on finding the specific microörganism, *Coccidioides immitis,* a species of mould belonging to the Ascomycetes. In the animal body it exists as spheres enclosed in doubly contoured capsules and multiplies by endogenous spore formation. As to *blastomycosis,* caused by the Cryptococcus dermatidis, Brown and Cummins point out well-defined differences in the pathogenicity of the two diseases, coccidioidal granuloma being always and often rapidly fatal in man, while blastomycosis is commonly not so, except for the systemic cases. The clinical and pathological aspects of coccidioidal disease are those more closely resembling tuberculosis, as there is a greater predilection for the lymphatic system than in blastomycosis, and cutaneous lesions are likely to be more ulcerative. There appears but one reported case of coccidioidal disease in the female sex, whereas there have been many of blastomycosis.

Iodides have temporarily benefited many, and apparently cured a few, blastomycosis patients, whereas they have had no effect on the rapidly progressive lesions and toxemia of coccidioidal granuloma. Morphologically, in pus and solid tissues they are differentiable by the endosporulation in the one and budding in the other. Generalization may occur in the bones, joints, meninges, etc. Iodides, *x*-rays, radium and vaccines are indicated.

MYCETOMA.

Madura foot, first described by Kaempfer (1712), is largely seen in India; seven American cases are reported. Infection of the foot occurs usually in those going bare-footed—by the Streptothrix maduræ; its varieties are the red, black and yellow. A nodule on the sole forms, softens, ruptures, and by extension leads to enlargement of the foot, sinus formation and involvement of all structures of the part, muscles, bones, etc. Amputation is required.

ASPERGILLOSIS.

Aspergillus fumigatus infection may occur as a primary or secondary disease. As birds, cattle or even dogs may be affected, the disease may occur in pigeon-feeders or in hair-sorters, from rye-flour dust employed to remove grease from the hair. Bronchopneumonic patches develop, from which induration or necrosis ensues, resembling phthisis and generally ending fatally.

PROTOZOAN INFECTIONS.

SYPHILIS (POX, VARIOLA MAGNA, LUES VENEREA).

Syphilis is an ancient disease, as indicated by syphilitic bones belonging to the stone age. Nusi King described syphilis in China 2600 B.C. The first clear outbreak occurred in 1494 among the troops of Charles VIII. Fracastorius in 1530 called the disease Siphilus (a shepherd smitten with the disease by Apollo because of blasphemy). A full account of this disease will be given because of its collossal importance. Metchnikoff's historical sketch distinguishes three epochs: First, the era of superstition, in which the only discovery was mercury; the second empiric period concerned the clinical pathology and the differentiation of gonorrhea and chancroid; the third dates from 1905, since when the cause, experimental inoculation of syphilis, its serodiagnosis and the cultivation of the treponema have been determined.

Definition.—A specific chronic infection, caused by the Treponema (Spirochete) pallidum, and found in man only, either hereditary or acquired by inoculation, in the seat of which a primary lesion or hard chancre develops, followed in two or three months by secondary lesions

in the skin and mucosæ. After months or years, tertiary lesions or gummata develop in the viscera, bones and skin, followed in some instances by the so-called parasyphilitic manifestations, as tabes dorsalis, dementia and aneurysm.

Stages According to Ricord.—The Primary Stage.—(1) Incubation lasts until the *chancre* appears, when syphilis is already a general disease; (2) the chancre develops, the lymph vessels and adjacent glands enlarge, and there is a period of latency, the second incubation.

The Secondary Stage.—(1) Symptoms prodromal to the eruption, as fever or anemia, develop and then (2) the *eruption* appears on the skin and mucosæ, with headache, "rheumatism," iritis, or sometimes jaundice, albuminuria and splenic tumor.

The Tertiary Stage.—This may follow very closely upon the secondaries or appear with them (syphilis maligna and hereditary forms) but usually is not manifest for three or four years (even fifty or sixty). Tumor-like gummata occur in the skin and viscera, especially in the liver, scrotum, brain and cord. Unlike secondary manifestations, they are asymmetrical, persistent, recurrent and less contagious. There is a close histological resemblance between the primary, secondary and tertiary lesions.

The quaternary or parasyphilitic stage of Fournier, Sigmund, Lancereaux and Baümler is not included in Ricord's division. In this group of affections, the spirochete is also found, as in paresis.

I. Acquired Syphilis.—Etiology.—1. Transmission by Sexual Intercourse.—This causes the great majority of cases, but the term *lues venerea* is often inaccurate and unjust since syphilis may occur in the innocent (syphilis insontium); in certain districts in Russia 70 per cent. of syphilis is innocent. In 1905 Schaudinn and Hoffmann described a *treponema* or *spirochete pallida*, so named because of the difficulty in staining it. It is long, delicate, thread-like, refractile, actively motile, pointed at its ends and twisted like a corkscrew; it measures 4 to 20μ in length; it lies free and not in the cells; it resembles the spirochete of relapsing fever and is a flagellated protozoön. It is present in all cases, stages, types and tissues of syphilis. It has been inoculated in monkeys from man, from monkeys to rabbits and then to monkeys again through many generations. With Giemsa's stain the spirochetes are a delicate violet and the leukocyte nuclei deep red. The spirochete refringens, found in the mouth, ulcers, smegma and venereal warts, is differentiated by its being more quickly stained, its greater size and, particularly, by its broader and more wave-like undulations. (See Plate VII.) It was cultivated by Schereschewsky but first by Noguchi in pure cultures, thus fulfilling all of Koch's postulates.

The sound skin and mucosæ resist infection, and hence a raw surface, erosion or wound is necessary for infection. Syphilis not only has a certain choice of place in its inoculation, but persists with peculiar obstinacy in certain localities, notably about the genitalia, breasts, axillæ, navel, toes, angles of the mouth, tongue, iris, palate or tonsils, because of irritation by secretions, clothing, trauma, smoking, etc. These foci are of great moment in the recrudescence of the disease.

PLATE VII

Spirochetes of Syphilis. (Simon.)

a, S. refringens; *b*, S. pallida. (Stained with Giemsa's stain.)

Syphilis is called a disease of cities; though more common in cities, syphilis very frequently escapes recognition in the country.

2. Accidental Infection.—Accidental infection occurs in surgical practice, skin-grafting, postmortem infection (2 cases of personal observation), ritual circumcision, nursing, vaccination, kissing (as in the epidemic recorded by Shamberg), from use of pipes, in shaving, by scratches, from instruments used in glass-blowing, or from catheters. Bardinel describes infection of over 100 women by one midwife.

Symptoms.—1. Primary Stage.—After an incubation of two or three weeks, the *initial lesion* occurs (the *ulcus durum*, Hunterian chancre or initial sclerosis). Multiple lesions occur in 25 per cent. The *induration* is usually oval, always sharply localized, of variable or even cartilaginous hardness, freely movable, and usually papular and copper-colored. *Hardness* is the most constant characteristic of the chancre, which heals only when the hardness disappears. It is most characteristic in the third or fourth week, when the adjacent glands enlarge. Retrogressive ulceration or necrosis is frequent. The *ulcer* is round, elliptical, slit-like or irregular; it is hard, painless, with even surface, seldom with elevations or excavations; it has no areola, but a smooth, glistening base with thin secretion containing the spirochete. Contact ulcers around the corona are hard. The sclerosis may resemble herpes; an herpetiform chancre becomes indurated in about sixteen days, while simple herpes lasts but three or four days; herpes may be the atrium for syphilitic inoculation. If the sclerosis occurs on a mucous surface there is little elevation; maceration and bleeding readily occur. (*a*) The *genital sclerosis in men* occurs on the urethral orifice; in the fossa navicularis; on the glans, often as an erosion, and difficult to diagnosticate because of the hardness of the glans; on the corona, circular and hard; on the (broken) frenulum or in the sulcus coronarius, usually with much induration, giving the "split pea" appearance, one-half lying on the glans and the other half on the prepuce; on the prepuce, often with phimosis; or on the dorsum. (*b*) The *genital sclerosis in women* occurs mostly on the labia; on the labia majora, as large, readily ulcerating contact ulcers with inflammatory edema; on the labia minora as small, hard and circumscribed nodes; on the prepuce as hard fissures; in the urethral orifice with induration felt from the vagina; on the posterior commissure, as erosions, which are especially prone to infect during coitus; on the hymen relics, as fissures, or large, hard ulcers reaching into the vagina; rarely in the vagina, because of its paucity in glands and its thick epithelium; in the vaginal portion of the cervix, usually in women who have been pregnant, in which location it is difficult to differentiate from erosions. (*c*) The *perigenital sclerosis* occurs on the scrotum, mons veneris or inner thigh. (*d*) *Extragenital scleroses* (5 per cent. of chancres) are especially dangerous in midwives, physicians and wet-nurses. They occur on the scalp, conjunctiva, nose, eyelids, lips, tongue and tonsils (from sexual perversion), cheeks, fingers, arms and mammæ. In rare cases of accidental syphilis the chancre may not be found. Among 9058 extragenital chancres, 1863 occurred from vaccination, 745 from cupping or phlebotomy, 179 from circumcision, and 82 from tattooing (Bulkley).

The primary sclerosis is inoculated from an individual with a chancre or secondary syphilis; Williams reports 41 infections from gummata. *When the sclerosis is recognized, the blood is already infected.*

Histology of Chancre.—The round cells increase the vessel walls tenfold, and the vessel lumen is narrowed from pressure and endothelial proliferation, even to obliteration, when ulceration ensues. Epithelioid and giant cells, and proliferation of the connective tissue in and about the vessels are noted. The Spirochete pallida is present.

Course.—Usually induration disappears in thirty to ninety days, generally with scar formation. In exceptional cases (1) relics of the sclerosis may be found after years (even 50) due to permanent vascular injury; (2) acute or (3) phagedenic inflammation sometimes intervenes. Chancre and chancroid are separate affections—the *dualistic doctrine* of Bassereau, Layroyenne and Ricord.

Diagnosis.

Chancre or Sclerosis—*vs.*—Venereal Ulcer or Chancroid

Incubation: two or three weeks.	Three days.
Form: indurated erosion, ulcer, nodule.	Pustule, ulcer, etc., without induration.
Number: single in 75 per cent., rarely successively inoculated.	Often multiple at beginning or successively inoculated.
Depth: superficial, flat, rarely deep excavations.	Through entire cutis or mucosa; "punched-out" appearance.
Border: gradual descent.	Abrupt, sharp, indented, undermined.
Surface: red, livid.	White, gray.
Induration: marked, cartilaginous, sharply outlined, movable, sometimes thin, lasting weeks or months.	No basal induration; not sharply outlined; merges gradually into adjacent tissue; short duration.
Secretion: scanty, serous; autoinoculation difficult; *spirochetes.*	Rich, purulent; autoinoculation easy; Bacillus cancri mollis, pseudodiphtheria bacilli or staphylococci.
Pain: little.	Much.
Destruction: rarely phagedenic.	More often phagedenic; diffuse.
Reinfection: extremely rare.	Frequent.
Lymph glands: regional, indolent, swelling hard, painless, movable; rarely pus formation.	Often absent or acute inflammatory swelling; often pus formation, capable of autoinoculation.
Nature of the disease: constitutional.	A *local infection.*
Wassermann reaction: positive soon.	Negative.

Mixed infection may occur, whence the now abandoned idea of the unity of all venereal ulcers; the practical lesson from this doctrine is *diagnostic reservation* in doubtful cases.

Lymphangitis syphilitica is marked in 70 per cent. The lymph vessels may become as thick as a lead-pencil, bulging at their valves (bubonulus), hard, painless, and narrowed in their lumen from endothelial proliferation. They are best seen on the dorsum penis. Resorption occurs in three to eight months. Lymphangitic edema is particularly suggestive in women when the initial lesion cannot be found. *Local lymphadenitis* is present in 97 per cent. of cases, appears in three or four weeks, and in six weeks reaches the contralateral inguinal, iliac and lumbar groups, in seven weeks the cubital glands, and just before the secondary eruption, the other glands.

2. Secondary Stage.—Secondary symptoms appear in eight weeks. (*a*) *General infection. Fever* occurs in 20 to 33 per cent. It is far more common in secondary syphilis ("fever of syphilitic invasion") than in primary or tertiary. It often just precedes the eruption, mostly in the pustular variety. It may be continuous, remittent or intermittent, resembling typhoid, malaria or phthisis. Other toxemic symptoms are *anemia, headache, nocturnal bone-aches,* neuralgias, palpitation, night-sweats, polydipsia, digestive disorders, synovitis and icterus; swollen tonsils, spleen or liver, and albuminuria may occur as prodromata.

(*b*) The *exanthem* is *polymorphous,* sharply marked, frequently grouped in a circular arrangement and without pain or itching; it is highly infective and is *copper-colored,* a fact recognized in the fifth century; it appears where other eruptions are rare, as on the forehead or nasolabial fold; recurrent crusts may develop on an infiltrated basis. The most frequent form is the *macule,* next the papular syphilide, and then types resembling pustules, varicella, pemphigus or impetigo. In folds or damp parts of the skin *mucous patches* appear, as at the angles of the mouth, the groin or perineum.

(*c*) The *enanthem* on the mucous membranes is observed on the tongue, lips and cheeks (*mucous patches*), often with pharyngitis.

(*d*) The *lymphatic enlargement* becomes general. "Rheumatism," bone-aches, iritis, etc., are treated under special symptomatology.

Late secondary symptoms occurred after two to thirty years in 5.8 per cent. of Fournier's 19,000 cases ("recurrent syphilis").

3. The Tertiary Stage.—This develops in 10 per cent. of luetics and is characterized by the *gumma* which occurs in many tissues. It is not known what determines tertiary development; in 2396 tertiary lesions, 78 per cent. of the cases were not treated at all, 19 per cent. moderately and only 3 per cent. well treated (Fournier). The topical symptomatology of gumma will be described below. The gumma may be microscopic, or an inch or more in diameter. On section it is usually hard, grayish-yellow, homogeneous, centrally caseous and peripherally fibrous. Tertiary syphilis, particularly of the viscera, may be attended by fever. In 30 per cent. of tertiary syphilis, no evidence of earlier lesions can be found.

Special Symptomatology of Syphilis.—Lues affects different tissues variously: Some organs are very vulnerable, as the liver or brain; other organs are comparatively invulnerable, as the spleen or bone-marrow, in which the parasite may linger for years without causing particular trouble; and in still other tissues, if affected, the parasite dies off rapidly. (These facts have a bearing on the Wassermann reaction, *v. i.*). Generalization of the parasite occurs chiefly in the early septicemia, and its localization in a given tissue, governed perhaps by the strain of parasite, seems to inhibit somewhat its localization in other tissues.

1. The Skin.—The *macular* (*roseolous*) *syphilide* is *the most frequent secondary eruption.* The spots are hyperemic at first and later somewhat infiltrated; the macule develops rapidly, lasts eight to ten days, varies from a lentil to a dime in size, and comes out more clearly by chilling the skin, and after administration of mercury (Herxheimer reaction). It

occurs mostly on the trunk. The face, hands and feet are usually free except in severe cases. It is symmetrical, mostly on the flexor surfaces, more frequently recurrent than any other variety and prognostically favorable.

The *papular syphilide* presents various forms: (*a*) The small miliary lichen syphiliticus usually develops on the forehead, chin, nose, shoulders, buttocks, back and flexor surfaces of the extremities. At first red, it rapidly becomes brownish-red. Fine scales or crusts may form, which on falling, leave a lacquered appearance. Recurrences are infrequent. It must be differentiated from psoriasis, the development of which on the scalp, ear and extensor surfaces is not observed in syphilis, where the scales are smaller, thinner and less glistening. On the palms and soles differentiation may be difficult, but in syphilis the centre of the papule is likely to be sunken. (*b*) The large papular syphilide is often associated with fever, constitutional disturbance or synovitis, and may occur on all parts of the body, as the *corona veneris* on the forehead, around the nose, dorsum of the hand, furrows of the chin and neck or the junction of the skin and mucosa; its polymorphism is pronounced, as psoriasis palmaris, rhagades, onychia or verrucose forms. (*c*) The *condyloma latum* or mucous patch is the papule altered by secretions or excretions, and is found about the genito-anal region, axillæ, etc. *It is the most important syphilide in the propagation of syphilis;* it is flat, dirty, ill-smelling, and produces a spirochete-laden secretion. (*d*) The *pustular* syphilide includes the acne form, the varioloid, varicelliform and impetiginous forms; a brown pigmented scar usually results.

The *general characters* of *secondary eruptions* are: (i) Their circular form and circular groupings; (ii) slow evolution; (iii) symmetry of distribution; (iv) polymorphism; (v) induration; (vi) copper-color; and (vii) diffuseness.

Gummata of the skin are asymmetrically disposed; are cutaneous or subcutaneous, involving deeper structures; are non-infective; are prone to develop at the site of secondary involvements; occur in the skin more than in all other localities combined; are promoted by incomplete mercurial treatment, vicious habits and constitutional diseases; and are seen in 10 per cent. of cases, most frequently in the third year after infection. Small gummata may resorb by fatty degeneration, but the larger ones caseate, ulcerate and leave a kidney-shaped scar with deformation. Gummata are differentiated from malignant ulcerations by the absence of adenopathy, by their slow growth, painlessness and multiplicity, and by the results of therapy. In gumma, ulceration is more rapid than in lupus; it is deeper, less sharply marked, redder and less symmetrical; recurrence after cicatrization is less frequent; the scars are less flat and smooth, and the bacteriological findings of lupus are absent.

Leukoderma syphiliticum is observed after macular or lenticular syphilides as white spots with peripheral brown pigmentation, mostly on the face and neck; it occurs in 4 per cent. of syphilitic men and 45 per cent. of syphilitic women and fades in a few years.

The *hair* falls out except from the crown of the head, during the secondary stage, proportionately to the intensity of the eruption. It is greater

in poorly nourished cases, and its regrowth is usually good. In the bald areas, there are no broken hairs.

Onychia syphilitica occurs particularly in women. The nails may hypertrophy, fall out or fail to grow. Peri- or paronychia rarely causes pain, ulceration or suppuration.

2. Lymph Glands.—The inguinal glands are first enlarged because they are nearest to the most common genital chancre. Adenopathy of any particular group is not pathognomonic of syphilis. The posterior cervical glands are involved more than the anterior. Next in frequency are the axillary, epitrochlear and pectoral. Dietrich observed that 99 per cent. of healthy individuals have palpable glands. Inflammation, pain and suppuration, from mixed infection occur in but 3 per cent. In extragenital sclerosis, other glands are enlarged first, *e. g.*, the anterior cervical, from chancre of the lips. The glands are freely movable and are not usually larger than a hazel-nut, but occasionally become massive, resembling Hodgkin's disease.

3. Gastro-intestinal Tract.—On mucous membranes the eruption (enanthem) is less frequent and is less polymorphous than the exanthem, with which it is synchronous.

Mouth.—This is *more involved than any other part except the skin* and recurrence is very frequent. (*a*) *Erythema* may occur as diffuse or circumscribed, dark red maculæ, especially on the cheek, pharynx, uvula, palate and swollen tonsils—the *acute syphilitic angina;* it ceases sharply at the border of the soft and hard palate. Infiltration or fissures, with a slightly granulated and vesicular appearance may develop. Erosions may last long in users of tobacco and alcohol.

(*b*) *Mucous patches,* which are papules modified by moisture, develop with the exanthem, mostly on the tonsils, cheeks and lips. They are elliptical or irregular, flat or slightly elevated, circumscribed patches, of variable color. On the lips they may measure 6 to 17 millimeters. The course is usually chronic; the patches swell, become granular, ecchymotic and often ulcerated. On the tonsils they may simulate diphtheritic patches, and in smokers a cure cannot be effected until the habit is stopped. They are the *most important syphilide because so highly contagious.*

(*c*) *Chronic angina* is of a granular appearance, accompanied by hyperemia, edema and erosion, a gray coating on the swollen tonsils, and the appearance of pharyngitis granulosa.

(*d*) *Pachydermia, ichthyosis* and *leukoplakia* occur on the mucosa of the cheeks, and on the angles of the mouth, lips and tongue, but are not always syphilitic. They are swellings with fissures, are gray in color, irregular, vary in shape and size and are thick and scar-like. They occur especially in users of tobacco and alcohol. Leukoplakia in tobacco users usually occurs on the tongue, lip or palate, while in syphilis it is mostly on the cheek. Psoriasis linguæ presents elliptical, red, flat spots, with thread-like coating; fissures are frequent on the edges of the tongue, from the use of tobacco and from bad teeth.

(*e*) *Gummata* of the oral cavity occur without glandular swelling. Gummata of the tongue are most frequent, occurring (*a*) as a diffuse

glossitis with later cirrhotic shrinkage or (*b*) as circumscribed nodes or ulceration, without pain or adenopathy. Gummata of the palate or pharynx are mostly multiple. They are frequently diffuse, deep or phagedenic; they destroy the uvula, produce adhesions, obliterate the posterior nares or Eustachian tube, stenose the pharynx or draw the tongue back, stenosing the respiratory passages or reaching the spine or brain. In the tonsils they may erode the carotid or palatine artery. Differentiation is required from tuberculosis and epithelioma in which the glands are usually involved. The diagnosis *ex juvantibus* and Wassermann test are important.

Esophagus.—*Strictures* result from deep gummata, ulcerations in the larynx, or extension from the bronchial glands. Differentiation is required from tumor of the mediastinum, aneurysm, carcinoma, round ulcer and cardiospasm. Diverticula may result.

Stomach. — (*a*) *Acute catarrhal gastritis* in the secondary stage is usually toxemic—rarely organic. (*b*) *Chronic gastritis* is more common, in part explaining the anemia and malnutrition and differing from vulgar gastritis only in the influence of treatment. Amyloid and glandular wasting are infrequent. (*c*) *Ulcers* of the stomach may result from syphilitic arteritis; Garré collected 30 cases. (*d*) *Gummata* are rare. They begin in the submucosa, are usually multiple, leave radiate scars and differ from the ordinary round ulcer in that they are not funnel-shaped, have undermined edges and are broader at the base. (*e*) *Hemorrhages* from the stomach are most rare and Hayem's case is almost unique. They may result from cardiac, splenic, hepatic or renal lesions.

Intestines.—Little is known of small intestine syphilis in the adult. (*a*) *Acute catarrhal enteritis* may *induce icterus;* it resists treatment other than specific. (*b*) *Chronic enteritis* in the syphilitic newborn, is manifested by the viscid meconium. Obstinate diarrhea and stenosis may follow. (*c*) *Ulceration* may result from enteritis in secondary syphilis or from gummatous, diphtheroid and amyloid changes in the tertiary stage. Meschede collected 54 cases of ulcer in the small intestines. They occur around the axis of the intestine and are characterized by spirochetes and productive inflammation which prevents perforation but leads to stenosis. (*d*) Intestinal *amyloid disease* is associated with amyloid liver, spleen and kidneys. The gut is pale, smooth, waxy, thick and rigid and there are obstinate diarrhea, stinking stools and hydrops. It is most frequent in the small intestine and is essentially a vascular change. Amyloidosis in general occurs in 67 per cent. of tuberculosis and in 21 per cent. of syphilis.

Rectum.—Ulceration may occur in papules or fissures near the anus. Rectal disease is usually secondary by contiguity. (*a*) Irritative *proctitis* is usually chronic, attended by evacuations of pus, itching, tenesmus and external excoriations. (*b*) *Syphilitic ulcers* are tertiary. They occur high up, extend to the colon and often produce stenosis. Perforation is rare. The periproctal tissues are invaded. Fistulæ may develop, but the symptoms are less marked than in cancer or tuberculosis. The feces are covered with mucus and blood. Diarrhea from catarrh of the colon,

myositis of the sphincter, involuntary evacuations, burrowing of pus and sepsis necessitate a long course. Tuberculous ulcers rarely occur in the rectum alone, and are marked by the tubercle bacillus. In dysentery the involvement is higher up, colic is more frequent, diarrhea is more profuse, and the resulting stenosis is higher in the intestine. In carcinoma, age, cachexia and local appearances are suggestive criteria; the stricture is higher and adhesions are more common.

Pancreas.—Acute syphilitic pancreatitis, interstitial inflammation with cirrhosis (rarer in acquired than in hereditary syphilis) and gumma are recorded. The *peritoneum* sometimes participates in syphilitic disease of the organs it envelops, *e. g.*, perisplenitis, perihepatitis, etc.; it is extremely rare as an independent gummatous peritonitis.

4. Spleen.—(*a*) *Acute splenic tumor* occurs in 31 per cent. of acquired and 61 per cent. of congenital syphilis. (*b*) *Interstitial splenitis* occurs chiefly with syphilitic liver, marked by increased size, hardness and pain from perisplenitis. (*c*) *Gummatous splenitis* is very rare. (*d*) *Amyloidosis* occurs very frequently as the "sago" spleen, which is not necessarily very much enlarged; or as the diffuse amyloid spleen, which causes considerable swelling.

5. Liver.—Syphilis of the liver occurs pathologically in two main forms, interstitial and gummatous, which are often associated with perihepatitis, amyloid or fatty change, and sometimes with parenchymatous changes, as acute yellow atrophy. (*a*) *Interstitial hepatitis* (syphilitic cirrhosis) is twice as frequent in congenital as in acquired syphilis. The volume of the liver is increased in children, and sometimes decreased in adults. The surface is uneven, with furrows or nodules; the edge is thin, indented and sharp, whereas in other cirrhoses it is rounded. Perihepatic adhesions to the colon or abdominal wall are frequent, and clinically evidenced by lessened respiratory excursion. Connective-tissue forms in Glisson's capsule, follows the branches of the portal vein into the liver (peripylephlebitis), and shows as bands of grayish tissue. The liver cells are degenerated or destroyed by fibrous tissue or obliterating endarteritis. The atrophy is most marked anteriorly and in the left lobe. The connective tissue is not only perilobular, but reaches more into the lobules than in alcoholic cirrhosis. In acquired syphilis the liver is not equally or completely diseased, whence protruding granules of sound tissue are noted. New bloodvessels from the hepatic artery develop in the connective tissue and thrombosis may occur in the portal vein and its radicles (pylephlebitis).

Symptoms.—The enlargement is at first insidious—without pain or ascites. The abdominal veins may be distended; the urine is often dark and albuminuric, possibly icteric. The splenic tumor results from hyperplasia, stasis or amyloid degeneration. Ascites is less frequent and less early than in alcoholic cirrhosis. Icterus develops in 33 per cent. More bile is present in the stools than in ordinary cirrhosis. Gastrointestinal dyspepsia and vomiting of blood are not infrequent. This form develops more often in men; is promoted by alcoholism; runs a chronic course; and its prognosis is better than in non-syphilitic cirrhosis, since relative, even spontaneous recovery is possible. When the liver

shrinks death results from intercurrent pneumonia, cholemia or hemorrhagic diathesis.

(*b*) *Gummatous hepatitis* (syphilitic hepatitis *par excellence*) is more frequent than the first form, and occurs oftener in acquired than in congenital syphilis. The gummata are either miliary, or large nodes which may fill the abdomen; they vary from 1 to 50 and may be superficial or deep. The surface of the liver is smooth, warty, or adherent to the diaphragm or colon. The liver is distorted, mammillated or *lobulated* (*hepar lobatum*). Deep, irregular, radiating *furrows* are frequent, resulting from cicatrized gummata, affecting either lobe and mostly on the anterior surface near the suspensory ligament or on the edge. In congenital syphilis gummata are often observed at the hilum, in the wall of the portal vein or near the bile vessels.

Symptoms.—Gummata are frequently latent, *i. e.*, are discovered only at postmortem. Considerable *pain* occurs in the shoulder, as well as pain on motion or *tenderness* on pressure in the epigastrium or hypochondrium. Pain is due mostly to perihepatitis; a friction-rub is heard when the inflammation is fresh; adhesions and loss of respiratory excursion result, in older cases. Icterus is infrequent and results from gummatous compression of the bile ducts, perihepatitis or cicatrices. The liver usually shows *furrows* and *nodules*. *Early enlargement* is more frequent than in alcoholic cirrhosis; ultimate shrinkage has been observed. Ascites is rare except from syphilitic cirrhosis, amyloid liver or cardiac insufficiency, and is usually terminal and often associated with albuminuria. Splenic tumor is infrequent, except from gummata or amyloid degeneration in the spleen, coincident liver cirrhosis or gummata at the porta hepatis. Hemorrhages and inflammations of the serous membranes are not infrequent.

Diagnosis.—This depends upon other evidences of syphilis; it is often difficult. Syphilis of the liver may remain stationary for a time and in general a *longer course* is expected than in alcoholic cirrhosis. In thirty instances of apparent cirrhosis, the Wassermann reaction was positive in twenty-two. Gummata and cancer may be confused; age is only a relative criterion, yet most cases of liver syphilis occur under the fortieth year; ascites, icterus, enlarged liver and cachexia may occur in both diseases, occasioning doubt and confusion. The nodules of syphilis are usually small but may be larger than those of cancer. Rapid growth is an indication of cancer; more constant size, of syphilis. Albuminuria and splenic tumor are more common in syphilitic liver; in 66 per cent. of hepatic syphilis, occult bleeding and marked gastro-intestinal symptoms may simulate malignancy. The safest rule is always to think of syphilis when cancer seems the obvious diagnosis. The lungs are compressed in cancer more than they are in syphilis. The smooth edge of the liver, signs of fresh hepatitis, and lack of respiratory excursion, indicate syphilis. Riedel found similar symptoms in syphilis and *gallstones*, such as pain, fever and tenderness. Fever is not uncommon in hepatic syphilis, which may then be easily confused with *liver abscess* (*q. v.*), malaria, tuberculosis, *typhoid* or *septicemia:* there may be pain, tenderness, rigors, sweats and leukocytosis, which, with the fever, readily

lead to diagnostic errors; the Wassermann test is invaluable. (See DIFFERENTIAL TABLE OF DISEASES OF THE LIVER.)

Amyloid liver (*q. v.*) is most frequent in the tertiary or quaternary stage of acquired syphilis; it is rare in the liver alone.

Syphilitic *pylethrombosis* (see PYLEPHLEBITIS).

Icterus is attributed to swelling of the portal lymphatics, early hepatitis, cholangitis, injury to the liver cells or to gastroduodenal catarrh.

Devic and Bériel summarized 21 instances of *hepatic hemorrhages*, due to infarction or luetic endarteritis.

6. KIDNEYS.—(*a*) *Albuminuria* occurs in recent syphilis from toxemia or in the later ulceration from vascular changes. It is usually transitory and nephritis rarely follows it, but the later in the disease that albuminuria appears, the poorer is the prognosis. Energetic (not moderate) mercurialization may produce albuminuria and cylindruria. (*b*) Paroxysmal *hemoglobinuria* occurs relatively often after mercurial therapy. (*c*) *Acute parenchymatous nephritis* is focal and cortical. It is early, toxemic and rare. The albuminuria is intense. When it develops late from ulceration, the prognosis is unfavorable; anemia, vomiting or other symptoms of acute nephritis may be present, but uremia is rare, since it is a focal disease. (*d*) *Chronic parenchymatous nephritis* may follow syphilis (0.5 per cent.). (*e*) *Chronic interstitial nephritis* differs from the ordinary form in its disseminated focal involvement, which leaves scars. Polyuria is rarely great, casts and albumin are present in greater amount, and the course is more rapid than the ordinary course of three to seven years, because of marantic, amyloid and other visceral changes. It occurs at an early age when arteriosclerosis is rare. There is said to be no cardiac hypertrophy. (*f*) Miliary or large *gummata* are found in the cortex chiefly, usually multiple and unilateral. They coexist with amyloid kidney and chronic parenchymatous nephritis. The symptoms are indeterminate. (*g*) *Amyloid kidney*, *q. v.*, under diseases of the kidney. (*h*) Syphilis is an occasional etiological factor in *glycosuria*. The *suprarenals* are rarely involved, except in congenital syphilis; fatty degeneration, gummata, inflammation and amyloid changes in the Malpighian bodies have been recorded—in Addison's disease.

7. CIRCULATION.—HEART.—(*a*) *Syphilitic myocarditis* originates in *coronary arteritis;* interstitial foci occur as infiltration along the small vessels, especially in congenital syphilis, into the intermuscular septa, and areas of myocardial, fatty degeneration, with spirochetes. It involves the front or apex of the left ventricle, or the interventricular septum. In congenital syphilis it occurs in the right more than in the left heart. Resulting changes are cardiac hypertrophy, aneurysm of the heart, Dittrich's heart stenosis (conus stenosis) or amyloid change. Only 97 (62 perfectly clear) cases of *myocardial gummata* are recorded (Stockmann, Goldfrank). Gummata may attain great dimensions in the interventricular septum. The disease is often latent until, in 50 per cent. of the cases, *sudden death* results, largely in persons between thirty and forty years of age; myocardial alterations often occur in the secondary stage. The heart action may be incompetent or irregular, and there

may be dyspnea, angina pectoris, heart-rupture or cerebral embolism. Hydrops is rare.

(*b*) Syphilitic *pericarditis* is invariably a complication of myocardial syphilis, occurring over the large vessels or anterior surface of the heart. The symptoms are those of disordered compensation or myocarditis, its invariable associate.

(*c*) *Syphilitic endocarditis* may cause valvular disease (*v. i.*, Aorta). Endocardial gummata may occur.

Bloodvessels.—Endarteritis syphilitica, known to Morgagni and Paré, causes wide-spread, often irreparable or fatal complications. (*a*) The usual *fibrous* form is the *obliterating endarteritis* of Heubner (see page 211); the wall is thick and opaque, the intima and elastic tissue hyperplastic and the lumen eccentric. When the muscular coat is not involved, thromboses occur; when both the muscular and elastic coats are diseased, aneurysms result. It may begin one year after infection. (*b*) Gummatous arteritis or periarteritis is very rare. (*c*) General arteriosclerosis involves the vessels more widely than does the syphilitic arteritis which is largely localized in the aorta, coronary and brain vessels and exceptionally in the other vessels.

Aorta.—Syphilitic aortitis is oftenest seen in the ascending aorta, its arch and thoracic segment, and the lower part of the abdominal aorta. The early stages show gray, gelatinous patches of productive cellular inflammation and spirochetes in the intima; older foci are grayish-white, round or oval, elevated and stellate plaques. The process sometimes becomes deep and calcification occurs in the intima with thrombosis. It may involve the aortic valves. Three-quarters of cases of pure aortic insufficiency (*q. v.*) result from luetic aortitis, and two-thirds react to Wassermann's test. (See Aneurysm.) Its origin may be (*a*) in the intima, (*b*) in the adventitia and media together (mesaortitis) or (*c*) in the intima and adventitia (vasa vasorum). It ultimately involves all the walls in varying degrees. The resulting loss of elasticity and resistance promotes aneurysm. Symptoms of aortitis, as precordial pain, paroxysmal dyspnea or substernal dulness may begin soon after the secondary stage, to reappear after one or two decades as typical aneurysmal signs. The author believes 90 per cent. of aneurysms are syphilitic. Syphilitic aneurysms may be single or multiple and usually occur in the arch, and sometimes also in the abdominal aorta or its branches. Aortitis occurs in 98 per cent. of paretics, and in 50 per cent. of syphilitics. Only 6 cases of gummata are on record.

Pulmonary Artery.—Occlusion of the pulmonary trunk, its compression by scars and valvular gummata are very rare. Syphilitic aneurysm of the pulmonary artery is not known.

Vein Syphilis.—Thirty-three cases of phlebitis were collected by Roussy. Periphlebitis originates in the adventitia, especially in hereditary syphilis. Gummata exist in the veins or involve them secondarily.

Blood.—Oligocythemia and oligochromemia occur most markedly when the glands are widely involved. The anemia disappears under administration of mercury, to reappear if too much be given. A count as low as 1,700,000 may be observed. The hemoglobin is reduced 15

to 30 per cent. The "Justus test," based on a 10 to 20 per cent. reduction in the hemoglobin after a large inunction or injection of mercury, in the florid secondary stage, is suggestive of syphilis. The white cells are increased, especially the lymphocytes; the eosinophiles are increased but not constantly. Myelocytosis may develop in the tertiary stage. The blood contains the *spirochetes* and *by way of the blood, syphilis becomes a sepsis.* The *Wassermann test* (*v.* Diagnosis).

8. Respiratory Tract.—*Nose.*—(*a*) An *initial nasal lesion* is described (only 95 cases recorded). (*b*) *Acute rhinitis* is much rarer in adults than in the newborn; it is characterized by its persistence, erythematous papular foci, hemorrhagic erosions and fetor. (*c*) *Gummata* usually develop late, though they have been seen in the seventh month; they begin in the floor and septum, periosteum, bone and secondarily in the cartilage. The diffuse necrosis involves the cartilaginous and membranous septum and cartilages of the alæ; sequestra may be blown from the nose. Septal perforation may develop and a long course is usual, with discharge of pus and crusts; the bone is often denuded and may crepitate on palpation. The deformed *"saddle-back" nose* is due to loss of the triangular cartilage and vomer. Differentiation must be considered from noma, tuberculosis, carcinoma and the perforating ulcer of Zuckerkandl (beginning as a hemorrhage and due to streptococcic or staphylococcic infection). The diagnosis is best made by Wassermann's test and *ex juvantibus.*

Larynx.—(*a*) *Catarrhal laryngitis* occurs with the eruption, with redness, swelling, coughing and hoarseness. (*b*) *Papular laryngitis* is often associated with ulceration, infiltration and nodes. The epiglottis is involved. Stenosis of the glottis results if ulcers invade the submucous tissue. (*c*) *Gummata* of the larynx are usually multiple, and vary from the miliary size to that of a pigeon's egg; sometimes a diffuse infiltration occurs. They may produce dysphonia, dysphagia, dyspnea, hemorrhage or suffocation. They always leave *stenosis* or deformity.

Diagnosis.—In cancer the glands are usually enlarged, the growth is harder than in syphilis and the microscopic examination may be final. Syphilis and tuberculosis may occur together; the syphilitic ulcer usually begins *near the epiglottis,* is rounder and larger and has sharper edges, a redder border and a whitish-yellow coating of its deeper base, while the tuberculous ulcer is less red, less infiltrated and less deep.

(*d*) *Perichondritis.*—Any cartilage may become excoriated, necrosed and surrounded by edema. Syphilis causes 13 per cent. of cases of *glottis edema;* tuberculosis, 9 per cent. The prognosis is always grave, yet the lower the necrosis, the worse is the prognosis, for deformity and stenosis occur, with dyspnea or dysphagia.

(*e*) *Syphilitic vegetations,* due to irritating secretions, are sessile or pedunculated and cause dysphonia or even suffocation.

Trachea.—The trachea is red, swollen, sometimes ulcerated. Tracheal stenosis, resulting from healing gummata, is usually angular and involves mostly the middle third. The symptoms are inspiratory dyspnea on exertion or on lying down; cyanosis; and a cough resembling pertussis,

with sanguinopurulent sputum, which may contain fragments of cartliage. The diagnosis is made by the laryngoscope.

Bronchi.—(*a*) *Acute bronchitis* may be a secondary symptom.

(*b*) *Chronic bronchitis* accompanies laryngeal, tracheal or bronchial affections. Phthisis may be suggested. The breathing is disproportionately disturbed by irritation of the vagus by enlarged bronchial glands.

(*c*) *Kidney-shaped ulcers* may cause stenosis or perforation into the vessels, mediastinum or esophagus; in Conner's 128 cases of bronchial and tracheal syphilis, 56 per cent. affected the trachea alone; gummata occurred in 15 per cent.; ulcers in 44 per cent., frequently with severe hemorrhages; cicatrices and stenoses in 40; and bronchiectasis in 20, peritracheitis in 6, and pulmonary syphilis in 7 per cent.

Lungs.—Syphilis may simulate pulmonary tuberculosis. Patients with tuberculous signs should be searched for syphilis.

(*a*) In the *white pneumonia* of hereditary syphilis, the lung is firm, heavy, airless and grayish-white, the chief changes being infiltrated alveolar walls, desquamated, swollen epithelium and spirochetes in the air cells and miliary foci about the arteries.

(*b*) *Diffuse infiltration* differs from tuberculosis in that it rarely occurs in the apex; in Grandidier's 30 cases the right middle lobe was affected in 27. The lung is red, hard, large, airless and smooth on section and later becomes gray, uneven and nodulated. Connective-tissue infiltration occurs about the vessels, impinging upon the air cells. Necrosis, fatty change, caseation and later, cavities may result from vascular occlusion. Disproportionate dyspnea is present. Temperature may occur with or without ulceration.

(*c*) *Gummatous pneumonia* may occur alone or with diffuse infiltration. Gummata develop largely in the lower lobes near the hilum. Differentiation from tuberculosis is often difficult and both lesions may occur together. Dulness and other signs of consolidation or cavity formation often result. The sputum is mucous, purulent, profuse, blood-tinged, stinking, and contains masses of tissue but no tubercle bacilli. The course is usually chronic, and advanced cases may heal with appropriate treatment; Brambilla (1777) described a supposedly phthisical patient, who, by mistake, took mercury and recovered. Tuberculosis occurs more frequently in the upper, syphilis more often in the lower or middle lobes. Rise of temperature is more frequent in tuberculosis. In syphilis, actual hemorrhage is rare, as the vessels are obliterated, and night sweats are infrequent.

(*d*) In *syphilitic interstitial pneumonia* the lung may be lobulated (pulmolobatus) like the lobulated liver; marked induration dilates or distorts the bronchi; diagnosis is impossible.

(*e*) Syphilitic pleura disease occurs chiefly with lung lesions.

9. Nervous System.—Syphilis of the nervous system occurs eight times more frequently in men, mostly between the years of twenty-five and forty, and after trauma; in brain workers or those debilitated by worry or excesses; or in children as perhaps the first symptom of parental disease.

In 20 per cent. of cases there is no history of chancre. It occurs where

the secondaries have been light and ineffectually treated. Syphilis occurs in the brain in 16.5 per cent. of syphilitics and more often than in other viscera, excepting the liver.

Fifty per cent. of brain syphilis occurs within three years after infection. Formerly described as occurring many years after infection, cerebrospinal syphilis may develop as early as five weeks after infection (especially in those advanced in years). Nervous symptoms in the secondary stage as headache, neuralgia, increased reflexes or fleeting paralyses, are partly toxemic but largely anatomic, as is demonstrated by the pleocytosis (over ten cells in the cell count), lymphocytosis, Wassermann and globulin reactions of the cerebrospinal fluid.

In general, the symptoms are marked by: (*a*) *variability* and inconsistency, due to alternate regression and recurrence in the lesions; (*b*) *incompleteness*, as partial paralysis or disturbance of consciousness; (*c*) the symptoms are partly tumor-like, partly vascular, or partly inflammatory; they are partly meningeal, partly basal or cortical, and rarely occur in the centre of the brain except from secondary vessel lesions and gummata. Syphilis produces no symptom not produced by other disease.

Types.—1. *Syphilitic arterial disease* is the *most frequent form*, and aside from neuritis paralysis, *the most frequent cause of syphilitic paralysis.* As prodromal disturbances, headache is usual; vomiting, vertigo, psychic changes, convulsions, hemianopsia and aphasia may occur; choked disk is rare. *Thrombosis and obliteration of the vessels* are gradual in onset and intermittent—*e. g.*, involving the leg and, in a few hours or days, the arm—and the multiple softening in 95 per cent. of cases occurs in the distribution of the Arteria fossæ Sylvii.

Course.—The first attacks are mild; the later are more severe, and occur with somnolence, bilateral, alternating or crossed paralysis, general or rarely Jacksonian convulsions, or with partial aphasia, mental changes or progressive bulbar phenomena.

In diagnosis the ordinary *arteriosclerosis* in over 90 per cent. of cases occurs in late life, its progress is slower, the changes are more disseminated, all coats of the vessels are involved, its fatty and calcareous changes lead more often to widening than to obliteration, and hemorrhage is more characteristic than softening; whereas in *syphilitic endarteritis* the onset is more acute and headache more conspicuous, encephalomalacia occurs at a younger age (see Brain Embolism), the changes affect chiefly the aorta and cerebral vessels, the intima is principally involved, calcareous and fatty changes are rare and obliteration is usual; the hemiplegia occurs by "instalments," associated with meningitis, apathy, dementia, somnolence or delirium; and finally the symptoms are more diffuse. The Wassermann test and spinal puncture are final. Without treatment, the average duration is one to three months.

Multiple *aneurysms* of the basilar, Sylvian and carotid trunks are generally *tertiary*, but occasionally secondary; their rupture produces symptoms of apoplexy or meningeal hemorrhage. Otherwise hemorrhage is rare in brain syphilis.

While *softening is usually ischemic* (non-inflammatory), *encephalitis syphilitica* may rarely cause disseminated softening or indurated sclerosis.

2. *Basal gummatous meningitis,* the best-known type, and the next most frequent form, begins in the dura, especially about the optic chiasm, interpeduncular spaces or cavernous sinus; less frequently in the fossa of Sylvius and the cortex; pathologically it consists of fibrinous exudate, granulation tissue, gummata and cicatrices.

General Symptoms.—Headache is the most important and early symptom (in 75 per cent.). It is paroxysmal, is increased at night, sharp, boring and deep-seated. Other symptoms are *projectile vomiting,* vertigo, *somnolence,* semi-intoxication, motiveless activity, nocturnal automatism or dementia, alternating with delirium, epileptic attacks or paralytic seizures; the symptoms may resemble those of uremia, meningitis or typhoid. Between attacks, cerebration may be normal. *Epilepsy* may be typical or unilateral, frequent or violent. Polydipsia, diabetes insipidus from disease in the third ventricle, or diabetes mellitus, of which Oppenheim collected 20 cases, may occur. Fever may be present.

The *cranial nerves,* especially the second and third are often affected. Involvement of successive branches is most characteristic (85 per cent.); *80 to 90 per cent. of nerve involvements result from syphilis of the brain, tabes* or less frequently, general paralysis and brain tumor. (*a*) The third nerve is involved in 65 per cent. of these cases—"the sign of syphilis." (*b*) The optic nerve is diseased anatomically in 82 per cent. and clinically in 40 per cent. of cases; choked disk (10 per cent.) is almost always bilateral (see Plate IV). Simple atrophy, with complete blindness (in 6.6 per cent.), is most frequent in tabes and next most frequent in brain syphilis and general paralysis. Amaurosis is common and often unilateral. Hemianopsia is homonymous. Temporal heteronymous hemianopsia is very often syphilitic; the nasal form is rarely so. Eye changes, due to meningitis, bone disease or gumma are susceptible to treatment. (*c*) The fourth, sixth and fifth nerves are next in frequency of involvement. The fifth is unilaterally affected, and more in its sensory than motor branches. The olfactory nerve is rarely involved. The facial nerve may be paralyzed, nearly always unilaterally and peripherally. The eighth nerve is sometimes involved, and the resulting Ménière complex is ominous. The vagus or hypoglossus may be affected.

With the above changes (i) *gummata may grow into the pons,* crus or medulla, and cause hemiplegia and crossed paralyses, as hemiplegia plus oculomotor paralysis (Weber's paralysis); plus facial paralysis (Gubler's paralysis) or plus abducens and trigeminus paralysis (Leyden's paralysis); or (ii) *arterial phenomena are often noted,* syncope, apoplectiform attacks, encephalomalacia, hemiplegia, hemianesthesia and hemianopsia, usually as later symptoms. Syphilitic meningitis is typically subacute with remissions and exacerbations, and lasts rarely more than half a year.

Differential Diagnosis.—Carcinoma and sarcoma at the base of the brain, involving the meninges, run a continuous progressive course, with definite localization, while in syphilis the course is intermittent, localization varies, and vascular changes are often marked. In *tuberculous meningitis* there is less development of connective tissue, vascular changes are rarer, the nerves less frequently involved, the course more

acute, febrile and progressive, and remissions less frequent and less marked. Mental obscurity is more marked, sudden and stationary. The initial irritation is followed by later paralysis, while paralysis may occur at once in syphilis. Tuberculous meningitis usually occurs before, and syphilis after, twenty years of age. Other syphilitic or tuberculous foci, the Wassermann reaction, lumbar puncture and the results of therapy determine the differentiation. Spirochetes have been found directly in the cerebrospinal fluid only five times. Differentiation from Quincke's *serous meningitis* is not difficult. The *multiple nerve root affection* of Kahler, involving the seventh and third cerebral and the cervical and dorsal nerve roots causes neuralgia and paralysis. *Periodic paralysis* of the third nerve is rhythmic in its attacks, usually involves the entire nerve, occurs in young children especially, and with attacks of migraine.

3. *Syphilis of the convexity* is a meningo-encephalitis, with circumscribed symptoms like cortical tumor or with diffuse manifestations.

General Symptoms.—Headache is usually the first and most common manifestation. *Focal Jacksonian attacks* occur with mono- or hemiplegia, develop by starts or "instalments" and occur without an aura. *Ninety per cent. of convulsions in those over thirty years of age, not uremic or alcoholic, are syphilitic* (Fournier). The symptoms are more diffuse than in vulgar epilepsy. The convulsions may number even 400 in twenty-four hours. Aphasia is frequent, usually of the transitory motor type and occurs less often from gumma than from vascular disease. Diffuse meningo-encephalitis may resemble an acute psychosis or dementia. Convexity syphilis recovers more frequently than other forms.

4. *Gummata,* the least frequent variety, occur usually in the meninges, sometimes in the central ganglia, cerebellum, pon or crus. The symptoms are those of brain tumor, except that there is often regression, either spontaneous or therapeutic. *Cortical gummata,* producing cortical epilepsy and monoplegia, require differentiation from *cortical tumor* (*q. v.*) in which the pressure symptoms, and slow pulse are more marked; the disk changes follow focal signs and the process advances less by epochs; while in syphilis the pressure symptoms are more diffuse and rapidly extensive; the disk is involved when coincident basal meningitis occurs; cortical paralysis is often associated with Jacksonian epilepsy (*v. s.*); the symptoms are more undulatory, the Wassermann test is positive, and treatment is successful.

5. *Cerebrospinal syphilis* produces suggestive spinal symptoms: (*a*) their asymmetry, (*b*) their less-pronounced character, (*c*) their variability, (*d*) meningeal and nerve-root symptoms, (*e*) the cerebrospinal fluid issues under pressure and exhibits pleocytosis (the normal number of lymphocytes being 4 to 6), Wassermann's reaction and globulin, and (*f*) response to mercury and iodides. Syphilis is never a system disease (Gowers). Syphilis of the cord is six times less frequent than brain syphilis.

The most frequent and chracteristic form is *meningo-encephalomyelitis.* While the symptoms may be strictly spinal, cerebral meningo-enceph-

alitis is frequently found anatomically. Heubner's arteritis is not noted. Brain changes may mask the spinal symptoms. The meninges are most involved in the cervical and upper dorsal regions, as evidenced by pain, stiffness or girdle sensation; while the cord itself is mostly affected in the lower dorsal region, and therefore causes weakness in the legs, paresthesia, sphincter disturbance and decubitus. Intercostal neuralgia, nocturnal pains in the extremities, spastic paraparesis, hemiparaplegia, triplegia, Brown-Séquard's syndrome, increased reflexes and sensory disturbances, are some of the symptoms of cerebrospinal syphilis. Erb laid special stress on the spastic paraplegic type. The so-called syphilitic myelitis is often only softening—*myelomalacia.* Gummata of the cord and perineuritis gummosa of the nerve roots are uncommon.

In cerebrospinal syphilis, tabes and dementia, the lumbar puncture is important (*v. s.*).

Prognosis of Brain Syphilis.—Only half of the cases recover. The later the disease, the worse is the prognosis. The prognosis is more favorable in meningeal, *i. e.*, extracerebral involvement, when nerve symptoms or epilepsy are dominant, or when early and thorough treatment is instituted. The prognosis is less favorable in specific endarteritis, bulbar forms, diffuse involvement, in great psychic alteration and in extragenital infections and hereditary syphilis. Recovery is rarely complete and recurrence is possible.

6. *Syphilis of the peripheral nerves* may cause facial and trigeminal paralysis, even in the secondary stage. The most frequently affected spinal nerves are the occipital and auriculotemporal. But 6 reports of luetic multiple neuritis exist in the literature.

10. Eye and Ear.—Syphilis explains 3 per cent. of all eye diseases and 3 per cent. of all syphilitics have eye disease. The *uveal tract is most involved;* next in frequency come the retina, optic nerve and eye-muscles; the lens suffers only from uveal tract disease. The lids are not often involved (chancre, condyloma and gumma).

Conjunctiva.—Twenty-two scleroses have been observed, as well as a few gummata; catarrh occurs in the secondary stage.

Cornea.—The cornea is frequently involved: (*a*) *As parenchymatous keratitis,* which is mostly observed in *congenital syphilis,* and occurs even in the third generation; in acquired syphilis it occurs in but 2 per cent. of cases. *"Hutchinson's triad"* in congenital syphilis consists of *keratitis, notched teeth* and congenital or early acquired *deafness.* The cornea is diffusely clouded and milky (the ground-glass cornea), and on close examination shows patches or streaks of inflammation; (*b*) as keratitis interstitialis, circumscribed or punctate; (*c*) as gumma, which is infrequent; (*d*) as keratomalacia, observed in the congenital variety.

Iris.—Of all inflammations of the iris, 75 to 90 per cent. are syphilitic. The symptoms are those of other iritides—ciliary injection, discoloration, slow reaction and synechia or occlusion of the pupil. It is plastic, or exudative with tiny papules; iritis with nodules, if not traumatic, is strongly indicative of syphilis. Iritis is usually secondary, rarely tertiary. Without cyclitis it is rare in congenital syphilis. Atropine should be administered early while mercury is being given, for expectant treat-

ment results in severe lesions and recurrence. *Ciliary* involvement is usually secondary, rarely tertiary. Injection and exquisite tenderness are characteristic.

Choroid.—The choroid is involved in the secondary period, perhaps following iritis or cyclitis. Its several forms terminate with the same appearances—exudative choroiditis with small, oval or irregular yellowish, bluish or rose-colored spots. Both the spots and pigment lie behind the retinal vessels, which branch over them in the ophthalmoscopic picture (Plate VI). Disseminated choroiditis consisting of spots of pigment especially in the peripheral eye-ground, usually indicates syphilis. When recurrent, with inflammation of the retina and choroid, blindness may result. The *vitreous humor* is rarely involved without lesions of the uvea. It results in opacities or dust-like bodies; in the absence of myopia it rather indicates syphilis.

Retina.—The retina is involved chiefly in its anterior layers, becoming cloudy about the disk, with opacity of the nerve and blurring of the disk outlines. The course of the arteries is lost or broken and the veins are tortuous and engorged. Most cases of idiopathic retinitis are syphilitic. Nodules, either papules or gummata, constitute the specific retinitis. Recurring retinitis, commencing at the macula, is of great importance. Retinitis with pigment formation occurs mostly in congenital syphilis. The *optic nerve* is usually involved with the retina, and anywhere from its central origin to the disk. Sometimes the change is simple blue atrophy; again the appearance is nearly normal, or again choked disk occurs. The causative lesions are gumma in the brain, syphilitic inflammation of the intracranial tract or basal disease. Neither its syphilitic nature nor its location can be diagnosticated by the ophthalmoscope. Amblyopia or amaurosis may result from transitory, circulatory, or permanent organic changes. Hemianopsia is often central, results from lesion in the optic tract and, in most cases, is transitory.

Eye Muscles.—The eye muscles are involved as frequently as is the iris; more than half the paralyses of the eye muscles are syphilitic. Ocular paralyses may be (*a*) peripheral; (*b*) intracranial as at the base of the brain; or (*c*) central (nuclear, fascicular and cortical). The following lesions occur: syphilitic meningitis, basal gummata, periostitis, cellulitis of the orbit, diffuse arterial disease, nuclear disease, polioencephalitis and gummata of the hemispheres or cortex. Other causes as tabes, general paralysis and tumor, must be considered. Thirty-six to 50 per cent. of nuclear paralyses are syphilitic. Syphilis frequently produces progressive paralysis of all the muscles of both eyes. Total paralysis of the *oculomotor* is rare; if it occurs, the lesion is usually at the base, is rarely orbital, or may be nuclear, especially if one twig after another become involved. If it is partial, it is usually nuclear, rarely neural or muscular. Ptosis is highly suggestive of syphilis. Abducens and trochlear paralysis may occur. Ophthalmoplegia interna, involving the sphincter iridis and the ciliary muscle, produces mydriasis and paresis of accommodation; 36 per cent. of all mydriases are syphilitic and nuclear. Ophthalmoplegia externa may occur with facial paralysis,

involving the orbicularis palpebrarum, or with trigeminal paralysis involving the sensory filaments of the eye.

Orbit.—Periostitis is indicated by deep tenderness over the eye-ball or edge of the orbit; by pain on movement of the eye; more rarely by blindness, exophthalmos and thrombosis. Periosteal gummata are often mistaken for tumors.

Ear.—Four instances of primary sclerosis on the external ear have been noted; 33 per cent. of children with hereditary syphilis are deaf; otosclerosis is specific in 25 and nervous deafness in 50 per cent. Condylomata, mastoid periostitis or gumma, sclerosis or gumma of the Eustachian tube, may be observed.

11. GENITALIA.—Some chancres occur in the *urethra;* gummata have been observed in the urethra and bladder (23 cases).

Penis.—Gummata, at the site of the primary lesion or elsewhere, often are confused with chancre, chancroids or carcinoma.

Testes.—(*a*) *Diffuse interstitial orchitis* occurs with great pyriform swelling and flattening of the epididymis; it is often nodular, due to periorchitis or gummata. Serous effusion and synechia occur. Absorption of this sarcocele syphilitica may result. The connective tissue is increased and the canals are compressed, fatty and infiltrated with round cells (*b*) *Gummatous orchitis* is less frequent, and usually involves one testis. Great symmetrical or irregular swelling may occur, and frequently the entire testis is firm and studded with gummata, single or multiple, hard, irregular, painless nodules. The testes sclerose and atrophy. The symptoms develop gradually or after trauma. The scrotum is infiltrated, nodular and red. Perforation is frequent, with a crater-like opening and irregular edges. Oligospermia, azoöspermia or impotentia does not always follow, because islets of functionating tissue frequently remain; syphilis has been inoculated into rabbits from the sperma. Differentiation is required from gonorrheal orchitis, which is more acute, painful and involves the epididymis first; plastic orchitis, resulting from stricture and traumatic orchitis; also from tuberculous disease, which is often bilateral or occurs in several foci, and frequently involves the cord, vesicles, epididymis and often causes perforation with pain, fever and prostration, in which case tubercle bacilli may be detected. Sarcoma is harder, develops more rapidly, is more nodular, produces lancinating pains and frequently affects the glands, epididymis and cord, which are less often involved in syphilis. Carcinoma, which requires differentiation from interstitial orchitis does not respond to specific treatment.

Vas deferens and prostatic involvements are rare.

Vagina.—Chancres are relatively rare, because of the thick epithelium and the paucity of glands; secondary eruptions are rarely observed. Gummata occur infrequently.

Vulva.—The vulva is very frequently the seat of secondary lesions, upon which tertiary lesions may develop, which may extend to the urethra or bladder. Enormous swelling, resembling blisters, is a common result of lymphatic involvement. Gummata are readily differentiated from carcinoma and tuberculosis.

Cervix.—Chancre occurs in 5 per cent. of cases. Scars may result from gummata, with stenosis and sterility.

Uterus.—Syphilitic *endometritis* is frequent; metrorrhagia may respond only to mercury. Virchow described a papular and a tuberous variety. Endometritis may produce sterility. In clear cases of *metritis* large tumors disappeared under specific therapy. Specific or simple perimetritis is secondary to rectal disease. Placental involvement and abortion are discussed under Congenital Syphilis.

Tubes and Ovaries.—Salpingitis, oöphoritis and gummata have been described.

12. Bones, Joints and Muscles.—*Periostitis* produces pain, tenderness and swelling, which is hard at first, and later is elastic even to fluctuation. It occurs especially on parts subject to trauma, as the head, sternum, ribs, tibia or clavicle. Fugitive pains occur with the secondary eruption, but the pain of periostitis is fixed, rarely migrates, is boring in character, appears at night—*dolores nocturnæ*—and disappears at 1 or 2 o'clock in the morning with sweating, but otherwise resembles the common types of periostitis. In 28 per cent. it occurs in the frontal or temporal bones, and forms circumscribed, smooth, elastic and immovable tumors. On the ribs, it occasions pain on coughing or neuralgia over the sternum. Tibial nodes occur in 16 per cent. of cases. Osteophytes develop. Periosteal changes last for five or six weeks.

Gummata of the bones, the first well-studied form of gummata, largely occur on the exposed parts as: (*a*) Diffuse subperiosteal infiltration, which causes molecular destruction of the bone. Sequestra may form, especially in the femur; osteophytes in the diaphysis sometimes resemble arthritis deformans. (*b*) Circumscribed gummata, the more important form, develop in the periosteum, as elastic, immobile, not especially tender tumors which afterward soften and, perhaps fibrose and calcify. Gummata of the cranium are attended by dull pain, especially at night; grow slowly; are at first soft, and later have a wall-like, hard edge. They rarely ulcerate except from pyogenic infection and often disappear spontaneously, leaving an elevated periphery and a deeper centre. They are often mistaken for supra-orbital neuralgia or migraine. Tuberculous ulcers rarely occur in this location. Meningeal symptoms may develop (perforating type). Gummata *at the base*, where periostitis is infrequent, occur chiefly in the middle fossa and involve the nerves or bloodvessels. Diffuse gummatous infiltration develops very slowly, with little pain, possibly with ulcerative exposure of the bone and is diagnosticated with difficulty. In 1914 Hunt collected 100 instances of luetic *spondylitis;* only 44 per cent. recovered, due to its late recognition; the affection is cervical in 70 per cent., rarely dorsal, very rarely lumbar, and affects the bodies of the vertebræ less than their processes and arches, in contradistinction to tuberculosis. Pain and immobility are observed. The head is held in the hands, if the atlas is involved; death results from ulceration, pressure on the medulla or luxation (10 per cent.). The *humerus*, *radius* and *carpus* are infrequently involved. The phalanges may be involved, usually one finger only, *spina ventosa*, the parts being swollen but soft, with periosteal thickening, shining appearance and rarely pain or ulcera-

tion. The clavicle, from its exposure to trauma, is very frequently involved; the course is slow and often painless. Ulceration of the sternum is frequent; of the ilium and sacrum, rare.

Joints.—(*a*) *Acute synovitis* occurs alone or with periostitis at the time of the eruption; pain alone may be observed or the swelling is polyarticular, with tenderness, nocturnal pain, immobility, temperature, and involvement of the muscles and tendons. The knees are most frequently involved and, according to Fournier, more than three joints are never affected. This luetic pseudorheumatism may last weeks or months and does not involve the heart. In 100 acute arthritides among negro laborers on the Panama Canal, Baetz found 63 per cent. syphilitic and 28 per cent. gonococcic. (*b*) *Chronic synovitis* or arthritis is usually monarticular, indolent, afebrile, painless, and affects chiefly the knee and elbow. (*c*) Joint disease of late syphilis originates in the bone or cartilages (*tumor albus syphilitique*), and affects chiefly the knee, elbow and ankle. The central portion of the cartilage is involved, and not its edge, as is the case in arthritis deformans, probably due to osseous gummata and most frequent in congenital syphilis. The onset is slow, with moderate pain and swelling; no temperature is observed. There may be a close resemblance to tuberculous lesions; tuberculosis is usually inflammatory, incipient syphilitic lesions non-inflammatory; in syphilis, sequestration and ulceration are infrequent; therapy and the seroreaction are the final tests. Syphilis is most prone to attack the periosteum, gonorrhea the joint, and pyogenic organisms the bone. The writer has repeatedly seen lues simulate arthritis deformans.

Muscles.—*Early* involvement may occur from *toxemia* or *myositis*. The latter is a diffuse or circumscribed infiltration, which possibly eventuates in muscular contractures, especially in the arm and finger flexors, sphincter ani, masseters, deltoid and sternomastoid. The course is subacute or chronic, depending on treatment. *Gummata* cause confusion with neoplasms; they may be as large as the fist, single or multiple, usually with a firm connective-tissue capsule; they involve the long muscles of the arms and neck, especially at their attachments. The course is long, and regression may occur with induration and atrophy (arterial disease and central nervous lesions). Myositis ossificans is rare. The *tendons* may be involved, partly from changes in the bones or muscles. (*a*) The early irritative, acute bursitis or tendosynovitis is sometimes confused with rheumatism. It occurs most frequently in sewing women and washerwomen. There is more tension than pain. (*b*) The gummatous form, occurring in the flexor tendons, is characterized by indolence, painlessness and benignancy. Churchman collected 26 cases of bursitis (1876–1908).

13. Mammæ.—(*a*) The diffuse infiltration, *syphilitic mastitis*, with swelling, occurs largely in the secondary stage, and often resorbs spontaneously. (*b*) *Gummatous mastitis* is deep-seated and is not very sensitive. Rapid growth may be observed with central necrosis, fluctuation or external ulceration. It requires differentiation from adenoma, fibroma and carcinoma; it is frequently bilateral, without cachexia or adenopathy, but possibly with gummata elsewhere. (*c*) 206 cases of mammary

chancre are recorded (Dimey). There is a reversion to the abandoned idea that milk may convey infection.

Diagnosis of Syphilis.—Differentiation has been treated under the primary chancre, secondary manifestations and special visceral pathology. The last ten years have brought within our reach special, we may say absolute, diagnostic helps for the careful diagnostician, and yet there is a tendency to leap at once to laboratory diagnosis and to disregard the frequently characteristic clinical physiognomy of the disease.

I. The Treponema.—In the initial induration, the detection of the parasite is invaluable, employing the dark-ground illumination. Doubtless cultural methods soon will be available for practical use.

II. The Wassermann Reaction.—Elaborate studies of this complicated test, discovered in 1906 by Wassermann, Neisser and Bruck, have shown its presence in 85 to 95 per cent. of syphilitics; in the chancre stage, it appears in 90 per cent. usually in six weeks; in the secondary stage, in 95 to 100 per cent. (in 80 per cent. when no outward signs are present); in the tertiary stage, in 96 per cent. (in 57 to 65 per cent. of cases showing no obvious symptoms); in the quaternary stage, in 95 to 100 per cent. of general paresis, in 75 per cent. of tabes and in about 66 per cent. of cases of aortic leakage, aneurysm (95 per cent. of those under forty years of age) and aortitis. In general, Wassermann's 1982 cases of syphilis gave the reaction in 90 per cent. of manifest lues and in 50 per cent. of cases without obvious symptoms. A positive reaction is sometimes encountered in leprosy, frambesia, trypanosomiasis, scarlatina, etc.

The positive reaction is the most common symptom of syphilis, but it is only a symptom and may be lacking; much less significance attaches to a *negative reaction*, which, in general, is encountered in 10 per cent. of cases undoubtedly syphilitic. A positive reaction denotes the survival in the body of some living spirochetes, and 99 per cent. of positive reactions are absolutely diagnostic of lues. Mercury and arsenic, previously administered, vitiate the reaction, as does also alcohol (taken within twenty-four hours of the test). The best results follow the original unmodified Wassermann technique. The practical bearings of the test are immense, *e. g.*, upon the questions of immunity, which is merely latency of the disease, reinfection, familial diseases, heredity, congenital lues, prostitution, marriage, life insurance, and treatment, all of which are discussed later. To mention but a few instances: Letulle found positive reactions in 43 per cent. of chronic obscure diseases and in 25 per cent. of chronic nephritides, Dean in one-sixth of 330 idiots, etc. *A Wassermann on the spinal fluid* may be positive in nervous lues when one on the blood is negative; the former is important diagnostically and also therapeutically, since a test of the spinal fluid should be negative before a case may be declared cured.

III. Noguchi's Luetin Test.—Luetin prepared from strains of spirochetes, sterilized from pure cultures, is injected as a fine emulsion in doses of 0.05 c.c. intradermally. Luetin (the antigen) elicits a cutaneous reaction or allergy comparable to v. Pirquet's test. The positive reaction, obtainable only in syphilis, begins as a small erythematous area

surrounding the point of injection, usually within twenty-four hours. This area may remain without further change for several days or may become papular or pustular, according to the severity of the reaction, which reaches its height in four or five days. In some cases a slight rise in temperature occurs, lasting a day or so, and accompanied by malaise, loss of appetite and diarrhea. The reaction is sometimes torpid, coming on after ten or more days as a pustular eruption. Noguchi concludes: (1) The luetin reaction is specific for syphilis. (2) The reaction is present in the majority of cases of tertiary (100 per cent.), latent (96 per cent.) and hereditary syphilis (96 per cent.). (3) It is less constantly present in secondary untreated and primary cases (50 per cent.). (4) In treated secondary cases the reaction is present in most instances, especially when treated by salvarsan. (5) In general paralysis and tabes dorsalis a positive reaction was obtained in 60 per cent. of cases. (6) In certain cases of tertiary and hereditary syphilis there may be a considerable inflammatory reaction at the site of injection of the control fluid, and as strong as at the luetin inoculation site. The Wassermann reaction is more constantly present than the luetin reaction in primary and secondary syphilis, especially when only a slight amount of treatment or none was given, but the luetin reaction is more constantly present than the Wassermann reaction in cases of tertiary and latent syphilis. Through the treatment the Wassermann and clinical symptoms gradually are made to disappear, while the luetin reaction becomes more distinct.

IV. Diagnosis ex Juvantibus.—*v. i.* Therapy.

Immunity.—Syphilis confers *immunity*, but this is neither absolute nor life-long, because (1) reinfection may occur; of 356 instances in the literature, only 132 stand criticism (F. John, 1909); (2) in hereditary syphilis, a later infection may be acquired (27 cases known); (3) a sound child of syphilitic parentage may acquire syphilis (29 cases reported); and (4) apes have been reinoculated. Gitting's case with a second chancre, occurring while the Wassermann was still positive, may have been a recurrence. In general, immunity means only latency of the luetic virus.[1]

Abnormal Course of Syphilis.—Of special importance is *malignant syphilis* (syphilis precox, acute or galloping syphilis, synonymous with

[1] Levaditi holds that during the period of incubation before the primary sore the parasites invade the body, causing the development of immune bodies, as manifested by a refractory condition of the skin to further infection; this resistance becomes well developed at the onset of secondary lesions. During the secondary stage the lesions are chiefly inflammatory, with evidence of great resistance by the tissues, which suffer little injury, despite the presence of great numbers of parasites. In the tertiary stage, the resistance to infection is highly developed, and the lesions show great tissue destruction produced by comparatively small numbers of parasites.

Levaditi suggests that the resistance of the host to the invading organism developed during the primary stage may hold the parasites in check for a time, until they acquire a certain degree of immunity of their own to the antibodies of the host; they then enter the blood and set up a generalized infection, characteristic of the secondary stage; in reaction to infection, greater quantities of antibodies are formed, suppressing the parasites to a large degree, although not destroying all of them. The tissues themselves may become hypersensitive, *i. e.*, a few spirochetes cause extensive local necrosis, characteristic of gummata. Gummata, then, are produced by parasites, which are refractory to the antibodies of the patient and develop in tissues which have grown sensitized. Iodides operate by rendering the tissues less sensitive or anaphylactic.

early tertiary development). The cause may be increased virulence of the spirochete, or decreased physiological resistance, as in tuberculous, alcoholic and other cachexiæ. Early ulceration, grave general symptoms and hemorrhage are noted.

Prognosis.—On *pregnancy* the disease exerts a malign influence and mercury is less universally successful (*v.* Congenital Syphilis). *Exanthematous diseases* may cause syphilis to disappear temporarily. The effect of coincident measles and smallpox is unfavorable. *Chronic diseases*, as tuberculosis, often induce ulceration and early tertiary symptoms. *Trauma;* in the treatment of fractures and in plastic operations in syphilitics, mercury is indicated. Wounds may become the seat of specific ulceration.

In *life assurance* the outlook is uncertain; Leser's figures indicate that 33 per cent. of luetics ultimately die of tuberculosis, paralysis or aneurysm; other main causes of premature death are cardiac disease, nephritis, dementia, tabes, thrombosis and suicide; the chief contributing causes are poverty, worry, overwork and alcoholism.

Congenital Syphilis.—Syphilis descends only to the first generation; among the 115 cases recorded in the second generation, many are doubtful.

It was formerly held that a child might acquire syphilis from the father, from the mother, or that mother and child might be infected simultaneously. Hutchinson still adheres to the possibility of paternal lues transmitting the disease. The Wassermann reaction and the presence of spirochetes have revolutionized our conceptions of congenital syphilis. According to Colles's (or Beaume's) law the syphilitic child is held to make the mother immune to infection; she cannot be infected from her infected child; and can nurse her syphilitic child without danger. In such cases, Fournier held that the mother is already syphilitic, and in 381 cases, 76 per cent. gave a positive Wassermann reaction, whence it is probable that all such mothers are luetic, the syphilis being latent. Profeta's law held that (apparently) sound children of syphilitic parentage possess an immunity against syphilitic infection; Wassermann's reaction proves that these children have syphilis.

The more recent the parental syphilis, the greater the probability of fetal infection; in tertiary syphilis of the parents, the child may escape. Thus the first product of conception may be aborted, the second macerated fetus prematurely delivered, the next die at birth or shortly afterward, the next viable though infected, and finally, as the toxemia wears off, even sound children. Only 28 per cent. of conceptions in syphilitic mothers result in full-term infants, and of these 85 per cent. die within the first year. Women who habitually abort, show the Wassermann reaction.

It is possible that excessive emphasis has been placed upon the significance of abortion *per se;* infectious abortion is common among mares and cows and is due to a small bacillus (Bang, 1895). Infection from the mother is carried to the fetus by way of the placenta, in the fetal portion of which, and usually also in the umbilical cord, spirochetes are found, although but seldom in the maternal portion of the placenta. Significant

also is the *heavy placenta* whose weight compared with that of the syphilitic child is 1 to 5, 4 or 3 (normally 1 to 6). In the placental villi end- and periarteritis occur, which may cause, by vascular occlusion, placental necrosis and adherence. Similar changes in the umbilical vessels may destroy the child. When the macerated fetus is examined, the spirochete is found in 84 per cent., most often in the adrenals, next in the liver and lungs in the vicinity of the vessels, and often in the heart alone. The ovary, testicle, and epididymis contain the spirochete. In living children the spirochete was found in the vesicles in cases of pemphigus. A difference exists between the congenital syphilis with signs at birth, and the hereditary type, which develops later (possibly very late, syphilis hereditaria tarda). Even when specific lesions are absent, pathological cell degeneration, rhachitis, slow intelligence, neuroses, psychoses and a high percentage of mortality to all diseases may result.

Symptomatology.—The primary, secondary and tertiary stages are not discrete and lymphatic infection does not precede that of the entire organism. Secondary and tertiary manifestations may occur synchronously. The symptoms are recognizable at birth or after a few days or weeks, and almost always within the first three months.

Appearance.—The *atrophic child* presents a rather characteristic appearance, with relaxed yellowish-gray skin, poorly developed hair, bent or undeveloped nails (onychia) and "the little old man" appearance described by Trousseau, but not pathognomonic of lues. The palms and soles are lacquered, the voice and muscles are weak and the child does not nurse well. Such children, especially when several are born in succession, point to syphilis.

Pathognomonic Symptoms.—The *exanthem* is *polymorphous*, but varieties, often severe, occur which are absent or infrequent in the adult, such as pemphigus, hemorrhages, furuncles and *diffuse infiltration of the skin*. The maculæ are dirty, brown-red, often confluent, and occur on the face, body, extremities and genital regions. The papules occur principally in the folds of the joints, axillæ, groins and gluteal region and tend to recur and ulcerate; papules of various ages coexist with desquamation, though never with condyloma, which is always a sign of acquired syphilis. Psoriasis of the hands and soles of the feet is common. Pustular lesions occur in severe syphilis and are prognostically unfavorable. They occur in the immature fetus; pemphigus is a subtype of the pustule; gangrene and necrosis may occur. The copper-colored syphilitic infiltration, especially on the nates and lower extremities, may be mistaken for erysipelas. Coryza ("*snuffles*") is very characteristic and almost invariable, the secretion being bloody and purulent, with crust formation, and spirochetes in 20 per cent. Fissures occur about the mouth, with an infiltrated base and cicatrix formation. Papular eruptions occur in the mouth, especially on the tongue and the palate, with a tendency to necrosis; atrophy of the base of the tongue occurs in most cases. *Rhagades* occur about the infiltrated genital and anal regions.

Teeth.—Erosions, furrows and notches may develop. The teeth are small, often irregular and poorly developed. Hutchinson held that the semilunar curving on the free edge of the upper middle permanent

incisors was pathognomonic of tardy congenital syphilis, appearing at the seventh year, but *Hutchinson's teeth* occur in other affections, and are expressions of constitutional derangement only. Later the lateral borders of the teeth become curved; the teeth may become peg-like or the notches grind off even. The other associates of Hutchinson's triad—deafness and the ground-glass cornea—are discussed on pages 214 and 216. Blue sclerotics are accounted symptomatic of congenital lues, and Heine found optic neuritis in 82 per cent. The *Wassermann test* is positive in 90 to 100 per cent.; it may be absent at first, to appear as the clinical signs develop; in one series it was positive in 298 out of 300 cases.

Bones.—As in adults, there may be periostitis, tophi or perforated palate. *Osteochondritis* occurs especially in the lower end of the femur; the diaphysis and epiphysis may be separated. There is (*a*) overgrowth of the cartilage of the diaphysis which produces the wavy line of white cartilage cells; (*b*) the line of cartilage cells becomes thick and foci of decalcification occur; (*c*) the cartilage is as prominent as in rickets, the part nearest the bone being soft. The immobility of the leg may suggest paralysis (Parrot).

FIG. 14.—Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (Jonathan Hutchinson.)

Viscera.—Diffuse infiltration is much more frequent than gummata. There are characteristic changes in certain organs which are rarely involved in acquired syphilis, *e. g.*, in the thymus, umbilical cord, pancreas, lungs, intestines and adrenals. The liver and spleen are greatly enlarged; the Spirochete pallida is found in these viscera in vast numbers, even in the macerated fetus. To conserve the classification, these changes have been described under SPECIAL SYMPTOMATOLOGY. Fournier describes enlargement of the peripheral veins. Vascular changes are especially marked, as in the umbilical vessels, with a tendency to hemorrhage—*syphilis hemorrhagica neonatorum.* Hemorrhages in the skin, mucous membranes and viscera, may be profuse, especially in the liver and lungs, where the circulation is precipitately altered after birth. Levaditi found spirochetes in the foci of hemorrhage. Some vessels may be thickened or infiltrated (disease of the vasa vasorum).

ACQUIRED SYPHILIS IN CHILDREN———*vs.*———HEREDITARY.

Less mortality and less malnutrition.	Marked.
After third month (are exceptions).	Before third month.
A primary lesion occurs.	Absent.
Lymph glands enlarged.	Absent (from accidental causes only).
Usual sequence of primary, secondary and tertiary symptoms. Secondary lesions, then tertiary.	Snuffles, anemia, diffuse pigmentation, pemphigus, involvement of soles and palms, epiphyseal changes, etc. Secondary and tertiary together.

Syphilis hereditaria tarda develops its first symptoms, mostly between the tenth and eighteenth years (rarely before the tenth year). The symptoms are those of infantilism, in regard to puberty, bone development, teeth, hair and brain.

Treatment of Syphilis.—Prophylaxis.—The sexual instinct is even stronger than that of self-preservation. Irregular intercourse is promoted by class differences, lack of barriers among the ignorant and destitute, and the struggle for existence which makes early marriage impossible. Prostitution, hidden or open, is the basis of syphilis. In prostitutes, condylomata last years and repeatedly recur about the genitalia; the Wassermann test is found in all women who have been prostitutes over three years. Continence is the sole prophylactic measure. Enforced medical inspection is ineffectual. American sentiment is against licensed prostitution, which, besides, does not regulate men or hidden prostitution. Licensing also teaches the young that immorality is safe. Many infections are acquired under the influence of liquor (*Sine Baccho friget Venus*). Where ethical considerations fail, an appeal to fear may be effectual. Metchnikoff comments upon the remarkable fact that diseases conveyed by insects are rapidly becoming preventable, whereas diseases like syphilis and tuberculosis conveyed by rational human beings, are not. Efforts to educate the public will doubtless avail much; syphilis, however, must be presented as an infection and dissociated from ethics and retribution.

The curse of the disease lies in its long course, the involvement of vital viscera, its transmission to the wife and offspring whose mortality and morbidity are enormous, and its wide dissemination (13 per cent. of the population acquire the disease, and 1,650,000 persons are infected annually).

Marriage should be forbidden for at least three, better four, years or best, till the sero-test remains negative. In cases of doubtful infection of the wife or husband, both should be treated. Inunctions should be given the pregnant mother. The physician should never countenance employing a healthy wet-nurse for the syphilitic child. Circumcision is thought to lessen chances of infection. Blue ointment, applied before intercourse, is prophylactic.

1. Initial Lesion.—Early mercurial treatment is indicated, but only when the diagnosis is certified by a typical chancre and spirochetes. *Mercury is indicated in all stages of syphilis,* for abortive treatment as well as that of the established disease. Local treatment includes the use of antiseptics, iodoform, mercurial plasters, iodoform in suppository (rectum or vagina), bichloride washes (mouth) and calomel for condylomata (calomel 6 parts, boric acid 3, and salicylic acid 1 part).

2. Secondary Stage.—In the secondary stage *mercury is a specific* against the spirochetes. The teeth should be cleaned first, and bad teeth filled. Calomel acts especially on the lesions of the mouth and is prone to salivate. Mercury may disagree (*v. i.*) in tuberculous, cachectic, alcoholic or anemic subjects, in bleeders and in cases of galloping syphilis, in which latter iodides may be better than mercury. Mercury must be

used with great care in nephritic subjects. During and after a course of mercury, *tobacco must be interdicted* because it perpetuates syphilitic sores in the mouth for years, even decades. Moderation in all matters of life is enjoined upon the patient.

Methods of Administration.—1. By *Mouth.*—Ninety-five per cent. of cases are thus treated in this country, whence some account of this method is appropriate despite the fact that it is uncertain and unwise, and syphilides, gummata, tabes, general paresis and aortitis often develop after oral administration, but rarely after systematic inunctions. (*a*) Calomel (hydrargyri chloridum mite) is especially adapted to children under three years of age with hereditary syphilis; gr. $\frac{1}{4}$ to $\frac{1}{2}$ t. i. d. It is insoluble, and is probably converted by the alkaline carbonates in the intestines into the gray oxide which is absorbed with the fats. (*b*) Blue mass (massa hydrargyri), gr. $\frac{1}{2}$ to 1, is less irritant to the stomach than calomel, but produces salivation oftener than any other mercurial. (*c*) Hydrargyrum tannicum oxydulatum (50 per cent. mercury) is non-irritant, produces less diarrhea and is easily assimilated; gr. 1 to $1\frac{1}{2}$ plus pulv. opii gr. $\frac{1}{10}$; (*d*) The hydrarg. iodidum flavum or protiodide is mild, insoluble and analogous to calomel.

℞—Hydrargyri iodidi flavi . . . . . gr. xv
Extr. opii . . . . . gr. v
Extr. gentianæ . . . . . q. s.
M. et ft. pilulæ no. lx.
S.—One after meals.

(*e*) The soluble sublimate or bichloride is irritating (hydrarg. chloridum corrosivum); it is not prone to salivate, is absorbed by the intestine and stimulates the liver. Mercury, administered internally, produces more irritation and is less likely to be absorbed, but patients are more apt to persist in this form of treatment. Mercury internally was first used by Benedictus (1525).

2. *Inunctions.*—Rubbings must be thorough and given where the skin is soft (flexor surfaces). Mercury is absorbed by the lymph vessels. The skin must be bathed daily. A simple diet should be given; as a rule, mercury somewhat loosens the bowels. The rubbing requires twenty to thirty minutes. Hairy regions, the axillæ, navel, nipple and areas of accidental eczema, psoriasis, etc., are to be avoided. If the hair on the body is redundant, the skin should be shaved. The occasional mercurial dermatitis is often due to decomposition of the fat in the inunction. On the first day the inunction should be given on the abdomen, on the second day on the chest, on the third on the calf, then on the thigh, forearm, arm, etc. After inunction, the part is enveloped in cotton. If an attendant gives the rubbing, his hand is protected by a rubber glove. Written instructions should always be given to the patient.

Twenty to thirty inunctions should be given for the macular and papular types; more than thirty, for all pustular or ulcerating eruptions, but not more than forty should be given continuously. Inunctions must be interrupted for diarrhea or stomatitis. Salivation is not desirable, nor is it a sign that enough mercury has been given. The *unguentum* contains 50 per cent. of mercury, and the more elegant oleatum but

25 per cent., which should be considered, as about ʒss of mercury should be incorporated daily.

℞—Unguent. hydrarg. . . . . ʒiv
Div. in dos. equal. no. xxx.
Dent. ad cartam ceratam.
S.—One as directed daily.

In children, mercurial ointment may be applied on a binder and allowed to remain for days. Children stand relatively large doses but women may endure only half the adult dose. The advantages of inunctions are as follows: They cause almost no digestive disturbance; they are especially good in children; they are the *most rapid and effective method,* and are attended by the least general ill-effects The disadvantages are publicity, troublesomeness and dirtiness, but these, however, are only relative objections. Inunctions were first used by the Galenists.

3. *Injections.*—Their advantages are prompt action and exact dosage. Intravenous injections may be imperative in cases with coma and other urgent symptoms. The disadvantages are the risk of embolism, sloughing and pain; once given, their action cannot be regulated or decreased. The soluble salts are best:

℞—Hydrarg. chlorid. corrosivi	1.00
Sodii chloridi	10.00
Aquæ dest.	100.00

One c.c. daily equals 0.01 gm. or gr. $\frac{1}{6}$ of $HgCl_2$; forty injections constitute a course. The succinamide is less irritating (hydrargyri succinamidum, grain $\frac{1}{5}$); the best preparation is the insoluble basic salicylate of mercury—grain j to ♏x of albolene, injected two or three times a week. Insoluble preparations are painful; ten deaths followed injections of gray oil (Grancher); since 1906, Riehl collected 90 fatalities from injection of insoluble salts. Mercury may first make the eruption clearer (Herxheimer's reaction).

4. *Baths.*—ʒij–iij of $HgCl_2$ to the bath are useful for skin lesions, as syphilitic psoriasis. 5. *Fumigations* are impracticable.

As a working rule, mercury should first be given two months and stopped one month, through the first year, *i. e.*, two-thirds of the time; in the second year, about half the time, with intervals; in the third year, one-third of the time. The Wassermann test is used to control the therapy; the positive sero-test lessens under mercurialization and recurs when therapy is intermitted. To convert a positive into a negative reaction requires very energetic, repeated treatment (*v. i.* page 229).

Mercury is *incompatible with nearly everything.* Coincident anemia is treated by iron, strychnine and arsenic. Syphilitics tolerate mercury well, in large doses, and for a remarkable period. Considering briefly the action of the drug; in the smallest doses it is tonic, and increases the number of red cells. Back of the tongue near the epiglottis and in the folds of the pillars, a white, cloudy film may develop (perhaps due to the organisms of Vincent's angina). In larger doses there develops a symptom-complex known as salivation, of which peculiar fetor is first noted; then

sore teeth, red, swollen, spongy and bleeding gums, and increased saliva, follow. The drug is never given beyond this point. In still larger doses the tongue becomes swollen, the gums inflamed and the teeth loose; the greatly increased saliva is viscid and stringy; the salivary glands intumesce; eruptions appear, resembling eczema, scarlatina, measles and more rarely, purpura and pemphigus. Fever, albuminuria and dysenteric passages may be noted (the kidneys cannot excrete more than 10 mgm. of mercury a day without irritation). Severe salivation results in loss of teeth, necrosis of jaw, ulceration of the mouth and contiguous parts, marked changes in the blood and profound exhaustion. The treatment consists of potassium chlorate as an antiseptic mouth wash and astringents, as tr. myrrh and tannic acid.

3. Tertiary Stage.—The resolvent action of iodides is little short of miraculous. They were introduced by Welch, of Ireland, and popularized by Ricord in France. They are sometimes used in the secondary stage, especially in febrile cases, secondary osseous and nervous manifestations, and weakly persons with a marked macular eruption. They act more slowly and far less specifically on the spirochetes than mercury, though they aid in absorbing gummata or infiltrations, and rendering the tissues less sensitive to the spirochete (*v. s.* Immunity). Their use should be intermittent and alternative with mercury. Mercury is conjointly employed in all tertiaries, particularly when organs of great importance are involved, such as the larynx, brain and eye.

The *therapeutic tests for syphilis* are cure with mercury and iodides and tolerance by the patient of very large doses. Regarding their diagnostic value the influence of the drugs must be unequivocal and a positive Wassermann be converted into a negative reaction. Before negative conclusions are drawn, it must be remembered that loss of tissue, *e. g.*, in arterial occlusion and paralysis from brain softening, cannot be restored.

Sodium iodide is milder, produces less iodism, is less bitter, and has less effect on metabolism than potassium iodide, which, however, is better than other iodides, though it irritates the stomach and bowel, and depresses the muscles and motor nerves.

The following proportions for a single ordinary dose may be employed; the mercury becomes mercuric iodide:

℞—Potassii iodidi	gr. xv (gm. 1)
Hydrargyri chloridi corrosivi	gr. $\frac{1}{32}$ (gm. 0.002)
Tincturæ opii	♏ij (gm. 0.13)
Syrupi aurantii corticis	ʒss (gm. 2)
M. et S.—After meals in a glass of water.	

Large doses of iodide are given in urgent cases, ʒj–ij t. i. d., to break down more rapidly the new-formed granulation tissue. Enormous doses are unnecessary and not without danger (*v. i.*). Fournier warns against their intemperate use—"iodide debauches." Iodide dissolves in its own weight of water; hence one minim of a saturated solution equals one grain of iodide. Iodides are eliminated as the sodium salt; nine-tenths is eliminated in a few hours. If the stomach is intolerant, iodides may be given in nutrient enemata. It is best disguised by syr. sarsaparillæ co.

Iodism is often produced by small, but usually ceases with larger, doses. It is obviated by intermittent administration, by daily baths, by flushing out the kidneys, the inactivity of which is the usual cause, by giving aromatic spirits of ammonia, arsenic or belladonna. Iodism is likely to occur in renal disease. Its leading characters are: *Increased secretion* from the eyes, nose, mouth, salivary glands, bronchi, stomach, bowels, skin and kidneys (*i. e.*, from all organs concerned in the elimination of the drug). The eyes, throat, larynx and salivary glands swell. The writer once nearly suffocated from edema of the pharynx and uvula after ingestion of 10 grains of iodide for a cold. Pulmonary edema and albuminuria, or even nephritis, may develop; ʒss induced edema of the larynx. *Gastric* irritation and *nervous depression,* mental derangement or excitement; *acne,* hemorrhages, blebs and telangiectases in the skin; *cachexia, anemia,* and hyperthyroidism especially in goitrous subjects; emaciation, wasting of mammæ and testes; fever, increased pulse and neuralgias are also symptoms of iodism.

Iodides are continued for two months and their use then remitted for one month and so on. After years, iodides and mercury are given for one month twice a year, *e. g.*, in January and July.

Salvarsan.—Ehrlich (1909) introduced this arsenical preparation as a spirillicide, with the hope that one massive dose would result in complete curative sterilization. It was welcomed as our first, scientific discovery in therapy, destroying all spirochetes in all stages and forms of the disease, acting most rapidly in its production of antibodies and operating in other spirilloses—as relapsing fever and Vincent's angina, as well as in other types of parasitic disease. Its *indications* were: (i) primary lues, in which its best results were claimed; (ii) secondary and tertiary lues, particularly in malignant types and (iii) types refractory to mercury; (iv) hereditary syphilis; and (v) parasyphilitic lesions, without optic atrophy. Its *contra-indications* included: (i) organic diseases of the heart (hypertension, aneurysm, etc.), of the lungs (tuberculosis) and of the kidneys (severe nephritis); (ii) cachectic states, as severe diabetes; (iii) when arsenic had been administered previously, or there was idiosyncrasy to it; (iv) optic neuritis or atrophy; (v) severe nervous lesions, as general paralysis; and (vi) profound hereditary syphilis and pregnancy. The *early results* were striking in rapidity of action, marked tonic effects, the remarkable healing of ulcerations in the mouth, genitalia and skin, coertion of malignant and pustular forms, relief from pain and the amenability of the occasional cases resisting mercury; its specificity in apes (experimental syphilis) and in human syphilis on the clinical lesions and on the Wassermann reaction. The initial optimism was soon followed by *complications; deaths,* sudden or slower; and *prejudice pro* and *contra.* Local complications were necroses at the point of intramuscular injection (20 per cent.), all bloodvessels obliterating and the arsenic being but slowly dissolved. Given intravenously, thrombophlebitis often occurred. *General reaction* was common, with fever, leukocytosis, chill, head and backache, nausea and vomiting, local pain and tenderness, rapid pulse and respiration, even with dyspnea, collapse, bladder irritation, anxiety and other developments, suggesting anaphyl-

axis. The fever was not wholly due to bacteria in the distilled water nor to the saline of the infusion. Paralyses (facial, polyneuritis, etc.) developed; these neurorecidives, specially of the cranial nerves, were attributed to the disease (Benario) or to the drug (Finger). Finger observed 44 neurorecidives in 500 salvarsan injections as against 5 in over 2000 mercurial treatments.

Deaths from hemorrhagic encephalitis, nephritis, cardiac failure, etc., have run into hundreds published and probably many more unpublished. Then salvarsan therapy was "modified"; first given intramuscularly and then intravenously, it was next given both ways; then it was preceded and followed by mercury; now neosalvarsan comes, with its milder local action and solubility, but with its quota of accidents and deaths; and salvarsan instead of sterilizing the body with one dose, is given many times, once a week for 6, 8 or more doses. Adrenalin recently is advocated, to prevent and cure untoward effects.

One may not state now what is the value of salvarsan. It is certainly no more a cure than mercury; it does not replace mercury and even demands its coöperation; it entails a far greater mortality; it entails a surgical procedure with its attendant expense; and it is less penetrative than mercury which becomes part of the tissues as an albuminate, whereas "606" remains a foreign body. When are we justified in using it? If used, the writer's personal preference is for the intramuscular injection, in oil, and repeated; it rarely kills in this form. It is most valuable early before the Wassermann test develops; when mercury fails; in malignant types; and where special prophylaxis is imperative. The intraspinal injection of salvarsanized serum is recommended by Swift and Ellis in lues of the nervous system, since the choroid plexus blocks the blood to the spinal fluid circulation.

Control by the Wassermann Reaction.—With some exceptions, treatment by mercury, iodides or arsenic is measured by the turning of a positive into a negative reaction. Salvarsan leads in the early effects; in later syphilis mercury and salvarsan are alike. After one course of mercury, the reaction becomes negative in 50 per cent.; after 8 or more, in 90 per cent.; it takes several or many intravenous injections to effect the same result. Jonathan Hutchinson held that even mercury pills caused a negative reaction in 70 per cent. Mercury, though slower in action, remains the remedy for the practitioner.

A SYPHILITIC SHOULD NEVER BE DISMISSED or be given any guarantee of the extinction of the disease (Ricord).

MALARIA.

Definition.—An acute infection caused by the Plasmodium malariæ, inoculated in man by the mosquito and characterized clinically by (1) paroxysms of intermittent, quartan, tertian or quotidian fever, or by (2) remittent or subcontinuous fever, and sometimes by (3) pernicious, or chronic cachectic forms.

History.—Malaria, known to Hippocrates and Celsus, is the most completely investigated of all diseases, in that (*a*) its cause is known;

(*b*) its prophylaxis is exact, since the discovery of infection by the mosquito; (*c*) the stages of the disease can be followed in the blood; and (*d*) there is a truly specific therapy.

Etiology.—Men are more exposed to infection; such occupations as sewer and railroad building, ditching and night work, especially, are predisposing factors; 32 per cent. of the French soldiers in Madagascar in 1895 acquired malaria; reduced physiological resistance, exposure, excesses and alcoholism predispose to infection. Negroes have a relative immunity. Epidemics may occur, as during the building of the Paris boulevards. Indirect causes, as climate, the warm season, moisture, rain, swampy localities, bad air (mal-aria) and impure drinking-water are *causes* only in so far as they *relate to the breeding of the mosquito.* In Africa, severe forms of malaria are found everywhere except in its lower part and in the Sahara. In the far East its chief seats are India, Burma, Assam, Faral and the East Asiatic coast; Asia Minor, Japan and the East Indies. In Europe, lower Russia, Italy, Hungary, the Balkans and Greece are most affected. Malaria was probably the cause of the decadence of ancient Greece. Malaria also abounds in the northern three-fourths of South America. In the West Indies, severe types prevail. In the United States, malaria seldom develops on the Pacific coast, in the Northwest, the dry West and the Lake region. Even in the South it is becoming less frequent.

Direct Cause.—The malarial parasite, described by Laveran, in 1880, and Marchiafava and Celli, in 1885, is a red-cell parasite, a hemocytozoön. The biology of the *Plasmodium malariæ* concerns (*A*) its life in the human blood, and (*B*) its life in the mosquito. Various species are recognized because (1) their morphology varies; (2) their geography, seasonal prevalence and malignancy vary; and (3) on inoculation, the type is maintained.

(*A*) The Plasmodium in Man.—Special forms of the parasite:

1. The *quartan parasite* (Plasmodium malariæ) has a life-cycle of three days. In the first twelve hours the young form is a small, amorphous, hyaline spot in the red cell; there is some ameboid movement upon warming the slide. Pigment or melanin appears as coarse, dark rods and granules with little movement. Melanin is hemoglobin digested by the parasite; it is dark or black and resembles the melanin of malignant growths. Movement of the parasite ceases as pigment increases. The parasite occupies one-half to two-thirds of the red cell after forty-eight hours, and almost the entire cell after sixty hours. Flagella and vacuoles (*v. i.*) are not frequent. The red cell on which the plasmodium feeds undergoes no change in size or becomes smaller and darker peripherally. As sporulation occurs, the pigment gathers centrally and the spores, usually numbering not more than ten, develop into the "daisy" form. After sixty-nine hours, segmentation occurs in the peripheral blood three hours before the paroxysm, which corresponds with their rupture from the red corpuscle into the plasma. Sporulation of the adult parasite means its "death in childbed," as Mannaberg describes it; the spores rupture through the red cell and escape into the plasma, whence they enter other red cells, to repeat their life-cycle. The pigment and frag-

PLATE VIII

Malarial Parasites.

A. Tertian parasite. 1, young form; 2-4, stages of growth and pigmentation; 5-7, segmentation; 8, rupture of spores from corpuscle; 9, extracellular form; 10, flagellate form.

B. Quartan parasite. 1-6, development and pigmentation; 7, 8, central grouping of pigment and spore formation; 9, rosette of spores with nuclei; 10, extracellular form; 11, flagellate form.

C. Æstivo-autumnal parasite. 1, ring-like body; 2, 3, ameboid forms; 4, parasite in brassy degenerated red cell; 5, pigmentation; 6, segmentation; 7, 8, half-moon bodies; 9, flagellate forms.

ments of red cells are carried by the leukocytes to the liver and spleen. Some forms (gametocytes) do not mature nor sporulate, but are taken up by the mosquito, to complete in it another cycle. (Plate VIII.)

2. The *tertian organism* (Plasmodium vivax) has a forty-eight-hour cycle. In its first day it is a small (1μ or 2μ), bright, unpigmented disk in the red cell, thrusting out pseudopodia, with active ameboid movement, and possessing a large nucleus—partly clear chromatin, and partly deeply stained chromatin. It fills one-third of the cell. In its second day, pigment accumulates as fine granules or rods, which are peripherally located, and have a swarming movement, like that of boiling water, due to currents in its protoplasm. The ameboid movement decreases in thirty hours, as the parasite grows and the pigment increases, but some movement remains for a long time. Vacuoles are frequent. The parasite occupies two-thirds to three-fourths of the red cell, which becomes distended and chlorotic. The most common form of sporulation is the concentric accumulation of pigment and the development of fifteen to twenty small, round, rough, refractile spores, which form in two concentric circles ("sunflower" arrangement), or are more frequently irregular, like a bunch of grapes. Sporulation occurs three hours before the chill and rise of temperature, and is completed in forty-eight hours, when the toxins secreted produce the rigor and fever. Few spores are seen at the time of the chill, because sporulation occurs to a greater extent in the internal organs than in the peripheral blood stream. When spores are seen between paroxysms they represent the sporulation of another generation of parasites, too few to induce a paroxysm. The generations are never absolute, as they are in the quartan type, and the parasites of the same generation vary six or eight hours in their cycle. This explains the clinical variations in periodicity. A few minutes after withdrawal of the blood, nodulated and clubbed flagella lash about and indent the red cells. They may break off and swim about. They occur in parasites which never sporulate in human blood, but remain as large bodies with vibrating pigment, representing gametocytes or "sexual forms," which develop later in the mosquito (MacCallum).

3. The *estivo-autumnal parasite* has a life-cycle of from twenty-four to forty-eight hours. There are two subtypes—the tertian (Plasmodium falciparum) and quotidian (Plasmodium falciparum quotidianum). It has a very small, unpigmented body, and possesses most active ameboid movement, thus attracting early attention, though it possesses almost the same refraction as the red cell. It becomes quiet after drawing the blood from the body, when it appears characteristically ring-like, often with a central vacuole. The shape may be successively ameboid, diskoid, annular, cruciform, or with a slight knob—the seal-ring form. Mature forms contain particles of hemoglobin. The pigment is extremely fine, powder-like, dark, peripherally located, and somewhat motile. When the parasite reaches one-third the size of the corpuscle, it is usually motionless, the pigment concentrating centrally. Motion in the parasite has been seen in the cadaver. The spores number six to sixteen, are very small, round, and without movement or vacuoles. Fever, pigment concentration and sporulation coincide (Golgi). Large parasites in the

peripheral blood with much pigment denote the onset of a paroxysm, and after the paroxysm, ring-forms without pigment may be observed. Young forms circulate in the blood stream, though sporulation occurs in the viscera. The *half-moon bodies* or *crescents* are delicate, highly refractile bodies, measuring eight to ten by two or three microns, develop in the bone-marrow or spleen, and possess pigment, which is usually mesially located and has little motion. They have no ameboid movement but may slowly change shape; they are spindle, oval or spherical. They occur singly in the red cell and do not multiply in the human blood. The adult forms are seen in the peripheral blood, which offers the best opportunity for leaving the body—through the bites of mosquitoes. The male form of the crescent has flagella (microgametes) which penetrate the sexual elements of the female crescents (macrogametes), outside of the body, either on the slide or in the body of the mosquito (Ross). Fever rarely occurs when crescents alone are found in the blood; in fever, ameboid bodies, also, are nearly always found. The red blood cell becomes copper-colored or brassy.

The parasite may be stained with the Nocht-Romanowsky fluid, methylene blue or hematoxylin, and counter-stained with eosin. For the beginner, study of the fresh blood under an oil-immersion is the best method.

(*B*) The Life of the Organism without the Human Body.—King (1883) first suggested that the *mosquito* was the intermediate host and sole source of infection. It takes the plasmodium from man and reinoculates him with it after the life-cycle within its body. Other analogous infections are known, such as the hemosporidia of birds, frogs, bats, dogs, sheep, monkeys, Texas cattle-fever inoculated by the tick, and the African trypanosoma in animals and man, inoculated by the tsetse fly. Manson (1894), especially, then Ross, Grassi, Bastianelli, Bignami and others, have developed this interesting subject. The Plasmodium malariæ is found only in man and the mosquito. The mosquito is the primary and higher host, since the sexual reproduction in its stomach is biologically higher than the simple sporulation in man. Of its two chief genera, the culex does not contain or convey the parasite. Its palpi are very short and its wings have no mottling. When sitting, its hindlegs are carried above the body and its body lies parallel with the wall, or may sag from the blood it has sucked. Its eggs are laid in tanks or puddles. It is the ordinary house mosquito and is found chiefly in cities. The *anopheles conveys malaria*. Its palpi are almost as long as the proboscis; the wings are mottled; the hindlegs usually depend, or touch the wall. Its body makes an angle of 45° with the wall. The eggs are laid in pools or sluggish streams with algæ growth, and the open country is its preferred habitat. Species: (1) The most important, the Anopheles maculipennis; (2) A. crucians; (3) A. punctipennis; (4) A. argyritarsis. After the female anopheles bites persons whose blood contains sexually mature forms of the plasmodium (gametocytes), and the blood reaches the insect's stomach, flagellation in the male form occurs and the flagella enter the female cells. After fecundation the impregnated cell enters the muscular walls of the intestine, and,

after two days, small, round, refractile, pigmented bodies appear, which, after a week, grow to 60 to 70μ in diameter. They develop into myriads of sporoblasts which reach the salivary glands of the insect, whence they are again inoculated into man by biting. The sporozoids thus resulting from sexual conjugation in the mosquito, are equivalent to spores formed by asexual sporulation in the human blood. Entering the red cells in man, they follow the cycle of their type as above described, and produce either sporulating forms or sterile forms. The sterile forms in the human blood—*i. e.*, the forms which do not sporulate—linger in the peripheral blood with the teleological purpose of preserving and continuing their life in the mosquito, and, on reaching its body, develop again the agents of reinfection. This knowledge concerning the mosquito explains many interesting and formerly incomprehensible details. We now know why those living in the first story of houses are more likely to be infected than those living in the upper floors and why those who slept in houses facing inland were infected while those in houses exposed to the sea air escaped, etc. In Rome, the fever prevailed just outside the Porta del Populo, while the Corso, three hundred feet distant in the city, escaped. During the winter, man, not the mosquito perpetuates the disease. Bass and Johns successfully cultivated all types of malarial parasites.

Symptomatology.—1. FEVER.—Fever is, next to the melanin and plasmodium, the most constant symptom. It develops after an incubation of one and a half to fifteen days. *Fever bears a close relation to sporulation* (Golgi, 1885). The fever-curve is irregular in some types or almost mathematically regular in others. It is usually irregular at first even in regular types. It may be remittent (subcontinuous), intermittent (tertian, quartan, quotidian), or continuous for a few days, when it ends by crisis or becomes remittent or intermittent before recovery. The paroxysm may be delayed beyond the time when it is due (*postponens*), or may anticipate this time (*anteponens*). In the algid variety there may be hyperpyrexia, or normal or subnormal temperature. Wade Brown's experiments suggest that the paroxysm is caused by the pigment.

The classical components of the paroxysm are chill, fever and sweating, whose respective duration is in the proportion of 3–2–7, but any component may be absent, especially in virulent varieties and in persons partly immunized. The paroxysm lasts ten (four to forty-eight) hours, and in 91 per cent. occurs between 10 A.M. and 3 P.M. (*a*) The *chill* is sudden in onset, often severe, with headache, emesis, coldness and lividity of the skin (*cutis anserina*); the temperature by rectum is elevated, the pulse rapid, small and hard; the respiration is rapid or even dyspneic, and the urine is increased. The parasites pervade the peripheral blood in the tertian and quartan types but largely the internal organs in the estivo-autumnal type. (*b*) The *fever* is high, perhaps not higher by rectum than during the chill, but is more evidenced by the red skin, thirst, delirium, headache, soft, full or dicrotic pulse, and other fever symptoms. In this stage the young parasites are free in the blood and begin to attack new red cells. After their entrance into these cells the fever ceases. (*c*) In the *sweating stage* the young parasites are found in the red cells and phagocytes take up the detritus and pigment. The sweat is usually

profuse, giving off an odor like that of semen. Postfebrile euphoria is usual.

Special Febrile Types of Malaria.—The quartan and tertian forms are regular and intermittent, and the estivo-autumnal forms are more irregular and remittent.

Fig. 15.—Quartan malaria. (Silvestrini.)

1. The *quartan fever* is the rarest of all types (0.8 per cent.), is found in the temperate zones, and occupies a position geographically intermediate between the tertian and estivo-autumnal varieties. It is caused by the quartan parasite, is the most regular of all types, and is called quartan because the simple form causes paroxysms every fourth

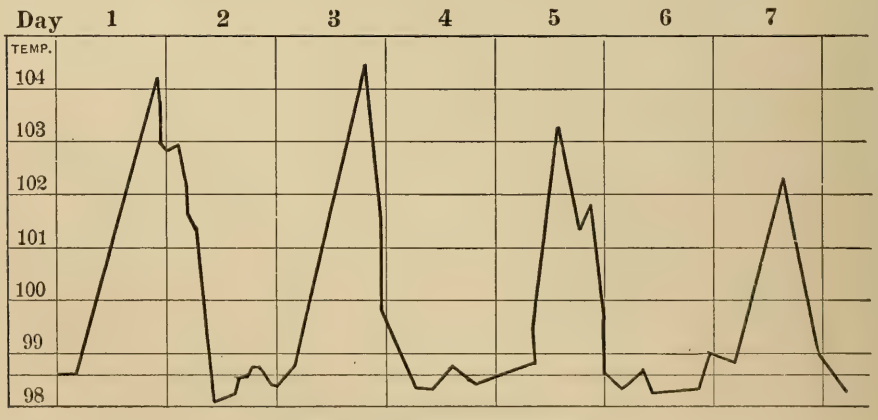


Fig. 16.—Tertian malaria.

day. The parasitic cycle is readily followed in the circulating blood, and the organisms do not accumulate in the internal organs. The fever is obstinate and recurrence is frequent, but quinine quickly exerts its specific action. The type may be single, from one generation of parasites; double, from two generations; or triple, from three generations producing,

respectively, temperature, every fourth, every first and second, or every day of the cycle.

2. *Tertian fever*, most frequent in the temperate zone, is found in all malarial countries and constitutes 50 to 65 per cent. of all malaria. The organisms accumulate in the internal organs. It is rarely pernicious. The type may be single, or more frequently, double from two generations of parasites, causing almost continuous fever. It responds readily to quinine and may recover spontaneously.

3. *Estivo-autumnal fever*, caused by the estivo-autumnal parasite, is often irregular, remittent, intermittent or continuous (subcontinuous). It is characterized clinically by severe recurrent fever, marked anemia, frequently by absence of paroxysms (chills occurring in 71 per cent. only) and sometimes by pernicious symptoms. It was called estivo-autumnal by Marchiafava and Bignami, in contradistinction to the milder or vernal forms. *Subtypes:* (*a*) *Regular* intermittent estivo-autumnal fever: (i) The estivo-autumnal tertian, in which the fever is apt to be

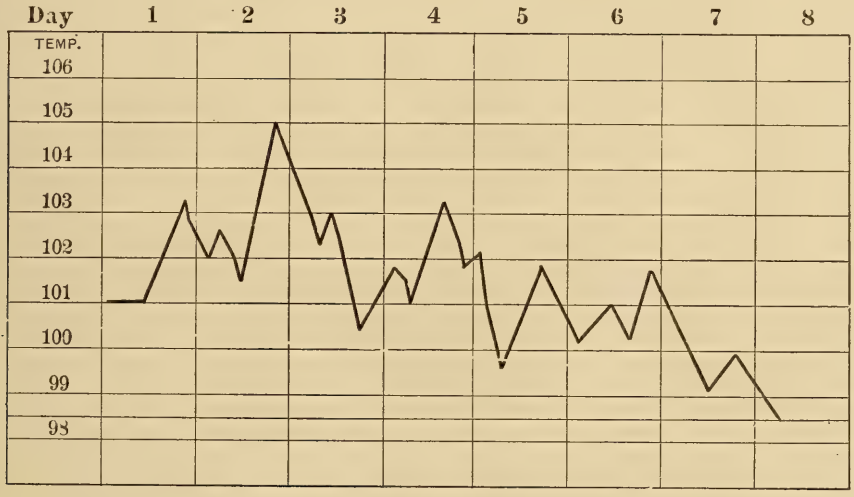


FIG. 17.—Estivo-autumnal malaria.

high, and the paroxysm doubly long. The typical curve may be modified by an exaggerated pseudocrisis, a briefer or longer duration of the attack, by anticipation or procrastination, and by double attacks. (ii) The estivo-autumnal quotidian, with a short attack, but an abrupt elevation of fever. This often irregular form has fewer symptoms than the other variety, and may recover spontaneously. It is readily confused with typhoid. (*b*) The *irregular* form presents marked irregular intermittency or remittency, or subcontinuous fever, because the attacks are duplicated, anticipated or prolonged. The paroxysm may last twenty hours. The symptoms may be mild or very severe.

2. BLOOD FINDINGS.—The *blood findings are pathognomonic*, viz., parasites, melanemia, anemia, and absence of leukocytosis.

(*a*) *Red Cells.*—Anemia results from deglobulization by the parasite. *Acute anemia* is more marked and rapid than in any other infection. The loss is greatest in the first attack, sometimes reaching half a million cells. Reduction to 500,000 red cells may give the picture of pernicious anemia.

(*b*) *White Cells.*—The absolute and relative *leukopenia* is due to the collection of the white cells in the liver and spleen, to destruction of the phagocytes, and to lesions of the blood-making organs. As in other anemias caused by disease of the bone-marrow and spleen, an increase of lymphocytes or large, mononuclear cells (20 per cent.), with decrease of the polymorphonuclear cells, may be observed. An absolute increase is sometimes observed in the "black-water fever," but the increase is in the lymphocytes. (Thomson describes a leukopenia (when many parasites sporulate) and a leukocytosis, even up to 50,000 or 125,000, when few parasites sporulate.)

(*c*) *Melanemia.*—Melanemia (Virchow and Heschl, 1847–50) is, *next to the presence of the plasmodium, the most constant change* in malaria. It is present during fresh infection and may occur shortly after the fever has disappeared, as in the half-moon forms. After infection it usually disappears within forty-eight hours. It is *practically pathognomonic of malaria,* though said to occur in recurrent fever, melanotic new growths and Addison's disease. It is found chiefly in the polymorphonuclears. It is seen in the smaller capillary walls where their diameter suddenly decreases (lung alveoli, intestinal villi, renal glomeruli, cerebral convolutions, spleen, liver and bone-marrow); the capillaries may be occluded by pigment. Besides this pigment copper-colored hemosiderin may be found in the vessels and hepatic parenchyma.

3. Spleen.—Enlargement of the spleen (Andouard, 1803) is the third important finding. It is *palpable in* 88 *per cent.* of cases and anatomically is rarely absent. Its size, hardness and plumpness are proportionate to the duration of the fever, increase with its rise, remit between attacks and disappear with recovery. In acute infections the spleen is increased three- to fivefold; it is soft and friable: the parenchyma is flooded with infected red and macrophagic cells; and there are areas of focal necrosis. The Malpighian bodies contrast strongly with the congested pulp. A sticking pain over the organ is due to perisplenitis, and is often associated with pain and tenderness in the bones. In chronic infections the spleen is permanently enlarged (fever-cake), reaching even into the pelvis. It may weigh ten pounds, the capsule is thickened, even calcified, perisplenic adhesions occur and friction is frequent. Sometimes a systolic murmur is heard. The redundant pigment gradually decreases. The acute hyperemia and focal necrosis lead to increase of the connective tissue; ultimately little remains of the pulp, follicles and splenic function. Rare complications are pressure on the intestines, nerves and ureters, torsion of the pedicle of a wandering spleen, abscess or rupture (of which Choux collated 147 cases).

4. Skin.—The vasomotor constriction during the chill gives way, during the febrile rise, to hyperalgesia and vasodilatation. Miliaria appear in the sweating stage. Herpes is very frequent, mostly nasal or labial. Edema and a cachectic hue occur in chronic cases. Slight urobilin icterus is not infrequent; icterus is sometimes intense. Infrequent complications are urticaria, erythema, roseolæ, petechiæ, etc.

5. Circulatory Symptoms.—The heart is little involved. The pulse rises parallel with the fever, small at first from vasoconstriction, later

full from vasodilatation. Endocarditis, myocardial degeneration with dilatation, and phlebitis are rare developments.

6. Respiratory Symptoms.—The rare dyspnea and pulmonary congestion during a paroxysm are due to parasites and phagocytes accumulating in or even occluding the capillaries of the lung. Plasmodia have been seen in the sputum. Chronic bronchitis, emphysema and "proliferating" interstitial pneumonia occur as complications. Bronchopneumonia is frequent in malarial cachexia. Lobar pneumonia occurs only as a mixed infection. It is atypical and, in malarial cachexia, is especially fatal.

7. Digestive Symptoms.—The oral mucous membrane is dry, the tongue heavily coated and the breath foul. The stomach may be the seat of hyperemia or infarction with plasmodia. Anorexia, vomiting and gastralgia are common symptoms. Ulceration from amyloid degeneration is observed only in a few chronic cases. Hematemesis from chronic splenic tumor has proved fatal.

The Intestines.—Swelling of the lymphatic structures, hyperemia, thrombosis from parasite aggregation, meteorism and rarely diarrhea are observed. Acute abdominal pain is not uncommon; it may simulate peritonitis. Rarely the disease may resemble dysentery, with ulcers in the large and small gut.

The Liver.—In acute infections the liver is enlarged, hyperemic, and its macrophagic endothelial cells are swollen, though few parasites are present. Icterus may develop from red-cell destruction; it is usually polycholic. Pigmentation from degenerated red cells is more common than actual melanosis. In chronic infections the liver may weigh eight or ten pounds, is hard and smooth, and has a thick capsule. The changes, *seriatim*, are focal necrosis, atrophy or partial regeneration, and pigmentation with hyperemia; angioma, lymph cysts or abnormal lobules; and hyperplasia of the perilobular connective tissue, with ultimate cirrhosis. Cirrhosis may follow chronic malaria without necessarily being of malarial origin. Amyloid degeneration and simple atrophy from pylethrombosis are uncommon sequels. Acute ascites may result from pylethrombosis, by plugging of the capillaries by melaniferous cells.

8. Genito-urinary Symptoms.—Polyuria is often observed during the paroxysm. The urine is decreased in the estivo-autumnal type. The postmalarial polyuria of convalescence is frequent. In one instance the author observed a veritable diabetes insipidus.

The urea, chlorides and specific gravity of the urine are increased. *Albuminuria* is present in 30 per cent. of cases, and usually disappears with the paroxysm. Ehrlich's diazo reaction is obtained in 5 per cent. of the cases. *Nephritis* occurs in 4.5 per cent. but is seldom fatal. Plasmodia frequently abound in the intertubular capillaries but rarely in the glomeruli. Glomerular desquamation and epithelial exfoliation, endocapsular exudation, necrosis and cast formation in the convoluted tubules occur. Chronic parenchymatous and interstitial nephritis may result from malaria. Hemoglobinuria, anuria and uremia occur in black-water fever. Gangrene of the genitalia is an uncommon sequel. Metrorrhagia and abortions occur frequently.

9. Nervous Symptoms.—The parasite especially affects the nervous system, whence Van Swieten called malaria a neurosis. There is scarcely a nervous symptom which is not observed occasionally in malaria. The sensorium, involved slightly in mild cases, is seriously affected in pernicious types (*q. v.*). Headache is usual, and neuralgias are frequent, especially of the fifth nerve. In pernicious types it is not easy to determine whether nervous symptoms are due to toxemia or cerebral thrombosis. The endothelial cells of the vessels are fatty, swollen and pigmented. Thrombosis in grave cases may result from swelling of the endothelium, though it is usually caused by the rough infected red cells. The infected erythrocytes never leave the vessels.

Paralysis (hemiplegia, paraplegia and monoplegia), or motor irritation (tremor, chorea, contractures, epileptic attacks or reflex spasms), may occur in urgent types. Neuritis, psychoses, ascending spinal paralysis, bulbar symptoms, symptoms resembling multiple sclerosis, tabes or acute ataxia are rare sequences.

10. Muscles and Bones.—The muscles and bones are often painful. The bone-marrow at first shows fat and pigment which then disappear with marked vascularization, and ultimately, proliferation of the marrow cells and connective-tissue thickening.

Latency.—Craig analyzed the latent malarial infections in which the plasmodia may be demonstrated in the blood, without conspicuous symptoms. The latent type amounted to 24 per cent. of the cases examined, which were discovered only in the routine examination; they might have been sources of infection to others for weeks or months.

Relapses.—Relapses are almost invariable in all forms, but are most obstinate in the estivo-autumnal type. Craig believes that the chief cause of maintenance of malarial infection is intracorpuscular conjugation, thus producing within the body a resting or zygote stage of the plasmodium which resists quinine.

Chronic Malaria.—Untreated infection, or reinfection, persists for months, notably in the estivo-autumnal variety. It is accompanied by irregular fever, marked anemia, the parasite in the blood, and splenic and hepatic swelling. In severe chronic cases, the *malarial cachexia* develops, usually after years of infection. It is most marked in intensely malarial regions, in estivo-autumnal infection, and in reduced subjects. The fever is slight, irregular or absent. The features of malarial cachexia are (1) anemia from defective blood regeneration, with its general symptoms, such as edema, rapid pulse, venous thrombosis and hemorrhages in the skin, retinæ, etc.; (2) vascular changes, possibly due to pigment occlusion; (3) enlarged spleen and liver; and (4) gangrene, nervous symptoms, alimentary and respiratory catarrh, amyloid disease, liver hypertrophy, and extreme malnutrition.

Relation to the Other Diseases.—Malaria and typhoid rarely coexist; Craig saw but 8 instances in 5000 personal cases. (The plasmodia, and typhoid bacillemia or the Widal test are necessary for diagnosis.) *Surgical operations* may arouse a latent malaria. Erysipelas, septicemia, dysentery, cholera, smallpox, tuberculosis, etc., very infrequently coexist with malaria.

Pernicious Malaria.—Pernicious malaria constitutes but a small proportion of cases of estivo-autumnal malaria, and very seldom follows infection with the ordinary tertian parasite. It is characterized by severe obstinately recurring fever, intractable anemia, pernicious visceral symptoms, abortive paroxysms without chills and by depression. The pathogenesis of perniciousness lies in the inelastic, irregular, infected red cells which cannot pass the visceral capillaries, and therefore slow the blood current. Melanosis, phagocyte aggregations and endothelial swelling are of secondary importance. In cases of capillary hemorrhages, only the normal, not the parasite-laden red corpuscles, escape from the vessels. Regressive organic changes may occur in the cerebrum, gastro-intestinal mucosa, spleen, liver and kidney. Accessory factors are (*a*) arteriosclerosis, renal disease, cardiac dilatation, alcoholism, sunstroke, overwork, malnutrition and lack of acclimatization; (*b*) the number of parasites and their abundance in internal organs. Estivo-autumnal parasites, with large blocks of central pigment, indicate many plasmodia in the viscera. The parasites are often few in the peripheral blood though abundant in the brain at autopsy; (*c*) great activity in multiplication may be observed in the peripheral blood even during the sporulating stage; and (*d*) necroses in the viscera and the great morphological alteration of the "brassy" red cells suggest an increased toxicity.

1. The pernicious *typhoid form* (*perniciosa typhosa*) is inappropriately called typhomalaria. Its clinical picture is variable; chills are often absent. Years ago the differential diagnosis was made *ex juvantibus* by quinine. Without blood examination malaria and typhoid may be confused; benign or malign malaria may resemble typhoid closely, with slow dicrotic pulse, splenic tumor, headache, fever-curve, cerebral and abdominal symptoms, and even roseolæ and intestinal hemorrhage; on the other hand, erratic typhoid with remittent temperature, chills and sweats, or typhoid in the latter part of its normal course, may simulate malaria.

The following differentiation is modified from Baccelli:

Malaria	Typhoid.
Begins intermittently.	Progressively remittent.
Remissions very irregular.	Regular in the morning; step-like.
Temperature may be high at first.	High fever only after several days.
Headache in beginning is rare, variable in site and intensity, pulsating and neuralgic.	Early, permanent, frontal, oppressive, characteristic.
Eyes dull, subicteric from beginning.	Glistening eyes; no icterus.
Stupor, dry tongue and sordes, less.	Marked.
Breath nauseating.	Mouse-like.
Delirium from beginning; recurrent.	In advanced stage; constant.
Lung congestion begins suddenly; foci change recurrently; dyspnea from deranged circulation.	Slow development, hypostatic; dyspnea rare (from tympany).
Nervous restlessness, jactitation.	Stupor, prostration.
Meteorism not characteristic; stools darker.	Meteorism and diarrhea begin gradually; pronounced; ochre stools.
Liver congestion frequent; subicterus.	Less congestion; rarely icterus.
Herpes frequent.	Rare. Roseolæ.
Cycle, none.	Typical.
Blood: plasmodia and melanemia.	Widal reaction and typhoid bacillemia.
Recurrence.	Relapses less frequent.
Convalescence more rapid and easier.	Tardy.
In adynamic type, icterus, hemoglobinuria, leukocytosis, great anemia, 25 per cent. mortality.	Does not respond to quinine, which is a specific in malaria.

2. Of *cerebral forms*, the comatose (*perniciosa comatosa*) is the most frequent. Mental depression is followed by contracted pupils, involuntary evacuations or paralysis. The pulse at first is slow; death is usually cardiac. The respiration may be noisy, hurried or Cheyne-Stokes's. If fever is present it is irregular. The author has seen four such cases in Chicago. Apoplexy, uremia and sunstroke must be considered. The splenic tumor *per se* suggests a blood examination. The "*meningeal type*" occurs especially in children; the slow pulse, headache, rigid, tender neck, stupor, convulsions, hyperesthesia and ultimate coma can be correctly interpreted only after a blood examination. Delirium may be the main symptom, and may appear as a mania, as fixed ideas, hallucinations or as melancholia. The rare *convulsive* type may be generalized, simulating hysteria, uremia or epilepsy, or localized, with contractures, trismus or opisthotonos. The *paralytic* is the least frequent type; hemiplegia with aphasia is more common than monoplegia, the bulbar form, etc.

3. The *algid pernicious type* occurs with cyanosis, collapse, Hippocratic expression, alimentary symptoms, and usually, early death.

4. After the cerebral type the *pernicious gastro-intestinal* variety is most frequent, with vomiting, epigastric distress, icterus and dysentery. In soldiers from Cuba, the author observed profuse hemorrhages from the bowel, promptly responding to quinine.

5. The *pernicious biliary fever* ("jungle fever") occurs with vomiting, polycholia, icterus, nervous symptoms, hemorrhages, adynamia, edema and dark albuminous urine; even after the administration of quinine this type may be lethal.

6. The *hemorrhagic types* resemble Werlhoff's disease, and scurvy.

7. The *febris biliaris hemoglobinurica* ("black-water fever") occurs in South America, in Africa and in our Southern States. The suspected causal factors are repeated malarial attacks, undue exertion, cold, and the use of quinine. Some maintain that it is a separate disease. The cardinal symptoms are (*a*) hemoglobinuria; the urine is always albuminous, neutral or alkaline in reaction; hemoglobin is detected only after the destruction of at least one-sixth of the red corpuscles; (*b*) icterus, invariable except in the mildest cases; (*c*) irregular fever; (*d*) the plasmodia, found in 96 per cent. before the attack and in 62 per cent. at the onset. The mortality of 50 per cent. is due to syncope, anuria and coma.

Diagnosis.—1. From Symptoms.—A probable diagnosis can often be made, for instance, by typically intermittent *fever* which occurs early in the day and is distinct from the vesperal hectic or septic fever. The three elements of the paroxysm are most marked in the benign malaria. In estivo-autumnal forms the chills may be absent. *Splenic enlargement* and *herpes* are suggestive, but also occur in ephemeral fevers, pneumonia, etc. Much emphasis was once placed on intermittent neuralgia, vertigo, herpes zoster, etc., but intermittence is not synonymous with malaria.

2. The Diagnosis ex Juvantibus.—Laveran excluded malaria, when the fever resisted 30 grains of quinine for more than four days; in tropical forms more quinine may be necessary.

3. The Plasmodium may be mistaken for vacuoles resulting from mechanical injury to the red cells; retraction of the hemoglobin; swarm-

ing particles from the red blood cells; blood plates, sometimes mistaken for spores, in unstained specimens; and poikilocytosis. Fewest mistakes are made when the inexperienced observer examines the fresh blood. Leukocytes containing pigment are distinguished by their large nucleus, and by their ameboid movement which is lacking in plasmodia sufficiently large to cause confusion.

Positive findings are obtained in 98 per cent. The malarial parasite is found in the blood of malarial patients only. It is usually stated that they occur in largest numbers before or at the time of the paroxysm, though it must be admitted that the early examinations are often negative. Quinine may obscure the findings. Melanemia is practically pathognomonic. (Pigment lying free in the plasma is most often contamination.)

Negative findings are of relative value only, and depend upon the skill of the observer and the time of examination. The findings were negative in only 2 per cent. of Baccelli's series.

Differential Diagnosis.—1. From *typhoid* (*v.* pages 46 and 239).

2. *Sepsis,* pyemia (page 46), ulcerative endocarditis, liver abscess or pulmonary phthisis may be confounded with malaria. We find in malaria the plasmodium, melanemia, response to quinine and absence of leukocytosis; whereas in sepsis, leukocytosis, positive blood cultures and septic localizations are distinctive.

3. Irregular grippe, recurrent fever, miliary tuberculosis, fever in malignancy, especially sarcomatosis, pernicious anemia, pseudoleukemia, Weil's disease and yellow fever may be confused with malaria, without careful blood examinations. Pernicious malaria may superficially simulate apoplexy, sunstroke, meningitis, cholera or purpura. Among 1267 malaria patients, 25 per cent. were "masked," simulating tuberculosis, pneumonia, dysentery, appendicitis, etc. (Craig).

Prognosis.—The type of parasite, reinfection, habits of the individual, and antecedent renal or cardiac disease, are most important considerations. The prognosis must be conservative in pernicious and cachectic forms. In Italy the yearly death-rate was 16,000, and the French lost 6000 soldiers in Madagascar in three months. On the other hand, fully 20 per cent. recover with rest in bed and a full diet, as is shown in cases treated with placebos and saved for clinical demonstrations; in spontaneous recovery the factors are the *phagocytes* in the spleen, bone-marrow and to a less degree, the endothelium of the hepatic and cerebral vessels, Kupffer's cells in the liver, and possibly the leukocytes.

Treatment.—1. PROPHYLAXIS embraces more than personal prevention. In Italy and elsewhere it is a national economical problem. Drainage, the filling-in of stagnant pools and swamps and the planting of pines, eucalyptus trees and sugar-cane, have proved successful; the drainage of the great Bordeaux swamp in 1805 cost 3000 lives. In digging canals or building roads, immunes should be employed, but whites may work successfully if they avoid alcohol and nocturnal infection by mosquitos, minimized by nettings on all doors and windows. The pools where the insects breed should be filled in, or covered with crude petroleum to kill the larvæ. The stocking of semistagnant streams with fish is of value. The prophylactic use of quinine in daily doses of 10 to 40 grains

is effective. Sick malarial patients and those with malaria but without symptoms, should be protected from the bites of mosquitos, which carry infection to others. In the Isthmian Canal Zone, Gorgas reduced the mortality to one-seventeenth of the French death-rate. The Italian death-rate has been reduced from 16,000 to 3500 annually.

2. Specific Treatment.—*Quinine* is one of our few specifics. Cinchona has been used for ages in Peru and was known as the "tree of health" to Pizzarro in the conquest of Peru. Introduced over one hundred years later into Europe by del Vego (1640), its alkaloid was discovered by Pelletier and Caventou in 1820. It *acts on the malarial organism while it is developing* (Marchiafava and Bignami); when the parasite has transformed all the hemoglobin into melanin, and segmentation begins, quinine is ineffectual. All malarial manifestations as fever, splenic tumor and anemia are cured by quinine except in pernicious forms. It acts as a direct protozoan poison. Malarial subjects escape many or all of the symptoms of cinchonism, such as tinnitus aurium, cephalic distention and slight deafness resulting from therapeutic doses (gr. x). (Deafness, disordered vision, flushed face, cerebral congestion, vomiting, staggering, tremor and twitchings result from larger doses; and delirium, complete deafness and amaurosis, dilated pupils, convulsions, paralysis, hemoglobinuria, dyspnea, coma, weak heart, or hemorrhages result from toxic doses.)

Administration.—Its antiperiodic action is enhanced by combining it with opium gr. ss. Laxatives are unnecessary. Pills are insoluble, and the drug is best given *in powders involved in starch wafers*, or with one drop of sulphuric acid to each grain of the sulphate. Coffee, cognac and extract of glycyrrhiza but poorly disguise its bitterness. If the stomach rebels, a warm-water enema with equal parts of quinine and tinct. opii deodorat. may be used. In severe cases the hydrochloride (gr. vij) in 15 minims of water may be used hypodermically, but, though every care be taken, abscesses develop often. In pernicious types, Baccelli injects quinine intravenously (sterilized solution of sodium chloride, quinine muriate and water, in the proportion of 1, 10, and 100 parts, respectively).

Time of Exhibition.—If the fever recurs regularly, gr. xx–xxx should be given as the fever falls, to act on the young growing forms in the red cells, to be repeated before the next paroxysm, in order to have the drug in the circulation when the spores are scattered (Torti and Golgi). If the fever is continuous or subcontinuous, or if the type is severe, gr. v should be given every four hours. Large doses are more effectual; small doses only aggravate malaria and develop quiescent forms. In the treatment of black-water fever, one view maintains that quinine is its cause; it should be given only when plasmodia are found, and given, as in Italy, a daily dose of six grains, for three months.

3. Treatment of the Paroxysm.—During the vasoconstriction of the chill, morphine gr. $\frac{1}{4}$ and nitroglycerin gr. $\frac{1}{50}$, hypodermically, and spirits of chloroform ℥j, in hot whisky and water afford great relief. The fever is short-lived and requires little beyond cool sponging. The sweating requires no treatment.

In chronic malaria, iron and full, increasing doses of arsenic should be combined with quinine. Arsenic is said to act on the gametocytes. Salvarsan is recommended for obstinate types.

RECURRENT FEVER.

Synonyms.—Relapsing fever; febris recurrens; typhus recurrens; seven-day fever; famine or prison fever; bilious typhoid.

Definition.—A specific infectious disease caused by Obermeier's spirillum; endemic in Ireland, Russian Poland and India; characterized clinically by cyclic febrile attacks lasting six or seven days, followed by an equally long apyretic interval and recurrence of the fever.

Etiology.—Recurrent fever was first described by Rutty, of Dublin, in 1739. It appeared in America in 1844, and was last seen in 1869. Obermeier in 1873 described a specific spirillum or spirochete (*Spirillum Obermeieri*). S. T. Darling concludes that the Panama spirochete is of a strain differing from the European; the African tick fever is caused by the S. duttoni, the relapsing fever of India by the S. carteri and the American form by the S. novyi. The secretions are apparently not infectious. The disease has been inoculated in monkeys and in man, from postmortem cuts. Bed-bugs and lice also convey it. Infection is carried chiefly by vagabonds and emigrants. Males are especially affected (90 per cent.). Most cases occur between the twentieth and the fortieth years.

Symptoms.—After an incubation of five to seven days, usually without prodromes, the invasion begins with a chill, high temperature (reaching possibly 104° on the first day), throbbing headache, and, in the young, convulsions and vomiting. The vertigo and cerebral confusion may resemble acute alcoholism. Because of the severe neuralgic pain in the back and legs, the patient lies motionless. There are great depression, conjunctival suffusion, subicteric scleræ, facial pallor, epigastric oppression, with or without vomiting, and tenderness over the liver and spleen.

Specific Findings.—1. *The blood* is dark and reveals the pathognomonic, motile, cork-screw-shaped spirillum. It appears with the fever, disappears before it falls, and reappears with the next attack. The spirilla are found in the blood only. Without the immersion lens they may be located by the lashing about of the red cells. They always lie between and never in them, appear as fine threads, sixteen to forty microns in length, with five to ten waves in each, and move by undulation along their long axes. They stain best with fuchsin, after extraction of the hemoglobin. Their life in the blood is usually thirty-seven days. The white blood cells are increased.

2. *The spleen* is enlarged, painful and palpable. In no other infection is the spleen so large, being increased five- or sixfold and weighing even twenty pounds. The capsule is tense, the pulp is a fluid red, the Malpighian bodies are increased, and necrotic areas and infarcts are common. Microscopically there are hyperemia, hyperplasia, spirilla and fatty,

desquamated endothelium from the veins, thus explaining the spindle cells found in the blood stream. Suppuration and rupture may occur.

3. The *bone-marrow* resembles the splenic follicles and contains granular cells, and aggregations of white blood cells.

Other Symptoms.—1. Nervous System.—Headache is frequent. The sensorium is usually free, delirium and insomnia being rare.

2. Temperature.—The temperature rises abruptly to 103° or 104°, and remains continuously high (105° to 107°) from five to seven days, when the crisis occurs. In no other disease is the defervescence so precipitate (5° to even 16°). In five to seven days the patient is well, when in 99 per cent. of cases the cycle is repeated, with usually two or three relapses (as many as seven are recorded). The temperature-curve

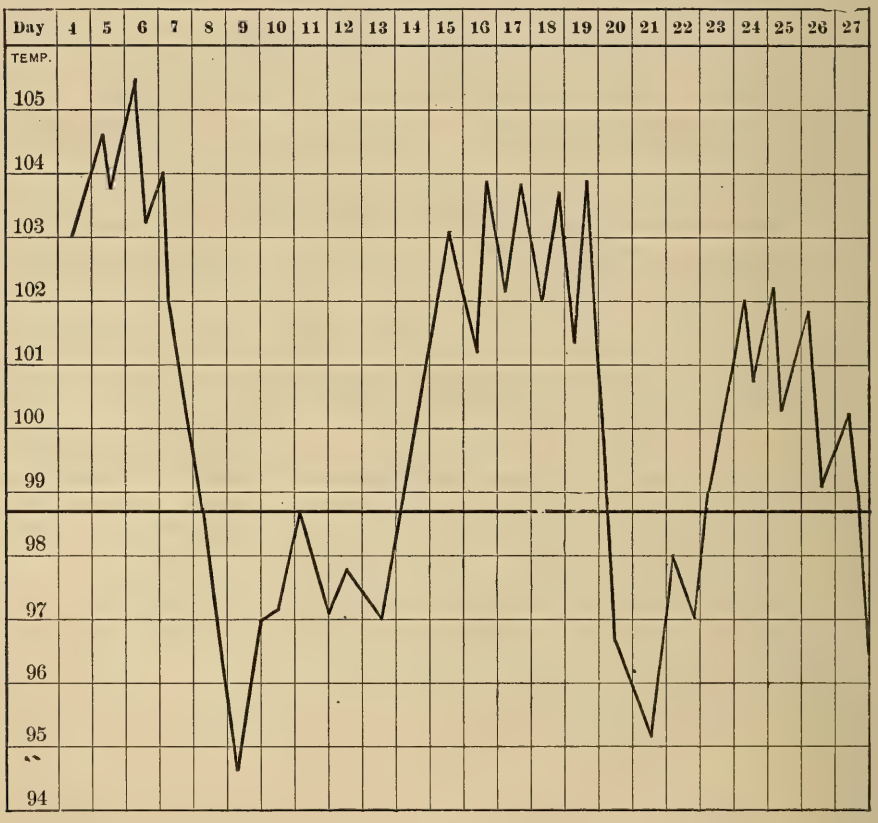


Fig. 18.—Fever chart in recurrent fever.

is diagnostic. Lysis occurs in 1 per cent. The crisis is followed by sweats, shrinkage of the spleen, disappearance of the spirillum and rapid convalescence.

3. Circulatory System.—The pulse is rapid (120 to 140), hard, sometimes irregular and slow after the crisis.

4. Respiratory Tract.—Bronchitis and increased respirations and in fatal cases hypostasis, pneumonia, glottis edema and pleurisy have been observed.

5. Gastro-intestinal Tract.—The tongue is white and later, yellowish-brown. Hematemesis, a tender, swollen liver, icterus, gall-bladder distention, bloody effusions in the alimentary mucosa, and hyperplasia of the lymphatics may be observed.

6. Genito-urinary Tract.—The urinary findings resemble those of typhoid. Nephritis is infrequent and abortion occurs in 66 per cent.

7. Skin.—The skin is usually subicteric, yet the stools remain colored. Herpes, erythema, petechiæ, desquamation and even roseolæ have been noted. The skin becomes drenched at the crisis.

Diagnosis.—The specific points are: A prevailing epidemic; onset with chill and fever; swelling and tenderness of the liver and spleen; muscular pains; abrupt crisis; apyretic interval with recurrence of the cycle, and spirilla found by blood examination.

In bilious typhoid or septic recurrent fever, there is severe icterus, hemolysis, and liver findings suggestive of acute yellow atrophy. Hemorrhages and death from collapse in the first attack are common. The colored stools and enlarged spleen are significant. Malaria, typhoid, typhus, yellow fever, acute yellow atrophy and Weil's disease, are excluded by examination of the blood.

Convalescence.—Convalescence is rapid, after four or five weeks. Death results from toxemia, heart collapse or complications. The prognosis is good, the mortality averaging 4 per cent. or 60 per cent. in the bilious type. One attack is not certain to confer immunity.

Treatment.—The treatment is that of typhoid. Pain often necessitates the use of opiates. Salvarsan has been used successfully.

KALA-AZAR.

The Leischmaniases, diseases caused by Leischman's organism, include two visceral and one cutaneous disease. Indian kala-azar (Assam or Dumdum fever) and infantile kala-azar (splenomegaly) are caused by the Leischmania donovani (Leischman and Donovan, 1903), and the oriental sore by the L. tropica.

Indian kala-azar is characterized by splenomegaly, progressive anemia and remittent fever. The protozoön is found in the spleen, liver, bone-marrow and sometimes in the blood. It can be cultivated and may be transmitted by the bed-bug. The disease begins with *fever*, which is oftenest remittent, rising two or three times daily; it recurs. Marked *emaciation* develops with abdominal tenderness, nervous toxemia, neuritic or arthralgic pains and various hemorrhages. The *skin* is *dark*, even icteric and pigmented. The *spleen* is greatly enlarged, tender and is crowded with parasites. The *liver* becomes swollen and tender from perihepatitis, is packed with parasites and frequently becomes cirrhotic. There is *secondary anemia;* there is relative lymphocytosis, usually with leukopenia. The *course* averages 6 to 9 months and the mortality is 96 per cent. The parasites are found constantly and in all stages by splenic puncture (which, however, may entail hemorrhage or rupture). Quinine hypodermically is of some benefit.

Infantile kala-azar or splenomegaly is identical with the Indian type and is found on the Mediterranean littoral and islands. It is probably transmitted by dog fleas.

Oriental sore is also known as Delhi boil, Bagdad sore, etc. It occurs on the face and exposed parts of the body as a small node, which becomes

covered with a crust. Beneath it is granulating tissue, which often ulcerates, invades the lymphatics, or perhaps the nasal mucosa—simulating lues, tuberculosis or leprosy.

TRYPANOSOMIASIS.

The trypanosoma is a protozoön found by Gruby (1843) in frogs, Döflein (1845) in rats, and later in many other animals and in man (Neprue, 1890–98). There are many species. The T. gambiense is leech-shaped; its body is granular, measures 13 to 25, by 2 to 4μ and contains a nucleus and micronucleus (centrosome); on one side and attached to an "undulating membrane" is a solitary flagellum by which the parasite moves. In cool and moist hanging-drop preparations they may live for a month and a half. Novy and McNeal cultivated the T. Lewis and Brucei. Infection is carried by the tsetse fly, flea, louse, mosquito and ticks. In man trypanosomiasis is conveyed by the tsetse fly (Glossina palpalis) or possibly by bites from infected rats; apparently the fly is the true host. Animals may be infected during coitus, by subcutaneous inoculation or by the stomach.

Trypanosoma fever was first described by Nepreu. It prevails in central and western Africa. The atrium may be present as an inflamed bite or the history of a bite may be obtained. Parasites in the *blood* may cause no symptoms, even for a year ("carriers"); parasites occur in the plasma, not in the corpuscles; some anemia is present and the large lymphocytes are increased up to 20 per cent., but the eosinophiles show no increase. Most parasites are found in the *lymph glands* which *are constantly enlarged*, especially the posterior cervical. *Fever* is irregular, subcontinuous or remittent, and lasts from three days to three weeks. An erythematous eruption is almost constant. Edema, particularly of the lower lids, weak and rapid pulse, prostration and splenic tumor usually occur. In *treatment*, arsenic (atoxyl and salvarsan), antimony (gr. 1.5, intravenously) and trypan red are apparently helpful, while quinine is without effect. Careful feeding and cardiac stimulation are indicated. The patient, as in yellow fever, should be so screened that flies can neither convey fresh infection to him nor bite him and thus infect others.

Sleeping sickness or *African lethargy*, endemic in Central Africa, is due to localization of the trypanosomes in the nervous system, causing a diffuse meningo-encephalomyelitis (Mott). Negroes are chiefly affected and slaves imported to America developed the disease; Caucasians may contract the disease.

The incubation is long, possibly years. Incipient symptoms are those of the trypanosoma fever or less often there are preliminary psychical phenomena, as epileptiform convulsions, melancholia or mania. There is a fine tremor and shuffling gait. Somnolence develops, from which at first the patient can be aroused, but which later develops into profound lethargy. The parasite is found in the blood in 92 per cent. (Bruce) and in 100 per cent. of cases in the cerebrospinal fluid, withdrawn by lumbar puncture (Castellani). Malnutrition, decubitus and sometimes

secondary infections develop, and after the convulsions deepen into coma the patient dies. The course lasts from months to several years. Treatment is usually unavailing; some 10 cures are reported. More than a quarter of a million people died in Uganda in the last few years from this affection.

OTHER PROTOZOA.

Amebiasis has been described with bacillary dysentery.

Yaws, or frambesia is a highly contagious disease, found in Africa, Asia, Central and South America and various islands. In the days of slavery it was common in the South, where cases are still found. It is due to the Treponema pertenue (Castellani, 1905); the parasite resembles the T. pallidum and the clinical course resembles syphilis. At the site of inoculation, usually on the legs or face, a papule forms, which in one week becomes an ulcer; this heals in another week; the fungoid tubercle resembles a raspberry (frambesia) and is called the initial lesion. In six weeks to three months, fever, malaise, headache and joint pains reappear—the secondary stage—in which the lesions resemble the primary or condylomata. In some instances destructive tertiary lesions develop. In differentiation from lues, the primary and secondary lesions are identical, extragenital and lack the endothelial proliferation and perivascular exudation of syphilis; the lesions are not pleomorphic, involve the mucosa only at its junction with the skin and never the viscera, brain, etc. The death-rate ranges under 1 per cent. and salvarsan is absolutely specific.

The *trichomonas* (cercomonas) is found in acid vaginal mucus and the intestines. It may initiate enteritis or cystitis. The *Lamblia* intestinalis, a pear-shaped flagellated monad, may occasion diarrhea; it has been found in pleurisy, pulmonary gangrene and bronchiectasis. The *Balantidium coli*, common in hogs, may cause dysentery (*q. v.*) in apes and man; it is found in the stool and submucosa and sometimes in the bloodvessels and lymph glands.

Coccidia may rarely be found in the liver, kidneys, bladder, spleen, intestine, peritoneum, etc. Tumors, like tubercles or mucous cysts may develop. A typhoid-like course is described.

INFECTIONS OF DOUBTFUL ETIOLOGY.

SMALLPOX (VARIOLA). VACCINATION.

Definition.—Smallpox is an acute, highly infectious disease. It begins suddenly with a chill, headache, vomiting and intense epigastric and lumbar pain, and is characterized by a typical fever-curve and by an eruption on the skin and mucosæ of papules, vesicles, pustules and crusts successively.

History.—Smallpox prevailed in China and India 1000 years before the Christian era. Galen's *pesta magna* of the second century was

probably variola. Epidemics occurred in the sixth century and during the crusades. Rhazes (Arabia, ninth century) gave us the first clinical description of the disease. Smallpox was introduced into Mexico in 1520 by the Spaniards, and three and a half million persons contracted the disease. Sydenham's classic of the seventeenth century is most accurate and reliable. In 1718, Lady Montague introduced into England preventive inoculation, which had been practised for centuries in Asia, and though the mortality fell to 1 per cent. the disease was disseminated in a mitigated form. Jenner's discovery of vaccination, in 1796, greatly lessened the terrors of variola, which in Europe alone carried off nearly half a million persons yearly.

Etiology.—The virus is unknown. Weigert and others found pyogenic organisms in the skin and elsewhere, but suppuration is due to a later mixed infection. The *cytorrhyctes variolæ* of Guarnieri (1892) is an ameba found in the blood in the incipient stage; and in the lower epithelial layers, in vacuoles in the cells; these small structureless bodies measure one to four microns and become granular and segmented.

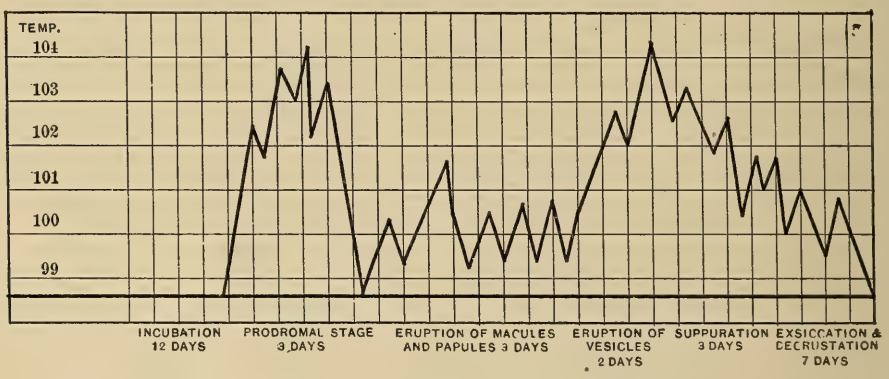


Fig. 19.—Temperature chart and stages of smallpox. (Pfeiffer.)

That the blood is infectious at an early stage was demonstrated by the infection of an individual on whom skin had been transplanted from a person in the early stages of smallpox. As variola without eruption is contagious, it seems that the secretions and excretions convey the virus. The greatest infectiousness is noted in dried pustules in which the virus localizes. Infection is direct from person to person, or indirect by a third party or by fomites. Cadavers of variola victims are dangerous. The virus is very *penetrative*, even beyond the room, building or area of the isolated case. The disease *persists* in infected communities for years.

Susceptibility to smallpox is almost universal; only 1 per cent. of people are immune. Temporary immunity is more frequent than congenital. Practically, immunity is only acquired by vaccination or a previous attack. Either sex and all ages may be affected. Mothers with the disease may deliver babes with the florid eruption or with scars. The virulence of the virus is great in aboriginal and colored races (Indians and Mexicans). Severe cases may follow mild infection, as shown in Osler's description of the Montreal epidemic, in which 3164 persons,

infected by a mild case in a Pullman-car conductor, died among the French Canadians, who oppose vaccination. Variola occurs sporadically, epidemically, or pandemically.

Atrium.—Infection occurs through the respiratory, and perhaps also the digestive tract.

Symptoms.—FORMS:

I. Variola vera	—1. Discreta, discrete.
	—2. Confluens, confluent.
II. Variola	—3. Purpura variolosa (black smallpox).
hemorrhagica	—4. Variola hemorrhagica pustulosa.
III. Varioloid	—5. Smallpox modified by vaccination.

1. VARIOLA VERA DISCRETA.—(*A*) *The incubation* is symptomless and averages twelve days (five to twenty).

(*B*) *The prodromal stage,* dating from the first symptom to the eruption, is the same in all types, and averages three days. It is shorter in children and in confluent smallpox; the longer it is, the more severe is the infection. Its intensity bears no invariable relation to prognosis, for mild smallpox may begin severely; however, if the onset is mild, the disease will not be confluent or hemorrhagic.

The *invasion begins acutely* with chill, fever, rapid pulse and respiration, nervous toxemia, intense headache, backache and vomiting. (*a*) The *chill* is severe, and frequently is repeated. In children it is often absent. (*b*) The *initial fever* rises suddenly to 103° or 104°, and reaches its maximum on the second or third day. The *pulse* is rapid (120) and full. The more frequent respirations (30 to 36) constitute the so-called toxemic, "cerebral respiration." The *skin* is red, hot (*calor mordax*) and dry; there may be sweating in the discrete variety and in favorable cases. (*c*) *Nervous toxemia* is evidenced by the benumbed sensorium, depression, restlessness, insomnia, delirium especially in alcoholics, or convulsions and meningeal symptoms in children. *Headache* appears with the chill and is frequently frontal or temporal. When severe, it may suggest meningitis, especially when accompanied by vomiting and neckache. (*d*) *Backache* appears with the chill, and lasts one or two days. It occurs in nearly all cases, though less frequent than headache and vomiting. It is severe, like lumbago, or actually agonizing. The "veritable paraplegia" described by Trousseau is only immobility due to toxemic pain. It is rare in other fevers likely to be confused with variola. Pain is sometimes observed in the intercostal nerves, sciatics, muscles, joints, pharynx, larynx or heart. (*e*) *Vomiting* and early epigastric pain are constant in children. The spleen is not often enlarged in the initial period. It is often tender, as are the liver and epigastrium. (*f*) The *initial* or *prodromal eruptions,* present in 10 per cent., have considerable diagnostic value. They are commonly limited to the lower abdomen, inside of the thighs, axillæ and sometimes to the extensor surfaces of the knees and elbows. There are two varieties:

1. The *morbilliform variety* (roseola variolosa) appears during the second day on the face and body as small, sometimes crescentic maculæ and usually lasts one day; it is hyperemic but may be purpuric. Most often seen in varioloid, it is a favorable prognostic.

2. The more rare *scarlatinal form* (erythema variolosum) occurs on the first day. It is less punctate, extensive and bright than the scarlet fever rash. If it is purple and purpuric, it is almost always variolous. It occurs on the lower abdomen, thighs and knees, forming a triangle with base upward when the legs are together. Sometimes it is found over the axilla or shoulders, and is seen in women more frequently than in men. These areas are rarely invaded by the pustular eruption. Some writers speak of an erysipelas-like and urticarial, prodromal rash.

(*C*) *The eruptive stage* comprises several serial substages.

1. *Macules and papules* (*stadium maculosum et papulosum*) occur on the fourth day, and advance for three days, beginning on the forehead near the hair and on the cheeks, with itching and burning macules, resembling flea-bites, which soon become papules. The papules are reddish, discrete, circular, elevated, and hard or "shotty." A day later they appear on the body, and on the next day, on the extensor aspects of the extremities; *i. e.*, the papular eruption advances for three days, and is descending. The papules are thickest on the face and trunk. The eyelids show collateral edema. If the eruption appears on the second day, the confluent type may be expected; if on the third day of the disease, the discrete type.

Weigert holds that the first changes are necrobiotic or diphtheroid in the rete Malpighii followed by inflammation in the necrotic areas. Other authorities consider inflammation the primary, and necrosis the secondary process.

Unlike measles and scarlatina, *the fever remits when the eruption appears*, and the backache and headache improve. In three days the fever reaches normal by irregular steps. The lower the fever falls in this remission, the lighter is the type of smallpox.

2. In the stage of *vesiculation* (*stadium vesiculosum*), lymph gathers in many but not in all of the papules. This occurs on the seventh day of the disease and lasts about two days. The vesicles are multilocular, since they occur in the cells of the rete Malpighii. Umbilication occurs in the centres of many vesicles, caused by elevation of their edges by infiltration and by the follicles in their centres; it corresponds to the area of primary necrosis and is suggestive but not pathognomonic of smallpox. On the soles and palms the fluid is situated deep in the resistant tissues.

3. The stage of *suppuration* (*stadium pustulosum*) or maturation begins on the ninth day with clouding of the vesicles and inflammatory congestion about them (areola); it advances for three days. The pustules become opaque, then yellow, and the thick pus obliterates the umbilication and renders the eruption globular. The areola or halo becomes more vivid and inflammatory edema may result from fusion of the areolæ. This causes increased tension, deformity especially in the face and parts where the skin is most loose, closure of the eyes, occlusion of the nose, and great tenderness and pain. Pustulation follows the descending direction of the initial eruption and the pustules are thickest on the extremities and head. On the volar and plantar surfaces they are less prominent and there is less edema. The pustules evacuate spontaneously,

especially on the face, but may dry without rupture. Various stages of the eruption may be observed in different parts of the body at the same time; not all vesicles become pustules. The skin exhales a peculiar, penetrating, offensive odor, and bed-sores may develop.

Anomalies are variola *siliquosa*—air in the pustules, and very large eruptions three-quarters of an inch in diameter. The pus originates from the vessels of the papillæ which undergo pressure-atrophy, or even necrosis.

Mucous membranes: The pock eruption occurs mostly in the mouth and nasopharynx; the successive stages of papulation, vesiculation, pustulation, etc., may develop, but less typically than in the skin; erosions, stomatitis, glossitis, phlegmonous angina, etc., may result. The eruption is found in very rare instances in the Eustachian tube, eye, respiratory, alimentary and genito-urinary tracts.

With pustulation there is a gradually rising *secondary fever*, due to the smallpox virus and mixed pyogenic infection, and proportionate to the degree of pustulation. In variola discreta the temperature does not remain high more than twenty-four to thirty-six hours, with morning remissions. A marked *leukocytosis*, of 10,000 to 34,000 attends the secondary fever and parallels the degree of infection. The lymphocytes are increased (to 66 or 50 per cent.), the polymorphonuclears decreased (to 50 or 33 per cent.), and the myelocytes may reach 12 per cent.

Delirium with suicidal tendencies, albuminuria, acute exhaustion and heart paralysis may develop.

(*D*) *The Stage of Involution.—Exsiccation, decrustation* begins on the twelfth day. It occurs in the same descending order as the florition and suppuration, with decrease in the edema, redness and pain, but with an intolerable itching. Crusts form, and remain longest on the soles and palms where they may form complete casts of the parts. Mucous erosions heal more readily. The hair falls out, and by the end of the second week the fever reaches normal. Fever at this stage indicates some complication. The higher the granulating surface, the deeper is the cicatrix. Scars occur when the true skin has been involved, and are seen chiefly on the face, scalp, palms and soles. Sydenham held that discrete smallpox rarely leaves its mark but others maintain that suppuration and cicatrices are necessary stigmata of variola vera. Scar formation lasts three or four weeks. Complete convalescence follows the disappearance of the last crust.

2. Variola Vera Confluens.—This malignant type was more frequent in prevaccination days, and is now seen largely in persons unvaccinated or vaccinated but once. The initial stage is violent. The headache and backache are agonizing. The status typhosus and gastric symptoms are marked, the fever remission is slight or absent, or if the fever remits there is little or no improvement in the symptoms. The exanthem is precipitate both in its development (on the second or third day), and in its extension. The earlier the exanthem in variola, the greater is the probability of confluent smallpox. The eruption becomes confluent especially on the face and head, and sometimes on the hands and feet; on the body and limbs it is largely discrete. With fusion of the eruption, great inflammatory edema appears, swelling and erosion of the

mucous membranes, closure of the eyes, keratitis and obstruction of the nose develop. The general aspect is desperate. High fever, high pulse, rapid, even irregular or stertorous breathing, delirium, albuminuria, persistent nausea and vomiting, great thirst, salivation in adults and diarrhea in children, husky voice, enlarged cervical glands and often parotitis, are present. As to prognosis, Sydenham says that "if upon the face the pustules are as thick as sand, it is no advantage to have them few and far between upon the body." Death occurs from acute toxemia (*variola typhosa*), with hyperpyrexia and cardiac asthenia usually within a week; or, if the patient lives longer, from septicopyemia, phlegmon, gangrene, pneumonia and nephritis. The patient usually dies if the eruption does not appear. Recovery from variola confluens is infrequent.

3. Purpura Variolosa.—This is the black smallpox—*smallpox with primary hemorrhage* in the initial stages. It is the worst type, results almost uniformly in early death and is important because of the difficulty in its diagnosis. It is seen most frequently in the unvaccinated. The incubation is short (six to eight days), the invasion is stormy, with agonizing lumbar pain, great prostration, a rapid, small and soft pulse, and disproportionately increased respiration. The initial epigastric pain and vomiting may last until death.

On the second day (or even on the first), there appears a plum-colored eruption, with brick-red, purple or inky ecchymoses, especially about the eyes. The aspect of the discolored and swollen face, and ecchymotic, sunken and alert eyes, is desperate. Hematuria is the most common of visceral hemorrhages. Other forms are hematemesis, enterorrhagia, epistaxis, metrorrhagia and hemorrhage from the gums, ears and bronchi, sometimes accompanied by gangrene of the pharynx. Tympanites and albuminuria may be noted. Miscarriage is frequent. The disease does not usually reach the real eruption of variola, or, at the most, only the papules are observed, because it is fatal in four or five days. The mind often remains lucid to the end. In most cases the fever is not high, but in the most fulminant type it reaches 105° or 106°, with collapse, coma and death in a few hours, even before cutaneous hemorrhage is seen, although internal hemorrhages are found at autopsy. The "blood dissolution" of the older writers is a mycotic coagulation thrombosis.

4. Variola Pustulosa Hemorrhagica.—Hemorrhage after the eruption appears (*secondary hemorrhage*) is more common than primary hemorrhage. This form occurs in weakly or alcoholic subjects. The initial stage is severe, and secondary hemorrhages occur into the vesicles or pustules, or into the lower parts of the body or there may be "blood dissolution," epistaxis, hematuria and metrorrhagia. Adynamic manifestations are usual, and the outcome is almost always fatal, though hemorrhage at the vesicular stage may be followed by rapid abortion of the rash and recovery.

5. Varioloid.—Varioloid (*variola modificata s. mitigata, variolois*) is *variola mitigated by vaccination.* The more marked the initial eruption, which is often morbilliform, the less the number of pustules. Varioloid begins suddenly. The fever is atypical; it may reach 103° and usually falls with the specific eruption, reaching normal on the fourth day;

secondary suppuration is rare or absent. The eruption varies in amount, being present on the trunk only, or being typical chiefly on the hands and feet. It may be absent entirely. It may not be umbilicated and may desiccate rapidly without rupture; scars are rare. The mucous surfaces are little involved. The entire course is shorter, more irregular and rudimentary, and more benign than in variola vera.

Complications and Sequelæ.—The many complications and sequelæ of variola embrace severe early toxemia and later, secondary infections.

1. The Nervous System.—*Delirium* is caused by cerebral toxemia, alcoholism or hyperpyrexia. Convulsions are frequent in children. The unusual *psychoses* are generally asthenic. Meningitis, encephalitis, embolism, cerebral softening, and abscess are occasional complications. Myelitis and neuritis are exceptional sequels.

2. The Vascular System.—Circulatory changes are more rare. The heart muscle may soften and dilate, microscopically revealing segmentation, cloudy and fatty conditions, coronary endarteritis and myocarditis; a systolic bruit may be heard at the apex, with weak first tone and tachycardia, as in typhoid. Aortitis, pericarditis, ulcerative endocarditis and phlebitis are infrequent.

3. The Respiratory Tract.—Edema of the larynx and perichondritis are usually fatal, for laryngeal anesthesia promotes inhalation pneumonia. *Bronchitis* is invariable in severe cases. *Pneumonia,* usually lobular, occurs in 60 per cent. of fatal cases; lobar pneumonia, lung abscess, embolism and gangrene are uncommon.

4. The Digestive System.—Glossitis, retropharyngeal abscess, parotitis, noma, phlegmonous esophagitis, and hemorrhage in the throat are ominous though uncommon complications. The early vomiting rarely persists. Diarrhea is not common, but a simple form is seen in children and a dysenteric form occasionally in adults. Peritonitis, retroperitoneal cellulitis, mediastinitis and perineal abscess are infrequent. The alimentary tract, with the exception of the rectum, is commonly exempt from the eruption. The liver may suffer cloudy or fatty change, focal necrosis or diffuse hepatitis. The spleen is usually swollen and soft.

5. The Genito-urinary Tract.—Albuminuria occurs early in 25 per cent. of cases, during the eruption, suppuration or convalescence. Nephritis now and then develops during the fever or in convalescence; it is often glomerular. Pyelitis, ovaritis and vulvar gangrene have been observed. Menorrhagia is common in the early stages of onset or in hemorrhagic forms, and abortion from hemorrhagic endometritis is the rule in pregnant women. Orchitis variolosa is very frequent; the multiple inflammatory foci lie mostly in the connective tissue, but are rarely suppurative.

6. The Special Senses.—The eye is involved in 1 per cent. Pox in the conjunctivæ are rare, as are conjunctivitis, keratitis, iridochoroiditis, metastatic panophthalmitis, etc. Otitis and suppurative thrombophlebitis may occur.

7. Locomotor System.—Muscular abscess and arthritis may occur during desiccation. Arthritis is more often suppurative than serous,

and is but one feature of the secondary sepsis. In the bones there are almost invariably hemorrhage into the marrow, hyperplasia of the marrow cells, and multiple foci of necrosis or non-suppurative osteomyelitis (in 72 per cent., Chiari).

8. Skin.—Decubitus, erysipelas, local or pyemic abscesses, desquamation, acute gangrene, hemorrhages into existing exudates, and less often into the viscera, also occur.

Diagnosis.—The diagnosis of smallpox is usually easy when chill, fever, headache and vomiting occur after exposure to infection. Vaccination and revaccination scars must be searched for. Mistakes in the diagnosis of the first cases of an epidemic or in abortive or purpuric cases, are often inevitable. Hemorrhagic scarlatina or measles sometimes causes confusion; in the former the mucous membrane hemorrhages are less frequent than in smallpox. The initial suggestive scarlatiniform eruption has been unduly emphasized; the prodromal eruptions *plus* purpura are highly suggestive. An acute onset and high temperature are significant, although acute onset and high fever may occur in typhus, pneumonia, scarlatina and influenza; lumbar and sacral pain is more common in smallpox. Intense frontal headache, severe backache, vomiting, chill and fever are sufficient grounds for a tentative diagnosis and isolation but the diagnosis is uncertain until the eruption is seen. In the *variola sine exanthemate* (*febris variolosa, variola sine variolois, varioles frustes*) the diagnosis must be made from the history of exposure, the presence of an epidemic, fever, pain, delirium, possibly the initial rash, and the absence of secondary fever because there is no suppuration. An eruption which appears on the third day of the affection, accompanied by a fall of the fever and euphoria, is most important. Jenner described an early cutaneous inflammation in the vaccination site, in those who are immune to smallpox; this diagnostic phenomenon was later described by v. Pirquet as allergistic (see diphtheria therapy, allergy).

Varicella		Variola.
Vaccination and smallpox do not protect.		Smallpox may closely resemble chicken-pox; mild cases especially misleading.
Age: usually before puberty; it may occur in adults.		Usually after puberty (numerous exceptions).
An initial stage is practically absent.		It is severe, even in mild cases.
Temperature: no remission with onset of rash. White cells normal or decreased.		Typical remission and a secondary fever (except in varioloid). Leukocytosis (mononucleosis).
Prodromal rash: very exceptional.		Frequent (10 to 16 per cent.).
Vesicles and roseolæ simultaneously and in crops; very rarely shotty.		Never in crops; first macules, then hard, shotty papules, vesicles, etc.
Rash evolution: much more rapid; vesicles on the first or second day.		Much slower; vesiculation on the seventh day (see Pfeiffer's table).
Eruption is universal; successive crops; most abundant and characteristic on back; begins on body; less on face, scalp, mucosæ, hands and feet.		Development progresses downward—face first, then hands and feet; less on trunk.
The vesicle is superficial, and the fluid transparent. (Wyss observed confluent eruption and umbilication).		The fluid is pearl-colored, not transparent, and has a thicker covering.
Areola (halo) is usually absent.		Marked.
Involution is rapid.		Tardy.

Mistakes may be made even by smallpox experts, but attention to the history, somatic findings and course, rather than to the eruption, prevents many disastrous results.

MEASLES———*vs.*———VARIOLA.

Both present the same prodromal duration; in each the eruption begins on the face; according to some observers, simultaneously on face and trunk.

Age: eminently a childhood affection.	Chiefly in adults.
Catarrhal stage characteristic.	Catarrh, if any, rudimentary.
Fever: present in catarrhal stage, but low before exanthem, and rises with its appearance.	High before eruption and falls with the exanthem even in severe cases.
Koplik's spots on buccal mucosa; bluish-white surrounded by red.	Absent. Early papular eruption on palate, etc.
Eruption later with rise of fever.	Possibly an initial morbilliform eruption with the primary fever.
Papules "grouped," crescentic.	Never so.
Papules remain relatively flat, smooth, velvety.	Change into vesicles, pustules, crusts, scars; hard and shotty.
Grisolle's sign absent and the eruption disappears under pressure.	Grisolle's sign present; *i. e.*, persistence of the smallpox papule on stretching the skin, distinguishing it from scarlatina, varicella, etc.
Areola (halo) absent.	"Halo" present.

SCARLATINA———*vs.*———VARIOLA.

Rash on the second day, angina, strawberry tongue, desquamation, glands, nephritis, etc. (See cardinal symptoms of scarlet fever.)	Rash later; slow evolution of different phases. No angina; no strawberry tongue; the initial scarlatinal eruption is not punctate and less bright.
No double elevation of fever curve.	Double summit to curve.

In *drug rashes* (copaiba, etc.), the pustules occur largely on the face, head, arms and forearms. In *syphilis* the chancre, glands, polymorphic rash, and the moderate fever, are easily distinctive.

The lumbago form of *acute nephritis*, acute myelitis or erythema, septicemic rashes and acne only superficially resemble variola.

Glanders may cause more hesitation in diagnosis. Its features are pyrexia, pains, papules, pustules, nasal discharge, the bacteriological findings, and general symptoms severer than the skin eruption.

Prognosis.—The prognosis depends on (*a*) previous *vaccination*, the disease being fatal in 50 per cent. of those unprotected by vaccination. Smallpox is rarely fatal in persons vaccinated within five or six years; only 8 per cent. of those vaccinated once die, and only 4 per cent. of those vaccinated twice. (*b*) On the *genus epidemicus:* In the last American epidemic the death-rate was but 1.6 per cent. (*c*) On the *type*, being practically 100 per cent. in purpura variolosa; nearly 100 per cent. in variola pustulosa hemorrhagica; 60 per cent. in variola confluens—especially fatal in the first decade of life; 25 per cent. in variola discreta—particularly dangerous in those over fifty or under ten years, in pregnancy, the puerperium, in weakly and alcoholic subjects; 1 per cent. in varioloid; recovery usual in *variola sine exanthemate.* (*d*) On *individual symptoms:* (i) Incubation is short in unfavorable forms, especially in purpura variolosa. (ii) Initial symptoms are mild, chiefly in varioloid, but may be severe in both the benignant and malignant

forms. (iii) The initial rash is usually a partial erythema in mild forms of smallpox; in general, morbilliform eruptions occur in varioloid. Scarlatiniform eruptions occur in rather severe forms and a universal scarlatinal hemorrhagic eruption, especially in the groins, occurs particularly in purpuric types. (iv) The danger is proportionate to the amount of the eruption on the face and hands. A slow, typical exanthem indicates variola discreta, and a precipitate atypical eruption presages variola confluens. (v) A prompt fall in temperature after the eruption is favorable. A slow, complete fall occurs chiefly in the severe forms. Accession of fever after pustulation is ominous. Hyperpyrexia or temperature collapse during suppuration is a sign of imminent death. (vi) In the stage of suppuration the danger parallels the height of the fever and the amount of efflorescence. Enlargement of the spleen is ominous.

Treatment.—1. PROPHYLACTIC VACCINATION.—*Vaccinia.*—Protection by cow-pox vaccination was discovered by Jenner, who published his results in 1796, although certain of his evidence twenty years earlier; the idea had been long prevalent among farmers that cow-pox afforded immunity to smallpox. Professor Waterhouse, of Harvard, in 1800, introduced vaccination into this country.

The nature of cow-pox or vaccinia is still disputed. It is probable that cow-pox and horse-pox are human smallpox modified by transmission through animals. Variola inoculated in the cow produces typical vaccinia. The other view is based upon Chauveau's experiments, which conclude that the two affections are distinct. The bacteriology of vaccinia is also uncertain; micrococci, bacilli and amebæ (Guarnieri's *cytorrhyctes vacciniæ*) have been found.

Usual Symptoms.—During the incubation period of three days, there may be slight local traumatic reaction. On the third day a hard papule develops, surrounded by a delicate halo. The papule enlarges, and on the fifth (or sixth) day shows Jenner's vesicle filled with clear serum and umbilicated in its centre. In the indurated and painful skin around it the magnifying lens shows many minute vesicles by the eighth day. By the tenth day the vesicle becomes purulent and the surrounding tissues hard and painful. By the eleventh day a small central crust appears, which, by the end of the second week, covers the entire vesicle, when the adjacent cellulitis regresses. In one or two weeks the ulceration, decrustation and granulation are usually complete, and a scar or pit remains, which pales with time. Constitutional reaction is greatest in children. The fever in most cases appears on the fifth day, and lasts a few days, with leukocytosis, restlessness, headache or backache. Swelling, depending on the site of inoculation, occurs in the axillary or inguinal glands. School physicians should not accept certificates of recent vaccination but should watch the evolution of the vaccination.

Unusual Symptoms.—The vesicles may run a course shorter by half than the usual one, or the eruption may be tardy and atypical. In both instances revaccination is indicated.

Complications.—(1) *Erythema,* urticaria, roseolæ or erysipelas (third to eighth day); in Germany, in 1895, two deaths (from erysipelas) occurred in two and a half million vaccinations. To offset this theo-

retical risk there is the almost universal susceptibility to smallpox in all unvaccinated individuals. (2) Accessory vaccinia near the original inoculation; or generalized vaccinia, on the trunk and extremities; vesicles sometimes continue to develop for over a month, and in weakly subjects are very exceptionally fatal. (3) *Secondary infection* in the first week or two, either local (deep ulceration, gangrene, lymphangitis or lymphadenitis), or systemic (sepsis, pericarditis, meningitis and parotitis). In 95 reports, McFarland (1903) collected 68 certain cases of *tetanus*. Albuminuria is infrequent. (4) *Later infections:* Tuberculosis is probably never inoculated; it can be prevented by the Belgian method of not using the lymph until the animal has been killed and found healthy. In some cases a latent tuberculosis in the patient may be awakened by vaccination, which seems to act like tuberculin. There was danger of inoculating syphilis when human virus was used; in fifteen days a chancre, and in eight weeks more the secondaries appeared. Syphilis may be inoculated accidentally and also intentionally, as in the oft-cited case of Dr. Cory. In exceptional cases, latent syphilis may be aroused by vaccination. There are no clear cases of inoculation of leprosy. Howard found the ray fungus 24 times.

Age.—Children should be vaccinated in the third or fourth month, but never later than the first year.

Technique.—The arm or leg should be washed, but no antiseptics should be employed, lest they neutralize the virus. A fold of skin is raised and rubbed with sterile gauze or scraped with a knife held almost parallel to the skin, until the epidermis is removed and slight oozing of lymph appears. The virus must reach the lymph stream, and therefore blood should not be drawn, as coagulation somewhat inhibits "taking." The vaccine should be slowly rubbed over and into the abraded skin. Only after the virus has thoroughly dried should the spot be covered with a sterile dressing or cloth held in place by adhesive plaster. Boric, carbolic or bichloride gauze must not be used.

Lymph.—Vaccinia virus is harmless if it is collected in the vesicular and not in the pustular stage, and preserved in sealed sterile tubes with three parts of glycerin. This obviates pyogenic infection, preserves the virus four months, and is more economical. Human virus must be taken from a perfectly sound child, carefully examined for tuberculosis and syphilis, and taken on the seventh day from the clear, unbroken vaccinia vesicle, which must be free from pus; no blood should be withdrawn.

Results.—The successful vaccination, accompanied by typical papulation, vesiculation, pustulation, crustation, cicatrization, and by local and often general reaction, confers immunity in almost all cases. Vaccination is the greatest triumph of prophylaxis. The nature of the immunity conferred is still disputed, but, unlike the immunity of diphtheria, it is probably due to actual tissue change. Vaccination immediately after exposure is thought to prevent or modify an impending attack. Partial immunity is evidenced by varioloid (smallpox mitigated by vaccination). Immunity is not life-long, as Jenner thought, nor is it absolute. In some few cases, the individual is permanently protected by

one vaccination, but protection lasts on an average ten years, after which 60 (to 90) per cent. can be successfully revaccinated once or twice. Conversely, the child should be revaccinated in his tenth to twelfth year, and if the vaccination does not "take," it should be repeated. The reaction, fever, induration, and size of the vesicle and scar, are less in revaccination. As immunity is more enduring when more than one inoculation has been made, the Germans vaccinate two areas or even four or six. The general beneficence of vaccination needs no multiplied statistics. There has been no epidemic of smallpox in Germany since 1875. Epidemics prevail in communities which are least protected, and conversely, they disappear with systematic compulsory vaccination.

2. Other Methods of Prophylaxis.—Isolation hospitals and stations are indispensable. Isolation is necessary from the invasion until disappearance of the last crust. The dead body is dangerous and the funeral must not be public. The clothes used by the patient must be steamed, and other articles must be washed with bichloride of mercury and fumigated with formaldehyde vapor; Schoch's method has the advantages of requiring no special form of apparatus, of cheapness, and freedom from danger of fire.[1] Disinfection of the face, hands, beard and hair of attendants by bichloride solution is imperative. The family should be isolated for sixteen to twenty days.

3. Actual Treatment.—There is no specific treatment. The *hygiene* and *dietary* are the same as in typhoid. For *headache*, the ice-bag, opium and hydrotherapy are employed. *Pain* is relieved by opiates. The initial *vomiting* is difficult to overcome, but its duration is fortunately short. Ice-pills, lime-water, iodine, chloroform, morphine, carbolic acid and bismuth may be used as in typhoid (*q. v.*); mustard plasters may necrose the skin.

Early hydrotherapy is important in *fever*. It lessens the dermatitis; when the rash develops, baths are contra-indicated.

Delirium may be relieved by hydrotherapy, by chloral, bromides and hyoscine. Morphine, in half-grain doses, is invaluable to lessen *itching* and pain and conserve metabolism. The patient, if delirious, may escape or injure himself. A classmate of the author killed himself by jumping through a window. Patients should never be confined by straps, lest serious skin necrosis ensue.

The *eruption* may be pricked and cauterized with silver nitrate, treated with bichloride or boric compresses or covered with mercurial plaster; 1 per cent. carbolic vaseline is the best, because it counteracts the particularly offensive odor, but should be used on the face only, since general use may cause carbolic poisoning. These measures do not prevent cicatrix formation, but relieve dermatitis and itching. Protecting the

[1] The following are required: Good quicklime in lumps, commercial sulphuric acid and ordinary 40 per cent. formaldehyde solution. For every 1000 cubic feet of space, the size of the average room, one pound of commercial formaldehyde (40 per cent.), one-half pound of sulphuric acid and three pounds of quicklime are required. The acid and the formaldehyde are first mixed in an earthenware vessel by pouring the acid into the formaldehyde. Then the lime is placed in a shallow vessel in the centre of the room. All openings to the room are carefully closed, the mixture is poured on the quicklime, and the operator leaves the room. The rooms should remain closed for from five to eight hours.

face from the light with wet compresses seems to lessen the pitting. The crusts should be kept soft with olive oil. The hair should be cut short and the eyes douched frequently with boric acid solution. The mouth, larynx, circulation, skin and diarrhea should be treated as in typhoid fever.

For sepsis, quinine (*v.* sepsis) and alcohol (*v.* typhoid) are given by the rectum, since dysphagia is always present. Egg-nog properly prepared will not irritate the throat. During convalescence the crusts should be carefully washed, and the patient may be considered safe only when the skin is clear of the last scab.

CHICKEN-POX (VARICELLA).

Definition.—An acute specific febrile contagious disease, chiefly of childhood, characterized by an exanthem of vesicles.

History.—Varicella was recognized by Ingrassias in 1553, Heberden in 1767, and by Trousseau, as a disease distinct from smallpox.

Etiology.—It is transferable by direct inoculation, personal contact, by the air or by a third person. It is infective while crusts remain. Epidemics of varicella may coincide with, precede, or follow, smallpox. Smallpox does not prevent an attack of varicella nor is the converse true. The disease occurs chiefly in epidemics. It occurs chiefly in the first year of life, or, according to others, 66 per cent. of cases occur under the sixth year. The actual cause is not determined. Guttmann described a staphylococcus; and Pfeiffer and Guinon, a protozoön.

Symptomatology.—1. Incubation.—This stage, like that of measles, lasts thirteen or fourteen days.

2. Stage of Prodromes.—Prodromal symptoms are rare, or they last but twenty-four hours. Severe intoxication is rare, but the following have been observed: delirium, convulsions, vomiting, hyperthermia, angina, conjunctivitis, dysphagia, muscle pains, bloody vomiting and stools, and an initial erythema (usually scarlatiniform, rarely morbilliform, in the fifteen cases recorded).

3. Stage of Eruption.—The exanthem appears usually in one day, as red *macules* or *papules*, comparable to the typhoid roseolæ, slightly elevated, disappearing on pressure, lenticular and becoming *vesicular*. The vesicles mature within a day, are very superficial, and do not occupy the whole roseola, but leave a slight areola about them, which is *not inflammatory* as in smallpox. In some cases the areola may be absent, the eruption appearing like drops of water sprinkled on the skin. Though usually discrete, confluent eruptions are described. The vesicles may be few or numerous; Thomas counted 800 in one case. The eruption is mostly elliptical or circular. The vesicles may be very large, and, in exceptional cases, resemble pemphigus.

There is no point of predilection for the eruption, and no part is exempt, though most vesicles appear on the trunk, fewer on the face, arms and legs, and the least number upon the hands and feet.

The vesicle, clear at first, becomes turbid within two days. *Pustulation* and hemorrhage into the vesicle rarely occur. The vesicle may con-

tain air. Umbilication is rare; crusts may simulate umbilication. The vesicles of varicella are not always unilocular.

Occurrence *in crops* is frequent, when the skin shows simultaneously macules, vesicles and crusts. Yellowish-brown crusts form on the third and fourth days and gradually disappear. Distinct scars may result from scratching or infection, and a few cicatrices are usual in every case of varicella, but they are smooth, white and some disappear with time.

Enanthems are not frequent; vesicles may easily be overlooked on the palate; breaking down, they resemble aphthæ. On rare occasions they are seen on the tongue, larynx, prepuce or labia.

Fever.—The fever is slight, atypical and not parallel to the eruption. Fever as high as 106.8° is recorded. It is very rare in the prodromal stage, but may occur with profuse eruptions, or with successive crops; its persistence suggests complications.

General Symptoms.—These sometimes include dysphagia, cervical and submaxillary swellings, tracheitis, bronchitis or albuminuria.

The entire course of an uncomplicated case lasts one or two weeks.

Complications and Sequels.—These are usually dismissed with a few words. Nephritis is generally tubular, and occurs within two weeks after the rash declines. Though usually mild, fatal cases have been reported, as well as some eventuating in chronic nephritis, cardiac hypertrophy and uremia. Gangrene of the extremities, scrotum or eyelids has been noted in delicate or tuberculous children. Infrequent complications are pleurisy, pneumonia, miliary tuberculosis, erysipelas, glandular swelling, pemphigus, hemiplegia, polyarthritis, otitis, neuritis, etc.

Diagnosis.—The diagnosis is very easy if the case is seen early. Differentiation from smallpox (page 254). In *pemphigus* the vesicles are larger and run a slower course of weeks or months. *Miliaria rubra* are preceded by sweats, occur chiefly on the covered parts, have acid contents, are smaller than the varicellous eruption and disappear more rapidly. Sometimes consideration must be given to molluscum contagiosum (soft, umbilicated tumors with white, granular contents); prurigo varicelliformis (with crop-like eruptions at longer intervals); eczema vesiculosum, which always occurs with great itching; herpes, always appearing in groups and often along nerve trunks; varicellæ syphiliticæ, and medicinal rashes from cantharides, bromide, arsenic and iodide.

Treatment.—Active treatment is rarely indicated. Kling reports success from vaccination.

SCARLET FEVER (SCARLATINA).

Definition.—A specific infective disease of unknown bacteriology, characterized by sudden onset, fever, diffuse exanthem and angina.

History.—It was recognized by Ingrassias and Coyttar in the sixteenth century, but first fully described and differentiated from measles by Sydenham (1660). It was introduced into America in 1735.

Etiology.—The etiology is unknown. The virus of scarlet fever produces severe necrosis, but no suppuration. Mallory describes a proto-

zoön, the *Syclaster scarlatinalis*. The *streptococcus* is the most important factor in the production of complications and in their mortality. Schleissner found it in the blood in 55 per cent. It is found in about 70 per cent. of the fatalities, *i. e.*, it is the cause of the malignancy of the disease, but not of the disease itself.

Susceptibility is not universal—and only 38 per cent. of children and 5 per cent. of adults exposed to infection acquire the disease; 90 per cent. of cases occur under ten years of age. It is rare in the first year of life.

So-called "*surgical and puerperal scarlatina*," noted in 1864 by Paget, is in the vast majority of cases, sepsis, erythema or a drug rash. Alice Hamilton (1905) collected 174 cases from the literature, and concluded that these forms are mostly septic, or that the scarlatina is merely accidental.

The *virus* circulates in the blood, whence the possibility of fetal infection. It was once held that the virus was disseminated during desquamation, but oral, nasal or otitic discharges probably perpetuate infection, perhaps months after scaling is complete. In no other disease is the virus so *tenacious;* it may persist ten years on clothes, furniture, etc. Light are as contagious as severe forms. Inoculations from the living subject have occurred and Leube acquired the disease by an autopsy cut. The atrium is probably the throat. Monkeys have been inoculated.

As to the degree of *infectiousness*, smallpox ranks first, measles second and scarlatina third. Infection may be spread by servants, physicians, any third person, and by means of toys, books, clothes, milk and schools; the mode of infection is often obscure. One attack usually confers *immunity*, but second, and even third, attacks are known. Some individuals temporarily or permanently resist infection. Scarlet fever occurs more often in the autumn and winter. Scarlatina is observed more in the cities, and measles prevails more diffusely through the country. Scarlatina is more permanent than measles. When it becomes epidemic it spreads widely and with increased mortality.

Scarlatina sometimes occurs with other infections, such as diphtheria or measles, and less often with varicella, pertussis, etc.

General Symptomatology.—1. INCUBATION.—The incubation, with no noticeable symptoms, lasts from two to four days (ten to fourteen days, McCollom).

2. INVASION.—The invasion lasts one day. Symptoms *begin suddenly*, perhaps with a chill, followed by (*a*) *vomiting*, early and characteristic, which occurs in scarlatina more often (75 per cent.) than in other diseases of childhood except pneumonia; (*b*) headache, *convulsions* and delirium; (*c*) *sudden elevation of temperature*, with disproportionate *pulse-rate*, 120 to 150, dry, burning skin and febrile urine; (*d*) burning and *pain in the throat*, dysphagia and *intumescence of the cervical glands*.

3. EXANTHEM.—The eruption appears on the second day, first over the clavicles, upper trunk and neck and then on the extremities. The skin about the mouth is pale. The exanthem pales on pressure. It first consists of small red spots which fuse as the skin swells, and result in

an intense diffuse lobster-colored erythema; it lasts four to six days. The tongue shows swollen red papillæ—"*strawberry tongue.*" On *the tonsils* and palate a grayish-yellow coating and *confluent spots* appear. The nostrils slightly swell, and the conjunctivæ redden. Some rales over the larger bronchi, a slight cough, swelling of the cervical and other glands, and sometimes albuminuria, may be noted.

4. Desquamation.—Scaling begins first on the face, from the sixth to ninth day and lasts several weeks. The glandular swelling disappears, the fever falls by lysis, and convalescence is established unless complications intervene.

Special Symptoms, Complications and Sequels.—1. Fever.—The fever is less typical than in smallpox and measles. There is an initial rapid rise to 104° or 105°, with slight remissions and gradual subsidence when the rash is mature.

In the most severe types there may be low fever or no fever at all, or it may rise to 107°. A close parallelism is generally noted between the abundance of the exanthem and the height of the fever. The temperature should be taken for one month after the rash disappears, lest such complications as sloughing of the throat, pleuritis, endocarditis, pericarditis, synovitis or adenitis be overlooked.

2. Eruption.—Typically, it begins as light red, pin-head-like points, especially in the axilla and groin; second, there are red spots in a diffuse *erythema;* and lastly, there are red spots alone. This *minute papulation* is a very important aid to diagnosis in the second week. There may be minimal white lines in the eruption, but rarely spaces, as there are in measles. The eruption appears first on the neck, below the mastoid and clavicles, then upon the trunk, arms and hands, and lastly legs and feet. It is most intense over the extensor surface of the joints and on the trunk, and is least developed on the face and scalp. The palms and soles are usually free. The pallor around the mouth is triangular, bounded by the nasolabial folds above and the chin below, and contrasts vividly with the scarlet cheeks and minutely papulated forehead. Though not pathognomonic, it is valuable in differentiation, and is due to vascular spasm. Herpes near the angles of the mouth is very common. The eruption is occasionally roseolous about the ankles and wrists. The skin is always swollen. The hyperemic eruption pales on pressure and disappears after death, unless there are punctate hemorrhages,[1] which are not uncommon in severe types of the disease. After twenty-four hours the tension disappears, and the bright red color becomes a yellowish-red. After complete florition *desquamation* begins from a slight exudation of serum between the layers of the skin; it is usually bran-like, as in measles, on the head, face, neck and trunk, but is more scale-like and lamellar on the hands and feet, complete casts of which are sometimes shed. In severe cases even the hair and nails are shed, or the nails become furrowed. It begins on the cheek with the "rouge-

[1] The Rumpel-Leede phenomenon, consisting of the production of hemorrhages above the elbow after constriction as by a blood-pressure arm band, is not specific; nor is Pastia's sign, the occurrence of two to four small, dark red o reven hemorrhagic lines on the anterior surface of the elbows.

and-powder" appearance, and the skin may present a "pin-hole" or "worm-eaten" aspect before desquamation begins. Its duration is usually from one-half to three weeks. *Scarlatina levigata* is the typical rash described above. *Anomalies of eruption* are *scarlatina levis* (rudimentary eruption); *scarlatina sine exanthemate* (the rash being absent); *scarlatina papulosa* (marked papulation); *scarlatina miliaris* (from sweating, and exudation between the rete Malpighii and epidermis); *scarlatina variegata* (like measles, though not equally elevated); *scarlatina hemorrhagica* (usually ominous, and at times part of a general hemorrhagic diathesis). In general, irregular forms are dangerous.

3. THROAT.—The throat changes are the *most constant lesions* in scarlet fever, and constitute the internal eruption (enanthem). The disease begins in the throat with uniform redness and early swelling. The *throat* becomes speckled with *fine red points* like the skin papulation. (*a*) In mild cases, a simple catarrh with little swelling, a thick mucous coating, and reddening of the palate and tonsils, develop. (*b*) In moderate cases there is more swelling of the palate, and tonsillitis. (*c*) In severe cases, membrane, small abscesses, gangrene, minute hemorrhages, sloughing, edema, adenitis or cellulitis develop. Severe inflammation in the throat occurs in 65 per cent. of cases.

Clinical and anatomical diphtheria are often confused. "Necrotic inflammation" in scarlatina is that caused by the virus of scarlatina alone. Genuine diphtheria may accompany or follow scarlatina.

DIPHTHERITIC MEMBRANE	*vs.*	SCARLATINAL MEMBRANE.
Firm, adherent.		Softer, loosened in fragments.
Yellowish-gray.		Brownish tint.
Deep ulceration rarer, in more severe cases only, and later.		More frequent and earlier (even in twenty-four hours).
Phlegmon rare.		Not infrequent.
Laryngeal membrane (croup) frequent and deeper.		Rare, membrane thinner; lung complications rare.
Bacteriologically, the Klebs-Loeffler bacillus.		Streptococcus (Klebs-Loeffler bacillus only as a complication).
Paralysis frequent.		Rare, except local paralysis due to local swelling of pharynx.
Recurrence frequent.		Infrequent.

4. RESPIRATORY TRACT.—Though often unaffected, the respiratory tract may become involved secondarily by a descending process. The nose is involved in direct ratio to the severity of the throat developments, such as necrosis or diphtheria. Acute edema of the glottis is infrequent. Bronchopneumonia, acute lobar pneumonia (usually in the upper lobe and coexisting with nephritis), gangrene, infarcts and hypostasis are not uncommon. Pleurisy occurs in 5 per cent. of cases. It is likely to be severe and is frequently purulent.

5. CIRCULATION.—The *heart* is greatly damaged by the scarlatinal virus in 35 per cent. of cases, not including the cases of sepsis and nephritis. Endocarditis is frequently mural, whence its frequent latency, but it may result in chronic disease, especially of the mitral valves. After rheumatism, scarlatina is the most frequent cause of endocarditis. Pericarditis is usually septic. Disquieting cardiac dilatation may be

seen in severe cases, accompanied by the usual evidences of acute cardiac insufficiency; the causal *myocarditis* may occur during the fever or in convalescence. The blood-pressure is reduced 25 per cent. Tachycardia may result from toxemia or acute infective myocarditis. The pulse is irregular in 90 per cent. of cases and is both weak and fast. Dilatation and hypertrophy are much more frequent in children than in adults, and often complicate renal lesions. *Blood:* Leukocytosis develops early and may become extreme in fatal cases. The average count in adults is 10,000 and in children 23,000. The polymorphonuclears are increased two or three days after the eruption. The eosinophiles are increased. In the polynuclears, Döhle's so-called "inclusion bodies" are found, oval, rod-like or sickle-shaped inclusions, and present up to the fourth day; they also occur in other infections. The hemoglobin suffers an early and increasing reduction. A hemorrhagic diathesis is generally septic.

6. Digestive System.—The tongue is swollen, indented, broad and thick. Its coating is first grayish, but on the third to fifth day disappears, leaving the tongue red, dry and glistening, on which the enlarged papillæ appear granular and warty—the "strawberry" or "cat's tongue." It is quite pathognomonic and McCollom found it the only constant sign in 1000 cases of scarlatina. Diphtheritic ulcers and secondary necrosis in the stomach, esophagus and intestines; vomiting from nephritis or toxemia; moderate swelling of the liver and spleen or abdominal pain may be noted. Constipation is usual. Diarrhea may result from (*a*) catarrhal enteritis; (*b*) dysentery, with tenesmus and blood; or (*c*) the so-called "scarlet typhoid," accompanied by tympany, hemorrhage, rapid pulse, lethal outcome, and autopsy findings of splenic intumescence, and hyperplasia and ulceration of Peyer's patches.

7. Kidneys.—The urine is febrile. It often responds to Gerhardt's reaction and the diazo test; urobilinuria is ascribable to hepatitis. *Albuminuria* occurs in 75 per cent. of cases. The initial "catarrhal nephritis" is marked by hyperemia, parenchymatous changes, little interstitial involvement, some fluid in the glomeruli and a few cylinders in the tubules.

Nephritis usually develops in the third week. Its frequency varies with the epidemic, between 5 or even 90 per cent. No relation exists between its frequency and the severity of the infection. Glomerulonephritis is the most frequent anatomical form, characterized *anatomically* by hyperemia, increased consistence, and such glomerular changes as gray color, diffuse fatty granular degeneration, increase of nuclei, and increase in the size of the tufts, which become sausage-like, solid masses. We also note exudation of leukocytes about the afferent vessels, multiplication of capsular epithelium with occlusion of the lumen, hyaline alteration of the vessels, and early interstitial changes, and later, connective-tissue growth.

Clinically (*a*) *the urine is decreased or suppressed,* heavy with sediment and *albumin* (averaging 0.5 per cent.), poor in urea, turbid (specific gravity 1.030), and *full of casts of all types*—especially, long, flat, band-like, twisted and frayed casts. Much oxalic and uric acid is found and,

in many cases, blood. Pollakiuria occurs in 10 per cent. (*b*) *Hydrops* shows the characters of renal dropsy; it appears first under the skin, the bloodvessels of which are altered by the virus. It shifts its location without cause, and may also produce hydrothorax, ascites, edema of the larynx, lungs or genitalia. Rarely hydrops occurs without nephritis, and kidney inflammation without urinary alterations. There may be lumbar pain, vesical tenesmus and (*c*) *uremic symptoms*, such as headache, twitchings, convulsions, wide pupils, amaurosis, coma, vomiting, diarrhea, cardialgia, rapid pulse and breathing, and pale skin tinged with cyanosis. Many *convulsions* usually imply a lethal termination in coma. The prognosis is more favorable if the pupils are not immobile. Uremia, hydrops and intercurrent inflammation are always dangerous. Recovery is possible after one or even three weeks of anuria. Convalescence is the rule after four to six weeks and the nephritis rarely becomes chronic.

8. Special Senses.—*Middle-ear* inflammation (22 per cent.) usually begins before desquamation, and is accompanied by fever alone or with pain, glandular swelling, difficult hearing, tinnitus, a reddened, glistening, sunken membrane, and, in children, by some brain manifestations. It is quite frequent in early life and rare after the fifteenth year; at least 10 per cent. of cases of acquired deafness result from scarlatina. Catarrhal conjunctivitis, diphtheritic inflammation, herpes or necrosis of the cornea; panophthalmitis, muscular paralysis and disturbance in accommodation, are possible complications. Uremic amaurosis, septic or nephritic neuroretinitis and retinal hemorrhage, are rare.

9. Lymphatic System.—The glands, spleen, follicles and in severe cases, all lymphatic structures, are affected by the sepsis. McCollom found enlargement of the cervical glands in 50 per cent., and suppuration in 8 per cent. The "collar of brawn" results from cervical adenitis and periadenitis. Gangrene, retrosternal abscess and jugular phlebitis with sepsis, occur in desperate cases.

10. Bones and Joints.—Scarlatinal arthritis (in 6 per cent.) usually occurs in the second or third week, with desquamation and subsidence of the fever, and lasts three or four days. It attacks the smaller rather than the larger joints, the wrists and ankles rather than the elbows, shoulders and knees. It is not so migratory as ordinary rheumatism. Swelling of the joints does not always occur. It occurs in old rather than in young subjects. The prognosis is usually good.

Scarlatinal sequels are anemia, retarded development, decreased physiological resistance and rarely tuberculosis. Latent sepsis with brain foci, *valvular disease*, chronic *nephritis*, chronic adenitis, otitis, mastoiditis, arthritis, meningitis, hemiplegia, facial paralysis, etc., are fortunately rare complications and sequels.

Diagnosis.—Typical cases are determined easily when an epidemic prevails, and when the eruption is subordinated to other criteria:

1. Sudden onset and rapid development, with fever and vomiting.

2. Angina, punctate spots in the throat, swelling and dysphagia are practically always present. Severe throat symptoms are always suspicious.

3. Strawberry tongue, which is constant.

4. Exanthem, first about the clavicles, with the face less involved; the mouth free and intense on the body; papules, then erythema develop; there are no free spaces; it results in desquamation.

5. Lymphadenitis. In this the cervical are less important than the inguinal or other glands.

6. Fever, hydrops, albuminuria and nephritis in atypical cases.

7. Scaling, tender joints and albuminuria help in making a retrospective diagnosis if the initial symptoms were not clear.

Diagnosis of Atypical Scarlatina.—1. Severe scarlatina. (*a*) The anginose form resembles septic diphtheria, with marked toxemia, necrosis and adenitis. (*b*) Atactic form; violent intoxication, high fever and death before the eruption appears; blood dissolution, vomiting, diarrhea, convulsions, coma and a rapid, weak heart, are the chief symptoms. (*c*) Hemorrhagic scarlatina, characterized by severe fever and brain symptoms at the onset; incomplete exanthem, necrosing angina, marked glandular and splenic swelling; subcutaneous, serous and mucous membrane hemorrhages with exulceration. It is usually lethal after ten to twenty hours from hyperpyrexia, vomiting, diarrhea, delirium, convulsions and dyspnea, especially in reduced subjects under two years of age.

2. Very light cases may present (*a*) all the usual signs, though but mildly developed; or (*b*) some symptoms may be absent (*scarlatina fragmentaria*), as in cases with no temperature (*scarlatina sine febre*), no rash (*s. sine exanthemate*), or no angina (*s. sine enanthemate*). In some atypical cases a definite diagnosis is impossible, as in acute nephritis with uremia, fever, sore throat and gastro-enteritis, without the characteristic rash. Recurrence is noted in 0.2 per cent.; many of these cases are septic complications.

Differentiation.—The differentiation is as follows:

Scarlet Fever	*vs.*	Sepsis.
Bright red erythema, with small red papules.		A very deep purple-red rash, sometimes spreading over entire body.
The eruption is much the same in both diseases, the same places being exempt.		
Miliaria are rare.		Frequent.
Rather typical desquamation.		Desquamation observed less frequently.
Criteria: angina, tongue, onset, glands, etc.		Etiology, chills, sweats, fever irregularity, polymorphous exanthems, etc.

Diphtheria is often differentiated with difficulty. A simple erythema is sometimes observed in diphtheria, which is darker, more on the trunk, and more transitory than in scarlatina (*v. s.*).

Drug rashes, from belladonna, iodoform, quinine, iodide, chloral or copaiba may be easily differentiated, if the cardinal signs of scarlatina, rather than its rash alone, are considered.

Measles and *German measles* (page 273). *Acute exfoliating dermatitis* also begins with fever, with a spreading erythema, and early desquamation, but the cardinal criteria of scarlatina are lacking. *Erythema* is more transitory; the points are absent; it occurs with less fever and

without angina or adenitis. *Erysipelas* presents edematous swelling, a sharp border, marked pain and a slow advance.

Prognosis.—The death-rate depends on the *genius epidemicus* (between 2 and 40 per cent.), but averages 10 per cent. The prognosis is least favorable in hospital cases and most deaths *occur under six years;* in children under five years the mortality is 25 per cent., and still higher in the first year of life.

Uremia accounts for 50 per cent., and *respiratory complications* for 20 per cent. of scarlatinal fatalities; 15 per cent. of nephritic cases die. The severity of the infection, hemorrhagic forms, severe toxemia, complicating sepsis, necrosis in the throat, otitis and heart complications are the other factors in prognosis.

Treatment.—The disease runs its self-limited course.

I. Prophylaxis.—Isolation is imperative in all cases; indeed, light or ambulatory forms more often convey infection. Susceptibility is not universal and the virus is less penetrative than in measles, whence it is possible to circumscribe its diffusion. Care should be taken to prevent transmission through schools, fomites or third persons. The infected room should be kept empty and exposed to the wind and sun for one month afterward. The physician should wear an operating gown and carefully wash his face, beard and hands after leaving the sick-room. Quarantine should be maintained from eight to ten weeks, or longer if discharges from the nose, nasopharynx, or ears continue. All discharges should be disinfected. Formaldehyde fumigation has been discussed (page 258). Care in surgical and puerperal cases is important, but the danger has been overestimated. Bichloride wrappings should be placed about the dead body, and the funeral should be private.

Schools must be closed during epidemics. In hospitals the cases of scarlatina with streptococcus infection are well separated from cases of simple scarlatina. Sound children should be sent away from the house and isolated lest they spread the disease. If they remain at home, one-quarter of them will contract scarlatina.

II. General Treatment. 1. *Decubitus.*—The prone position is imperative, during the disease and the first two weeks of apyrexia.

2. *Room.*—The room should be light, well ventilated, dry and warm, especially in convalescence. A grate fire is advisable and the windows should be open at the top.

3. *Skin.*—During the fever the skin should be covered with linen, and in convalescence with wool. The eruption may be brought out, when necessary, by mustard baths. During the fever, the patient is washed daily with warm water and soap, avoiding unnecessary exposure. The tension of the skin is thus lessened, the fever decreased and toxemic symptoms allayed. During desquamation, oil-rubs were once employed to decrease the dissemination of dry scales, but they decrease the functions of the skin, which are of great importance when the kidneys are involved; also, infection is carried by means of throat secretions. Soap and warm water serve equally well. The author is opposed to antiseptic soaps because of the chance of renal injury.

4. *Fever.*—The following fever formula is harmless:

℞—Liquoris ammonii acetatis . . . . . ℥iss
Tincturæ aconiti . . . . . ♏xxiv
Aquæ . . . . . q. s. ad. ℥vj
Fac misturam et signa.
One to three teaspoonfuls every four or five hours, according to age.

This mixture tranquilizes, promotes the rash and allays fever. Hydrotherapy is useful, such as the warm sponge or full warm bath, not the full cold bath. Special care is indicated in adynamic cases, because of danger of heart-failure, in pericarditis, stenosis of the air passages or dyspnea, in hemorrhagic forms and nephritis.

5. *Diet.*—Water should be given freely. A *fluid diet* is indicated by the fever, the renal complications and the dysphagia. Concentrated foods, milk, soups and jellies, and in defervescence, milk-toast, custard, puddings, bread and butter may be given. Eggs and meats should be avoided. In severe throat complications, rectal feeding is indicated. (See Gastric Ulcer.)

6. *Arthritis.*—The A-B-C liniment (aconite, belladonna and chloroform), warm wool and flannel bandages are helpful.

℞—Tr. opii . . . . . ℥iij
Phenolis . . . . . ℥j
Tr. belladonnæ . . . . . ℥iij
Ol. olivæ . . . . . ℥iv
M. et S.—Apply on muslin covered by cotton.

Salicylates are beneficial, but unless given carefully, may injure the kidneys. If the arthritis is obstinate or purulent, aspiration with all surgical care, and even drainage may be indicated.

7. *Angina.*—Ice in the mouth and Dobell's solution as a nasal douche should be given. Care should be exercised in the use of antiseptics, lest children swallow them. A 10 per cent. hydrogen peroxide, 2 per cent. chlorate of potash, or 2 per cent. carbolic gargle may be used; formalin is too irritating. Caustics are contra-indicated.

For the nose the following formula is excellent:

℞—Phenolis . . . . . ℥ss
Acidi borici . . . . . ℥ss
Glycerini . . . . . ℥j
Tr. myrrhæ . . . . . ℥j
Aquæ . . . . . q. s. ad. ℥v
M. et S.—Locally, in such dilution that no irritation is felt.

A 10 per cent. nitrate of silver solution is used with advantage in the early stages of the angina. Coincident diphtheria (*q. v.*) is treated by antitoxin, and antistreptococcus serum may be exhibited in streptococcic angina.

8. *Heart.*—Cardiac stimulants may be exhibited according to Rotch's dosage table:

Age.	Tincture of digitalis.	Strychnine.	1 per cent. solution nitroglycerin.	Atropine.
3 months	$\frac{1}{10}$–$\frac{1}{2}$ minim	$\frac{1}{2000}$–$\frac{1}{1000}$ grain	$\frac{1}{75}$–$\frac{1}{50}$ minim	$\frac{1}{3000}$–$\frac{1}{1500}$ grain
6 "	$\frac{1}{10}$–$\frac{3}{4}$ "	$\frac{1}{1500}$–$\frac{1}{500}$ "	$\frac{1}{25}$–$\frac{1}{15}$ "	$\frac{1}{2500}$–$\frac{1}{1000}$ "
9 "	$\frac{1}{4}$–1 "	$\frac{1}{500}$–$\frac{1}{300}$ "	$\frac{1}{25}$–$\frac{1}{10}$ "	$\frac{1}{1500}$–$\frac{1}{750}$ "
12 "	$\frac{1}{4}$–1$\frac{1}{2}$ "	$\frac{1}{500}$–$\frac{1}{250}$ "	$\frac{1}{25}$–$\frac{1}{5}$ "	$\frac{1}{1000}$–$\frac{1}{500}$ "
2 years	$\frac{1}{2}$–2 minims	$\frac{1}{500}$–$\frac{1}{150}$ "	$\frac{1}{15}$–$\frac{1}{3}$ "	$\frac{1}{750}$–$\frac{1}{250}$ "
3 "	$\frac{1}{2}$–3 "	$\frac{1}{300}$–$\frac{1}{100}$ "	$\frac{1}{10}$–$\frac{1}{2}$ "	$\frac{1}{500}$–$\frac{1}{200}$ "
4–10 "	1–5 "	$\frac{1}{200}$–$\frac{1}{60}$ "	$\frac{1}{2}$–$\frac{3}{4}$ "	$\frac{1}{250}$–$\frac{1}{150}$ "
10–12 "	3–8 "	$\frac{1}{100}$–$\frac{1}{40}$ "	$\frac{1}{2}$–1 "	$\frac{1}{200}$–$\frac{1}{100}$ "

9. *Lymph Glands.*—Secondary infection is decreased by prophylactic care of the mouth and throat. Ice is applied in the beginning only, and later, hot boric acid dressings when the blood and lymph streams stagnate. Iodine or ichthyol salves and surgical intervention are indicated in the order named.

10. *Otitis* is treated by copious boric irrigations, given quite hot and at low pressure, and followed by equal parts of glycerin and laudanum, or 5 per cent. carbolic acid. Leeches over the mastoid often relieve the pain. Examination of the drum is difficult in children with a small, sensitive meatus. Early puncture under cocaine is much safer than awaiting spontaneous rupture. After rupture a small amount of boric powder should be applied.

11. *Nephritis* is treated (*a*) according to the old prophylactic rule, by which all cases, severe or light, are *confined to bed* for at least four weeks, and to the *milk diet.* Irritants, like cantharides, always are to be avoided. Tepid baths lasting a quarter of an hour should be given daily. Watch the temperature and the urine in convalescence. The results with urotropin as a prophylactic are at least suggestive. (*b*) Treatment of established nephritis. Hot baths increase the sweat and flow of urine. They are given twice daily, for half an hour, and the patient kept afterward between blankets. Alkaline diuretics, such as potas. acet. and p. citrat., may be given; pilocarpine for a child two years old, $\frac{1}{40}$ to $\frac{1}{20}$ gr., and calomel as a laxative and diuretic must be used with great care. (See TREATMENT OF NEPHRITIS.)

℞—Infusi digitalis . . . . . ℥v
Potassii acetatis . . . . . ℥ss
Syrupi simplicis . . . . . ℥ij
Aquæ destillatæ . . . . . q. s. ad. ℥ij
M. et S.—One teaspoonful every two hours.

High arterial tension indicates nitroglycerin. For convulsions, chloroform, chloral and bromides are valuable. For vomiting, iodine is efficacious. Venesection and digitalis are necessary in pulmonary edema.

For uremia, two drops of croton oil placed undiluted on the tongue, should produce free cartharsis in two hours. If not successful by that time it should be repeated. Pulvis jalapæ compositus (gr. xv to ʒj) unloads the bowels in less urgent cases. Early evacuation is indicated in streptococcic pleurisy.

12. *Tonics.*—Such tonics as iron, arsenic and cod-liver oil are indicated in convalescence. The heart must be watched for weeks, because the myocardium is often degenerated. Salvarsan is recommended.

13. *Serum.*—Diphtheria antitoxin apparently benefits some scarlatinal infections. Streptococcic sera have given no definite results. Immunization against one variety of streptococcus does not produce immunity against other varieties. In Vienna, Moser's serum has apparently been efficacious; the mortality was 8 per cent., as against 14.5 per cent. without serotherapy.

MEASLES (MORBILLI, RUBEOLA).

Definition.—An acute, highly infectious disease, characterized by initial coryza, inflammation of the air passages, rapidly spreading eruption, a fever-curve with a double summit, and by toxemia.

History.—Its first clear description dates from Rhazes (980–1037 A.D.). It was widely epidemic in the eighteenth century.

Etiology.—(*a*) *Susceptibility* is almost universal and few individuals escape (14 per cent.). One attack generally confers immunity; Maiselis collected 106 cases with a second attack; third and fourth attacks are known. (*b*) *Age:* It is rare during the first five months of life, and occurs most frequently after the first year. It is more common in adults than is scarlet fever. In 16,981 cases, 50.4 per cent. occurred in the first five years; 42 per cent. between five and ten years; 5.2 per cent. between ten and twenty years, and 2.4 per cent. after twenty years of age. (*c*) *Season:* Measles occurs mostly in the winter and spring. (*d*) The unknown *virus* exists in the blood, tears, bronchial and nasal secretions, and in the skin or scales. Monkeys were successfully inoculated by Anderson and Goldberg, who proved that the virus was filterable and that infectivity was greatest during the catarrhal and eruptive stages. Propagation occurs by personal contact, and the healthy carrier is a most important mode of infection. In the home the disease usually runs through the family. The most frequent means of dissemination is the school-room. It is more often epidemic than scarlatina, especially in the country. After smallpox in the unvaccinated, measles is the most infectious disease.

General Clinical Course.—The stages are: (1) Incubation of nine or ten days; (2) initial or catarrhal stage of three or four days, with fever, catarrhal symptoms and an enanthem; (3) the eruptive stage, lasting four or five days, with renewed fever, intoxication, eruption and inflamed mucosæ; and (4) the desquamative period.

1. Incubation.—The incubation lasts nine or ten to fourteen days.

2. Catarrhal Symptoms.—Early catarrhal symptoms are more common in measles than in any other acute infection of childhood. Chilly

sensations, malaise and frontal headache precede redness of the eyes, lachrymation, photophobia, coryza and sneezing, pain in the nose, throat and bronchi, and cough, which develops after one day. Examination of the mouth reveals *Koplik's spots* in 90 per cent. of cases, described by him in 1886 and Filatow in 1885 (Plate IX). They are found on the buccal mucosa near the molars, on the first day, as round, bluish-white spots, surrounded by a reddish areola. The initial fever reaches 102° or 103°, and declines gradually during the third and fourth days.

3. Eruption.—The eruption begins on the fourth day with papules on the temples, cheeks, forehead, then on the back, wrists, forearms, and on the anterior surface of the body and legs, in order of progression as named; with the appearance of the eruption, the fever again rises. The eruption disappears on pressure and is palpable and superficial. There are always islets of normal skin. The glands are usually enlarged.

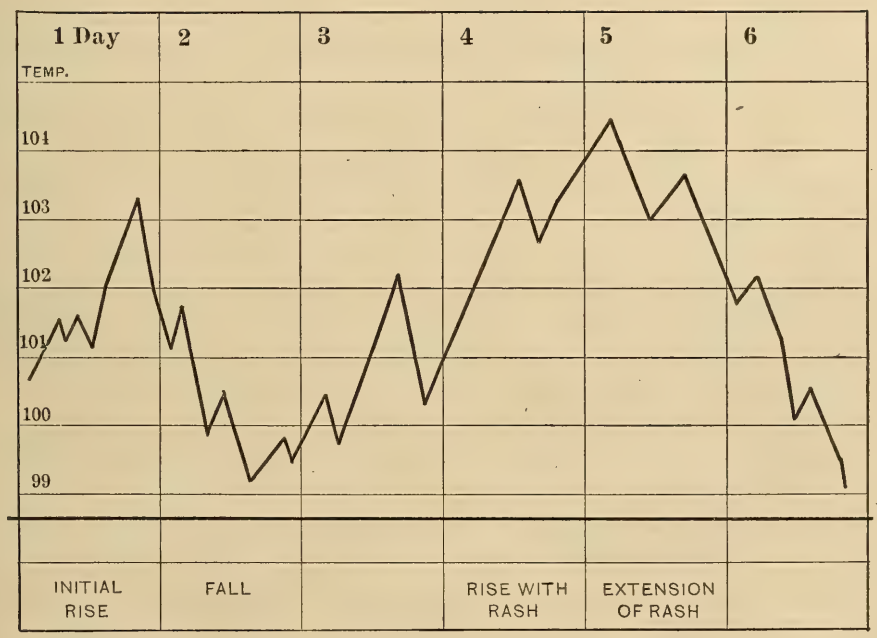


Fig. 20.—Fever-curve in measles.

All symptoms abate on the sixth day, by crisis. In severe cases, diarrhea, toxemia, bronchiolitis, pneumonia, cyanosis and asphyxia, may be observed, and death may result from lung involvement or mixed infection.

4. Desquamation.—The skin pales, becomes slightly pigmented, and fine scales form, with convalescence in eight or ten days.

Individual Symptoms in Detail.—1. Eruption.—It is essentially the same on the skin and mucosæ. In *the mouth* the mucosa is red on the first day of fever, and *Koplik's spots* are found; on the second day redness is noted on the tonsils and palate. The enanthem may reach the larynx, intestines and genitalia. The *skin eruption* (*exanthem*) is circumscribed, and hyperemic rather than exudative. It fades on pressure; the roseolæ may not fade on pressure in the nodular form of measles. The *roseolæ* usually are discrete, slightly *elevated*, light to dark red in

color, *round or crescentic* in shape, and average 2 to 6 millimeters in diameter. They *always leave islets of normal skin* between the independent spots of eruption, even in the most diffuse types. The eruption follows a descending order and reaches the lower parts of the body last. When the eruption develops on the body first, the course is usually anomalous. Desquamation follows the same order as the eruption and is usually fine. Scars never result. The eruption is rudimentary in those who are debilitated by chronic diseases, and is exuberant in the healthy.

The varieties are: (*a*) *Morbilli læves,* the usual form, with small elevations. (*b*) *M. vesiculosi seu miliares.* (*c*) *M. papulosi,* the nodular, slightly "shotty" induration is always superficial. (*d*) *M. confluentes seu conferti.* (*e*) *M. hemorrhagicæ,* benign or malignant (black, typhoid or septic type); early hemorrhage into the eruption is almost always fatal. (*f*) *M. sine exanthemate; sine enanthemate.* "Recession," "striking in" of the eruption usually denotes heart-failure.

2. The Fever.—The curve begins suddenly without a chill, and falls on the second day, perhaps to normal, usually rises again with the eruption, and reaches its maximum in thirty-six hours, thus giving *a curve with a double summit.* It usually falls by crisis before the eruption disappears. The temperature is normal during desquamation. Fever, after the eruption begins to fade, indicates complications. In rare cases fever is absent.

3. The Respiratory Tract.—*Nasal catarrh,* if obstructive, is of special moment in sucklings; 50 per cent. of cases suffer from erosions of the larynx, 16 per cent. from pseudomembranous laryngitis, and 15 per cent. from simple inflammation. *Bronchial catarrh* may reach the smaller tubules, usually late in the disease. It produces a clear, stringy, viscid sputum which later may become purulent and nummular. The bronchial glands are always swollen. Atelectasis may occasion indeterminate breathing and dulness, which decrease with change of posture or deep breathing. *Bronchopneumonia* occurs in 6 to 12 per cent. of cases and *is the most frequent fatal complication,* patches of bronchopneumonia occurring in every fatal case. It differs from other lobular pneumonias in its rapid development, and its greater tendency to pus formation, because the measles-catarrh favors mixed infection. Bronchial breathing and other physical signs are not always detected clinically. The temperature is often no higher than in bronchitis. Toxemia and carbon dioxide narcosis accentuate the patient's distress. Bronchopneumonia is rapid in development, but slow or sometimes incomplete in resolution. It may result in interstitial pneumonia, gangrene or *tuberculosis,* which dreaded sequel of measles occurs (*a*) as a miliary invasion, and in the brain more frequently than in the lungs, bacilli from old, caseated bronchial glands being released by solvent substances reaching them from the lungs by way of the lymph vessels. (*b*) It also occurs as a fresh tuberculosis developing upon a bronchopneumonia. Croupous pneumonia is as infrequent as pleurisy, which is in most cases secondary to lobular pneumonic foci.

4. Cardiac Symptoms.—Accidental bruits may be heard. Toxemia exceptionally causes myocardial degeneration.

PLATE IX

FIG. 1

FIG. 2

FIG. 3

FIG. 4

The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthem on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

5. Digestive Tract.—The tongue is coated. There is thirst, and aphthæ and stomatitis may be observed. Diarrhea, mucus in the stools and, anatomically, swelling of Peyer's patches, have been described. The liver is seldom swollen.

6. Nervous System.—Delirium, coma or convulsions in rare cases, result from severe toxemia, though even the delicate nervous system of children is seldom involved in measles. Nervous symptoms in protracted pneumonia, may arouse suspicion of miliary tuberculosis or tuberculous meningitis.

7. Genito-urinary Tract.—Acetonuria and albuminuria are frequent. Ehrlich's diazo reaction occurs in 75 to 100 per cent. of cases. Nephritis is very uncommon; gangrene of the genitalia has been observed.

Differentiation from German Measles and Scarlatina.

German measles.	Scarlatina.	Measles.
Incubation; 14 to 21 days.	2 (1 to 7) days.	9 to 10 days.
Prodromes; absent, short or light.	Brief, a few hours to 1 or 1½ days.	3 to 4 days, generally markedly catarrhal.
Invasion; very mild.	Sudden, stormy, vomiting, convulsions, angina, and adenitis.	Catarrhal symptoms.
Exanthem; the rash is the first symptom, usually appearing on the first or second day on the face; as small red dots, rosy and brighter than in measles; small, little elevated, and thick about the mouth. They do not fuse. Less crescentic than in measles.	Appears on first or second day, reaching a maximum on second to fourth day, first below clavicles, involving face later; dusky red, diffuse, burning, leaving mouth and eyes free.	Occurs on fourth day, greatest on fifth or sixth day on the face, forehead, and about the mouth; brick-red, elevated papules or crescentic islets of eruption, always with areas of healthy skin between.
Enanthem; may be diffuse redness of throat and conjunctivæ.	Angina proportionate to skin eruption; dusky red; tonsillar plugs; conjunctivæ, bronchi, intestines usually intact.	Koplik's spots; patchy redness of throat; photophobia; conjunctivitis, marked bronchitis, very often bronchopneumonia; diarrhea in some cases.
Glands; usually enlarged, hard, tender, especially posterior cervical, also axillary and inguinal. Spleen somewhat enlarged.	Proportionate to angina, hence largely cervical. Enlarged spleen.	Mostly at angle of jaw; rarely systematic enlargement. No enlargement of spleen.
Constitutional symptoms; few; no depression; no leukocytosis; little pulse increase and fever.	Much depression with rash, coated tongue with peeling on the fourth day (strawberry tongue); fever 105° to 106°; pulse increased more than fever; lysis on seventh day; polynuclear leukocytosis; eosinophilia.	Depression, coated tongue, anorexia; fever-curve with two elevations; crisis on seventh day; no leukocytosis. No eosinophilia.
Complications; practically none.	Nephritis, arthritis, endocarditis, sepsis, periadenitis. Diazo in 10 per cent.	Catarrhal pneumonia, pleurisy, tuberculosis. Diazo in 75 per cent.
Convalescence; rapid; desquamation rarely copious, and always fine.	Prolonged; tardy; desquamation copious and usually in shreds (hands and feet).	Often prolonged; desquamation rarely copious and always fine.

8. Other Symptoms.—The spleen and lymph nodes are but slightly swollen; bone and joint complications and keratitis are infrequent; otitis media occurs in a small percentage of cases. There is a relative lymphocytosis, with an absolute leukopenia.

Diagnosis.—The diagnosis depends upon the consideration of all the symptoms (coryza, cough, fever, enanthem) rather than of the eruption alone. Differentiation from *typhus* is made by the greater general disturbance in typhus, its more violent onset and the roseolous eruption becoming petechial.

Sepsis with a morbilliform exanthem, *influenza*, *syphilis* and *variola* (*q. v.*) sometimes delay the early diagnosis. In the *drug eruptions* of turpentine, iodide, quinine, chloral, cubebs, copaiba and antipyrin, the fever, Koplik's spots, catarrh, and central elevations in the eruption are indicative of measles (see page 273).

Prognosis.—The mortality is very great among aboriginal races; in the Fiji Islands 140,000 died out of a population of 160,000. The usual prognostics are: (*a*) The *genus epidemicus:* The mortality may be 30 to 37 per cent. in certain epidemics; or as in a German epidemic of 2881 cases, only 1 per cent. (*b*) *Individual resistance:* It is higher in institutional practice and is 16 per cent. in the children's hospitals, as against 3 per cent. in private practice. Measles ranks third among the exanthemata in mortality. (*c*) *Age:* 55 per cent. of fatal cases occur in the first two years of life. (*d*) *Complications* are more important than the disease itself, *e. g.*, tuberculosis and bronchopneumonia, which is the worst complication, with a mortality of 33 per cent.

Treatment.—1. Prophylaxis is important, especially in weakly children under two years, in severe epidemics with pneumonia and in cases of pertussis. It is of no advantage for the young to contract measles, and the prevalent practise of allowing the disease to run through the family must be condemned. The secretions from the nose, throat and bronchi must be disinfected, and general sanitary care should be exercised as in smallpox (*q. v.*) and scarlatina; quarantine is maintained for ten days after the onset.

2. Hygiene.—The patient should have fresh air, warm quarters, rest in bed until after desquamation, and a liquid diet.

3. Catarrhal Symptoms.—Photophobia does not necessitate a dark room; a boric acid solution should be dropped frequently into the eyes and vaseline applied to the lids. To relieve pain in the ears, belladonna, opium and a 1 per cent. cocaine solution may be used; paracentesis is indicated for gathering. The nose should be douched with Dobell's solution. Tr. belladonnæ, ♏j–iij, may be given every two hours for coryza until flushing of the face, increase of pulse-rate, or a dry red throat develops.

4. Antipyresis.—In high fever with cerebral toxemia and diffuse bronchitis or pneumonia, baths are most efficacious with cold affusions to the head and neck; wine, mustard plasters and laxatives may also be indicated.

5. The Circulation.—The heart rarely requires support except in pneumonia (*q. v.*), when camphor, aromatic spirits of ammonia, strychnine, or coffee is indicated. (See dosage table, page 269.)

6. Respiratory Symptoms.—Care of the mouth, a soft or liquid diet, inhalations of steam, hot fomentations over the larynx, warm baths and possibly intubation or tracheotomy, are indicated for laryngeal complications. Bronchitis should be treated with ipecac, codeine, inhalations of steam, massive hot packs to the chest, and cold affusions over the nape of the neck. (See Acute Bronchitis.)

7. Alimentary Tract.—Enemata relieve constipation. When diarrhea is excessive, opium, hot wine or cognac, demulcents, hot applications, and physiological salt solution by rectum, are indicated.

8. The Skin.—The skin should be rubbed with olive oil; a 2 per cent. menthol salve, or 1 per cent. carbolic solution, relieves itching.

RUBELLA.

Synonyms.—Rubeola notha, German measles, Rötheln. By the Germans it is called rubeola, though we employ rubeola as synonymous with measles.

History.—Rubella was first described by Bergen (1752) and its existence as an independent infection was established by Maton (1815). It is more often confused with atypical scarlatina than with atypical measles.

Etiology.—Its etiology is not known. Most cases occur during the first half of the year. The disease most frequently attacks children.

Symptoms.—1. Incubation.—The incubation lasts from ten to twelve (perhaps fourteen to twenty-one) days.

2. Prodromes.—Prodromes are usually lacking; for a period of two hours to a day, there may be cough, photophobia, sneezing, slight temperature, chilliness or headache.

3. Eruption.—The eruption appears, as the first symptom of the disease, on the first day, usually in the palate and throat, and consists of hyperemic punctate red spots (Forchheimer's spots). The skin is red, and the spots, which are more rosy than in measles, disappear at first under pressure, then become darker and more circumscribed; later, the eruption does not entirely disappear under pressure. The eruption does not fuse as in measles, and erythema is not present about the points of eruption as in scarlatina. It advances in "jumps," and fades in one place while it blossoms in another. It appears first on the scalp and face, and progresses downward to the neck, body (where the eruption is greatest), arms and lastly to the legs and feet. Unlike scarlatina, spots are aggregated about the mouth. Its average duration is from two to four days. The papillæ of the tongue show slightly through the fur. Desquamation is minimal and fine. Almost characteristic is the *swelling* of the *posterior cervical* and *postauricular glands*. There may be some angina, dysphagia, anorexia, tracheitis or, rarely, bronchitis.

4. Constitutional Symptoms.—The fever is usually slight, runs three days, and falls when the eruption matures. Except in a few American epidemics, urgent nervous and other complications, such as icterus, albuminuria, nephritis or colitis, have rarely been observed.

Diagnosis.—The diagnosis is often difficult and the unity of the disease is sometimes questioned. The "*fourth disease*," of Dukes cannot be

admitted to our nosology. *Erythema infectiosum*, emphasized by Escherich, is a weakly contagious, maculopapular erythema, prevalent in the spring and fall, and occurring chiefly on the extremities, and rarely on the trunk (*v.* page 273).

Prognosis.—The prognosis is good, and *therapy* is symptomatic. Isolation is theoretically advisable.

TYPHUS FEVER.

Synonyms.—Typhus fever; exanthematous typhus; war, prison, ship or famine fever.

Definition.—An acute, highly contagious, specific infection, with a sudden onset and a high cyclic temperature, falling by crisis, a characteristic eruption, and severe nervous symptoms.

Etiology.—All stages of the disease are highly contagious. Infection is usually direct from person to person. The danger to attending nurses and physicians is great. The more intimate the contact the greater is the danger of direct infection. It is communicable indirectly by a third person, fomites and fleas.

Nicolle, Ricketts, Anderson and Goldberger inoculated monkeys and noted that infection from animal to animal occurred by the body louse. The bacteriology of typhus is undetermined.

Geography.—Ireland, Russia, Galatia, Hungary, Italy, Asia Minor and Mexico are its most frequent homes. It is endemic in Berlin among the lower classes and vagabonds. Typhus was one of the greatest scourges of the middle ages, with its wars and famines, but it has decreased in the past century, the epidemics in Ireland (1817 and 1846), in the Turko-Russian war (1877–78) and the present European war being the last general outbreaks. Its most recent occurrence in this country was in New York (1881–82 and 1893), in Philadelphia (1883), and in Baltimore in 1901.

Age.—Most cases occur between the fifteenth and twenty-fifth years.

General Clinical Picture.—1. The *incubation* lasts twelve days or less, usually without symptoms.

2. The *invasion is sudden*, exhibiting chills and high temperature on the first day. Thirst follows, with vomiting, epigastric oppression, suffusion of the face, conjunctivæ, nose and pharynx, delirium, backache, severe pains in the extremities, rapid pulse, and splenic intumescence.

3. On the third or fifth day a pale red and purely hyperemic roseolous *eruption develops*, first on the abdomen and chest, extending rapidly to the back and extremities. The eruption then becomes petechial, with some serous infiltration in the flecks, which become dirty and copper-colored. It lasts seven to ten days. Pain is succeeded by apathy, prostration, delirium or mania. The tongue and mouth are dry and dark. Catarrhal inflammations of the pharynx, larynx and bronchi develop, with hoarseness and cough. The fever is continuous. Coma-vigil, rapid respiration and pulse, and hypostatic pneumonia may end the course.

4. A *crisis* occurs on the tenth day. The pulse becomes slower; the skin often desquamates in dusky scales; profuse sweats are common; and convalescence is established in two weeks.

Special Symptomatology.—1. Fever.—After a rigor, the temperature rapidly rises to 103°, 105° or even 107°, being highest on the fifth day. No other acute exanthematous disease produces an equally high, early temperature. On the tenth day it remits, except in severe cases, usually with a crisis in two or three days. Death occurs with a preagonal rise or with collapse temperature.

2. The Skin.—The eruption consists of two elements: Roseolæ changing to petechiæ, and a dusky red mottling under the skin. The *roseolæ* are seen on the third or the fifth day, increase rapidly though never in recurrent crops, are located chiefly on the lower abdomen, chest, back or shoulders, are least abundant on the thighs, and do not appear on the face except in women and children whose skin is delicate. The palms and soles are exempt. The hyperemic roseolæ are pale red, washed in outline, round or oval, the size of a pin-head or a lentil, and disappear on pressure. They are easily overlooked at night. They last one to three days, later becoming livid, copper-colored *petechiæ* persisting on pressure and after death, because hemorrhagic. The petechiæ occur mostly on the back or in the groins. The hemorrhage may extend more widely into the skin or subcutaneous tissue. Many petechiæ are ominous, especially in cachectic and alcoholic subjects. A morbilliform eruption on the arms and legs is occasionally observed before the usual typhus rash, especially in thin-skinned individuals. The skin is usually dry, but miliaria appear in 10 per cent. of cases, notably in the young. Icterus without decolorization of the stools is a frequent and unfavorable sign. Desquamation is sometimes profuse; herpes occurs in 12 and bed-sores in 3 per cent. of cases. The skin has a peculiarly pungent, offensive odor.

3. The Nervous System.—Toxemia is profound. Headache is constant until the exanthem appears; it is usually frontal and temporal and radiates into the neck or shoulders. It is associated with vertigo, backache and hyperesthesia of the fingers and toes. The sensorium first shows depression, apathy, insomnia, incoherent speech or disturbing dreams, which increase as the pains disappear. In the second week coma-vigil, low delirium, mania and hallucinations, involuntary evacuations and noisy cerebral respiration often develop. Tremor of the forearms and hands is almost constant. Convulsions are rare, but ominous, chiefly in alcoholic subjects and children. Paraplegia and aphasia are recorded.

Special Senses.—There is conjunctival catarrh and the "pin-head pupil" of Graves. The ears are involved in 32 per cent. of cases.

4. Circulation.—The heart and vasomotor system suffer greatly. The pulse is small and reaches 100 to 120, or more. It is slow only in light cases. Acute myocarditis may occur with acute dilatation (*v.* Typhoid). In fatal cases the heart muscle is granular and deep red. The blood is dark and fluid; the red cells and hemoglobin are decreased, and the white cells are increased.

5. Respiration.—Bronchitis and tracheitis are present at the onset, increase with the fever and are evidenced by cough and slight expectoration; and with weak heart result in hypostasis and lobular pneumonia. *Pneumonia* occurs in 15 per cent. of typhus cases, usually in the second week and is frequently atypical.

6. Gastro-intestinal Tract.—The tongue, at first moist and white, becomes dry and dark—the "parrot tongue." Nausea and vomiting are common, but meteorism is infrequent. The epigastrium only is tender. The liver is swollen, soft and grayish. There is constipation in 90 per cent. of cases; diarrhea may appear at the crisis. Gastro-intestinal hemorrhage, a hemorrhagic diathesis, parotitis, ulceration of the esophagus and pharynx, and phlegmons are infrequent.

7. Genito-urinary Tract.—The urine is febrile. The urea is increased. Albumin frequently appears in the second week. Nephritis (in 6 per cent.) is dangerous. Uremia and hematuria are rare. The urine frequently shows the diazo reaction.

8. Spleen and Glands.—An early splenic tumor is palpable in 50 per cent. and disappears in the second week. The splenic pulp is fluid. Rupture has been described. Lymphadenitis was noted during the Crimean and Turko-Russian epidemics.

Anomalous Courses.—Light forms run a course of six to ten days, with less fever, fewer nervous symptoms, and less rapid pulse, and include the abortive type without eruption, and the ambulatory type. Very severe forms are the hyperpyretic (fatal in two to three days), or the adynamic.

"Brill's Disease." In 1910 W. E. Brill, of New York, described a febrile affection characterized by: (i) a short incubation of 4–5 days; (ii) brusque onset; (iii) chills, intense headache, and a facies expressing pain and apathy; (iv) fever, which rises suddenly, reaching 103–104° on the third or fourth day and continuing until the eleventh to fourteenth day when it falls by crisis; (v) an eruption, developing on the sixth day, which is maculopapular, dark red, and does not disappear on pressure nor appear in crops; and (vi) by negative Widal reaction, blood cultures and at autopsy, by the absence of typhoid lesions. The pulse runs 86–100 and the white cells average 11,000. Anderson's and Goldberger's immunity experiments identify the disease with European typhus and the Mexican type—Tabardillo, in investigating which Howard Ricketts lost his life. The Manchurian typhus is mild like Brill's disease, but Tabardillo is virulent. Brill's type has been found in most of our larger cities.

Differentiation.—This is not difficult if the case is seen from beginning to end, or during an epidemic. There are no absolute pathological or bacteriological criteria. Mistakes in the diagnosis of sporadic cases are often inevitable. In the initial stage only a probable diagnosis can be made.

In *smallpox* the initial scarlatiniform eruption in the angles of the arms and groins is characteristic. Morbilliform eruptions occur in both. Smallpox invades the face more than does typhus, and the relation of the eruption to the temperature remission is not observed in typhus.

Even at postmortem examinations, differentiation between the hemorrhagic form of typhus and *purpura variolosa* is frequently impossible.

Typhus.	Typhoid.
Invasion: sudden; chill, coryza.	Slower and more gradual; no coryza.
Roseola: more numerous and earlier, second to fifth day.	Usually less points of eruption (are exceptions, *e. g.*, measles-like); on extremities, as in Franco-German war.
Never in crops.	In successive crops.
Distribution on body and extremities.	On body; rarely on extremities.
Sparingly on face.	Almost never on face.
Flat, pale, washed, hyperemic, not disappearing upon pressure when they become petechial.	Papular, pink, sharply outlined, always hyperemic, disappearing upon pressure.
Course: more rapid.	Slower.
Face and eyes injected.	Paler; conjunctivitis rare.
Fever: abrupt rise, higher, less remission, if any; shorter; crisis, or short lysis, on fourteenth day.	Slow rise and regular stages.
Soon bed-ridden.	Oftener ambulatory.
Earlier and severer nervous symptoms.	Second week or later, and less severe.
Pulse: rapid and not dicrotic.	Dicrotism and slowness.
Spleen: early swelling, but less frequent.	Later, but lasts longer; more frequent.
Less meteorism.	Meteorism.
Stools: not characteristic.	Ochre stools.
Blood: leukocytosis.	Leukopenia, Widal reaction; bacteria in blood, urine, roseolæ.
Diazo reaction in both.	

Prognosis.—The prognosis depends upon the intensity of the toxemia and the complications—especially pneumonia. Death from toxemia usually occurs in the second week; from pneumonia in the third week. The mortality varies between 6 and 20 per cent., but reaches 50 per cent. in virulent epidemics and in patients over fifty years of age.

Treatment.—The treatment is that of typhoid, with the added indication of strict isolation. Open-air treatment in tents is indicated, with thorough disinfection of everything coming in contact with the patient. Cardiac stimulants are often required. Tr. iodi, ♏iv, t. i. d., appears to be beneficial.

EPIDEMIC PAROTITIS (MUMPS).

Definition.—An acute specific infection, characterized by a primary inflammation of the parotid glands.

History.—Hippocrates described every essential of mumps, but later it was confused with other parotitides.

Etiology.—(*a*) Its cause is unknown. (*b*) Most cases occur between six and fifteen years of age; isolated cases are seen in adults. (*c*) Most cases occur in the cold, moist, spring months. (*d*) Contagion is more frequently direct—perhaps before parotitis sets in—from person to person, than indirect. (*e*) It prevails largely in epidemics. Immunity is usually conferred by one attack, yet Catrin observed recurrence in 6 per cent.

Symptoms.—After an *incubation* of two or three weeks, the disease begins with fever (101° or even 104°), and in two days *parotid intumescence and pain.* The severity of certain epidemics determines such severe

initial symptoms as vomiting or convulsions in children; or a typhoidal condition in adults. The *characteristic parotitis* is clearly inflammatory—swelling, pain, temperature and interference in function. It is bilateral; Bouchoud said "Mumps have no singular;" in rare epidemics the parotitis is unilateral. In the early unilateral involvement, the face inclines to the affected side to relax the swollen tissues; in the later bilateral parotitis, the head is often bent forward to the same end. We know little of its pathology beyond what Virchow described—hyperemia of the parenchyma, interstitial swelling, catarrhal secretion in the ducts and contiguous lymphadenitis. The swelling about the gland may reach the eyelids, forehead, mastoid region or even the clavicle, effacing the features and even invading the pharynx and larynx; it is due less to periparotitis than to pressure on the facial and other veins. It is soft and does not pit. Compression of the masticatory muscles by the swollen glands makes opening the mouth difficult; chewing elicits pain because it excites salivary secretion which often cannot pass the turgid ducts. Compression of the Eustachian tube or external meatus may occasion tinnitus. The salivary secretion may be normal, increased or decreased. Suppuration is due to mixed infection. Wile maintains that a lymphocytosis of over 50 per cent. is pathognomonic.

In severe cases the *submaxillary and sublingual* glands are swollen (in 50 per cent. of some epidemics); they may be the sole seat of the disease. The parotitic swelling reaches its acme within four days and an equal period is necessary for its subsidence. The fever is remittent or intermittent, and falls by lysis.

Complications and Sequels.—1. The most important are metastatic *orchitis* and *epididymitis,* described by Hippocrates. In about a week fever returns (102° to 105°). Swelling, redness and pain in the testicle develop, often accompanied by general symptoms. In some epidemics, orchitis precedes or even occurs without parotitis. Urethritis is sometimes observed, analogous to the catarrh of the parotid duct. Testicular localization occurs in the sexually active testis, whence its rarity in children or the aged. Comby observed it in 30 per cent. of mumps among French soldiers. Orchitis or epididymitis is usually unilateral. Testicular atrophy occurs in 50 per cent. and if bilateral may occasion sterility. In women, ovaritis and vulvovaginitis are infrequent. Mastitis is rather more frequent, and may also be observed in men and boys.

2. *Other glandular organs.* Of these the lachrymal glands are most often affected, then the thyroid, with acute Graves's disease, pancreas, thymus and spleen.

3. *Other complications* are very rare. These are meningitis, otitis, polyneuritis, optic neuritis, endocarditis, nephritis, bronchopneumonia; suppuration or gangrene of the parotid or testis.

Diagnosis.—Osteomyelitis of the jaw, periostitis, lymphadenitis, jugular thrombophlebitis and furuncle should be excluded. Epidemic parotitis must not be confused with *secondary parotitis,* which results from trauma, or pus burrowing from the middle ear, or with mercurial, iodide or lead parotitis, and purulent metastasis in typhoid, pneumonia and erysipelas, in which the pyogenic virus enters by the ducts or settles from the blood

current. In *postoperative parotitis,* after laparotomy, trauma to the gland, from holding up the jaw, may promote infection.

Prognosis.—The prognosis is good; but 7 deaths occurred in 58,331 cases. The course is lighter in children than adults.

Treatment.—The fever is controlled as in other fevers, and diet, isolation, hygiene and rest abed for ten days are the same. *Pain* often necessitates the use of opiates. The local parotid or testicular inflammation is self-limited, and resists all attempts at abortion or mitigation. *Local heat* is soothing and better tolerated than the ice-bag. Complications are treated *symptomatically.*

ACUTE ARTICULAR RHEUMATISM (RHEUMATIC FEVER).

Definition.—An acute, febrile, non-contagious disease, with multiple articular involvement and with a tendency to heart complications.

Etiology.—Rheumatism is an acute infection, as evidenced by its occasional epidemiology; its tendency to recur like pneumonia and erysipelas; the autopsy findings of exudation, hemorrhage, cloudy swelling and acute splenic tumor; the frequency with which an atrium may be observed, such as an angina, and its indubitable affiliation with endocarditis, whose mycotic origin is established. The *virus is unknown;* Schottmüller's Streptococcus viridans, Horder's saprophytic streptococcus and other streptococcoid organisms are under suspicion.

Common symptoms occur in rheumatism and septicopyemia—temperature, anemia, articular metastases, endocarditis, leukocytosis and peptonuria. Some explain the presence of organisms by bacterial invasion during the death agony, since bacteria are seldom obtained by aspiration of the joints during life.

Predisposing Factors.—75 per cent. of cases occur in the first half of the year; heredity is conspicuous in 25 per cent.; 70 per cent. occur in males. The greatest susceptibility exists between ten and thirty years (50 per cent.); M. Miller collected 19 cases in nurslings. Trauma, overexertion, exposure and occupation account for its frequency in sailors, divers, bakers and laborers. Rheumatism constitutes 2 per cent. of American hospital admissions, as against 3 to 7 per cent. in London hospitals.

Acute articular rheumatism was described by Boerhaave, who suffered from the disease, and elaborated by his pupil, Van Swieten. Sydenham gave an admirable account.

General Clinical Picture and Course.—The onset is sudden, with moderate fever, *prostration, drenching sweats* and *polyarthritis.* The joints are involved in succession, the inflammation lasting a few days in one joint and then passing to many others. In adults and adolescents there is febrile urine, constipation, conjunctival injection, coated, moist tongue, irritability, restlessness, and fever which revives as fresh joints are involved. There is marked anemia and loss of weight. If *endocarditis* occurs, valvular disease is the usual sequence. The usual duration without therapy is three or four weeks. Recovery is usual, but death may result from hyperpyrexia and toxemia. Recurrence and a family tendency to rheu-

matism and valvular lesions are noted. The articular disease is directly influenced by the salicylates, cardiac localizations much less so.

Symptoms in Detail.—1. Prodromal Symptoms.—Regarding the angina or tonsillitis (12 per cent.), there are two conceptions: (*a*) that the angina is primary and rheumatic, and (*b*) that the arthritis following angina is pseudorheumatism (polyarthritis anginosa), characterized by exanthems, occurring chiefly in the arms and reacting poorly to salicylates. Other possible prodromes are laryngitis, chorea, erythema nodosum, albuminuria or gastro-enteritis.

2. Polyarthritis.—(*a*) *Localization.*—The most frequently involved joints are those of the lower extremity, particularly the knee (70 per cent.); usually one side is affected after the other; the ankles, elbows and wrists are next most frequently affected. Rheumatism may early involve the smaller articulations, such as the carpus—thought by Trousseau to promote endocarditis; it also may invade the spine, jaw, sterno-clavicular joints, synchondroses of the ribs, sacro-iliac or pubic articulations and the larynx. Rheumatism may be localized by the occupation, *e. g.*, in the arms of washerwomen. If a joint of an upper extremity is first involved, the opposite arm is usually next inflamed. (*b*) *In general* the arthritis is *ascending* and invariably polyarticular; Van Swieten spoke of it as "migratory inflammation" or "fleeting gout." Persistency in one joint always suggests pseudorheumatism (symptomatic or secondary forms). (*c*) *Signs and symptoms:* Polyarthritis is a better term than polysynovitis because the inflammation pervades all structures of the joint, the periarticular bursæ, cellular tissue, muscular insertions and tendons—indeed, the external edema is usually greater than the synovial effusion. The joints are swollen, red, tender and painful. Pain is the most prominent and frequent articular finding. Lasègue attributes pain to inflammation in the associated muscles and tendons, affirming that if these be suitably supported the joint can be moved freely (Lasègue's phenomenon). The contiguous tendon reflexes are lessened or abolished. (*d*) *Duration:* The inflammation remains in one joint from one to eight days, seldom longer, and its involution is shorter than its evolution. (*e*) *Sequels:* The joints become normal, and ankylosis reflects doubt upon a previous diagnosis of rheumatism. Suppuration results only from a most infrequent mixed infection.

3. Temperature.—The fever is directly related to the intensity of infection, the number of articulations involved, and to the development and progression of visceral complications. As Wunderlich noted, it is usually highest when the patient is first examined. The fever curve is in nowise cyclic and rarely exceeds 102° or 104°. The fever rises with each new localization, be it arthritic, endocarditic or pericarditic. The temperature drops when the sweats occur, and averages ten days. The fever is longer when treatment is commenced late. Kahler held that fever could occur independently of joint inflammation (febris rheumatica). Simple fixation of the joints reduces the fever, tachycardia and tachypnea. Chills suggest septicopyemia rather than rheumatism. Hyperpyrexia is exceptional; with Dr. Lackner, the writer saw a patient with a fever of 112° who recovered. Severe nervous symptoms, coma,

convulsions, rapid pulse, transitory paralysis, stertorous breathing and hyperpyrexia constitute "cerebral rheumatism;" it is less frequent since the introduction of the salicylates. The pulse exceeds 100. The respiration follows the fever.

4. Heart Complications.—These are an integral part of rheumatism and constitute *visceral* as distinguished from articular rheumatism. Heredity seems to be a factor in certain cases. Heart lesions occur as frequently in light as in severe forms. They are most frequent and most dangerous at puberty and increase in frequency with repeated attacks of rheumatism. Lasègue says "Acute rheumatism licks the joints, pleura and even the meninges, but it bites the heart."

1. *Endocarditis.*—This, the most vital complication, was first described by Pitcairn (1788). Bouillaud (1832) held that in every severe rheumatism, endocarditis was the rule. The frequency of endocarditis is about 20 per cent. Mackenzie finds endocarditis in 58 per cent. of first attacks, 63 per cent. of second attacks and 71 per cent. of third attacks. We might divide McCrae's series into thirds; in one third the heart is normal; in another third, it is doubtful; and in the last third, it is diseased. Hospital statistics, however, represent the severest cases. Clinically, endocarditis often escapes recognition and is found more frequently at the autopsy table than at the bedside. It is usually verrucose, and rarely ulcerative even in the fatal cases. To be certain that an endocarditis exists, two points are absolutely necessary: (*a*) Continued observation after convalescence; and (*b*) attention to signs of valvular disease other than the murmur, such as dilatation, hypertrophy, etc. Therapeutically we must protect the patient and ourselves by keeping him sufficiently long in bed. We may interpret the disappearance of a murmur in two ways: first, as the disappearance of a functional murmur; or, second, as the healing of a slight endocarditis, always the less probable issue.

Valves Affected.—The mitral valve is involved in 95 per cent. of the cases, the aortic in 23 per cent., and both valves in 18 per cent. The fever of fresh or recurrent endocarditis may be continuous with that of the arthritis but is often higher. A patient with an old valvular lesion and recent temperature more often has *recurrent endocarditis* than malaria, pyemia or tuberculosis, which are commonly confused with endocarditis. Endocarditis exceptionally antedates the polyarthritis.

Masked or atypical rheumatism embraces rheumatism masquerading under the guise of an endocarditis or trifacial neuralgia and yielding to salicylate treatment. Rheumatic pericarditis or endocarditis rarely occurs without arthritis—polyarthritis rheumatica *sine* arthritide—the rheumatic equivalent.

2. *Pericarditis.*—Rheumatism explains more cases of pericarditis than all other causes combined. Its frequency is given as 5 to 10 per cent. In children, especially, a subacute or latent course is most common. Baginsky found that adhesive pericarditis was the most common autopsy finding in rheumatic children.

3. *Myocarditis.*—Disturbance of the heart in the acute stage is myocardial and toxic or mycotic. Some dilatation occurs in every case.

Acute interstitial myocarditis[1] (in 28 per cent.), coronary disease and myocardial invasion by contiguity from endocarditis in the later stages are possible causes. Recovery is more common than chronic fibrous myocarditis. Myocarditis may cause sudden heart pain with weakness —the "heart rheumatism" of the laity—or transient palpitation, cardiac oppression, tachycardia or bradycardia.

The right heart may be dilated as in other fevers, but it disappears with convalescence. When accompanied by a murmur it may lead to an incorrect diagnosis of endocarditis. French writers describe an acute arteritis with severe pain over the artery and pain over its smaller branches in the extremities; pathologically only part of the vessel wall is affected and healing usually follows; gangrene may follow complete arterial occlusion. Endophlebitis is more common.

5. Skin.—Sweating is most profuse, constant and characteristic; its odor is sour or mouse-like. The skin is frequently macerated and *sudamina alba* or *rubra* are common. *Rheumatic nodes* are subcutaneous, as large as buckshot, on the fingers, hands, wrists, elbows and spine and are aggregations of round or spindle cells; their structure is identical with that of endocarditic vegetations. They may appear during or after an attack, or independently of acute seizures. They sometimes number even sixty and are symmetrical, hard and usually tender. Nodes occur most often in chronic cases or in 88 per cent., with severe endocarditis or pericarditis. If very numerous or large they indicate an unfavorable prognosis. Hillebrecht collected 80 cases in children under fifteen years and 37 cases in adults. In contradistinction to these fixed nodosities, Feréol has described more fugitive and less indurated nodes (*nodositiés cutanées éphémères*). Erythema polymorphum is regarded as an independent affection but in rheumatism the nodose form may occur. Some eruptions are septic or anginose. Purpuric eruptions are not uncommon.

6. Muscles and Bones.—*Myositis* is infrequent, though *myalgia* is common. Muscular atrophy may follow inflammatory extension from the joints to the muscles. "Muscle scars" are much more often due to trauma or other infections than to rheumatism. Some cases of albuminous *periostitis* are clearly rheumatic.

7. Respiratory Tract.—*Laryngitis* may be the atrium for rheumatic invasion, or may occur later as diffuse catarrh or local nodes. The rare acute edema of the larynx is very dangerous. *Pleurisy* may result from rheumatic or secondary infection (in 2 per cent.). Pleurisy is found in 54 per cent. of cases of endopericarditis. The so-called rheumatic pleurisy (*q. v.*) is almost always tuberculous. *Pneumonia* occurs in but 1 per cent. of cases of rheumatism not involving the heart, and in 66 per cent. of cases with endopericarditis. Most so-called pneumonias

[1] Romberg, Bret, Aschoff and Tawara describe nodules in the heart as rheumatic myocarditis; their specificity is disputed but all observers found them. They are perivascular, subendocardial nodules containing large cells and giant cells, and surrounded by a zone of lymphoid elements, plasma cells, and fibroblastic cells. The adjacent muscle bundles suffer from pressure, so that functional heart disturbance may result. They tend to cicatrization. The same heart nodes have been observed in streptococcic sore throat.

are pulmonary infarction, stasis or compression from pleural or pericardial exudates.

8. Digestive Tract.—Angina or tonsillitis occurs chiefly as a prodrome (*v. s.*). The tongue is coated, and moist except in the severest cases. Gastric symptoms are rare, though Damash considers them the most frequent prodrome. Constipation is the rule. Diarrhea occurs as prodromal enteritis or as a rare complication.

9. Genito-urinary Tract.—*Albuminuria* is transitory, late in the course, and mostly after thirty years of age. The percentage ranges up to 30. Albumosuria is frequent, especially during resorption. Cylindruria may occur without albuminuria. Renal infarction usually occurs late in the course of cardiac cases. *Acute nephritis* is seldom seen (0.3 to 1 per cent.); it occurs early, chiefly in endopericarditis, and is usually benign. But few of the author's cases in private practice show nephritis, though it is present in 50 per cent. of hospital patients; it is probably latent until awakened by the fresh toxemia and is probably due to exposure and alcoholism. Hematoporphyrin has been found and the characteristics of fever urine, and reduction of the chlorides.

10. Blood.—The red disks are reduced 25 per cent. The fever, anemia and intensity of articular involvement run more or less parallel. The white cells, in moderate cases, range between 10,000 and 15,000, and higher figures (20,000) are only reached in extensive endocarditis, pericarditis, pleurisy or pneumonia. The increase is in the polymorphonuclear neutrophiles. The eosinophiles vanish, but reappear with articular improvement; if increased in a florid case, the course will be mild.

11. Nervous System.—The relation of *chorea* to rheumatism is unsettled; *chorea,* rheumatism and endocarditis are associated in 50 per cent. of cases. Cases of chorea with rheumatic or endocarditic association usually run a slow and often a relapsing course. *Cerebral rheumatism* is very infrequent; developing acutely at the acme of the disease, with psychical alteration, hyperpyrexia, delirium, convulsions and coma, it frequently results in death. High fever may be a cerebral symptom, but cerebral symptoms may occur without high temperature. The author has seen three recoveries after mania, convulsions, meningeal symptoms and coma. Chronic *psychoses,* hallucinations, mania, melancholia or convulsions, may occur in convalescence; they are due to toxemic inanition and generally terminate in recovery. Brain *embolism* from endocarditis, hemorrhage and rheumatic meningitis are infrequent. *Spinal rheumatism* may be confused with myelitis, multiple neuritis, local myopathies adjacent to the joints involved, or cerebral embolism. In Kraus's clinic the author saw one fatal case characterized pathologically by meningomyelitis. *Multiple neuritis* may be rheumatic; usually following it, neuritis presents the usual characteristics, as pain and tenderness over the muscles and nerve trunks, paralysis, muscle atrophy, reaction of degeneration, anesthesia and abolished reflexes. The prognosis is good in multiple and mononeuritis.

The Eye.—Rheumatism may result in benign episcleritis, iritis or iridocyclitis. Optic neuritis, with or without atrophy, and retinal embolism or thrombosis occur exceptionally.

Acute *thyroiditis* is uncommon. Lymphadenitis is rarely marked. It occurs with arthritis, erythemata, endocarditis or pericarditis.

Prognosis.—The prognosis as to life is excellent and is generally proportionate to the severity and duration of the infection and the number of joints involved. The mortality is 2 per cent. but hospital figures are higher than in general practice. The *causes* of *death* are hyperpyrexia, the most important; cerebral rheumatism, chorea, peri-, endo- and myocarditis, pneumonia and lung infarcts. *Recurrence:* a second attack occurs in 25 per cent., a third in 12, a fourth in 4, and five to eight recurrences in 1 per cent. The prognosis is *less favorable in children.* Salicylate *therapy* influences the disease, its relapses, and, to some extent, the heart complications. At the onset it is impossible to declare the case light or severe. Involvement of the small joints of the hands and feet presages a severe course.

Diagnosis.—1. Rheumatism in Childhood.—In the very young the disease is most rare (3 cases in 85,000 nurslings) but becomes more common after the fifth year. Heredity is most potent in the young. Generally speaking, the joints are less susceptible and the arthritis less intense, while the skin, nervous system and heart are more often involved. The various phases of rheumatism occur separately—first arthritis, then endocarditis, later chorea, subcutaneous nodules, pericarditis and so on. The child rarely lies quietly, but tosses about; the tendons and fasciæ are involved more often than the joints. The pain is less and the arthritis may entirely escape recognition. The lower temperature is remarkable at this epoch of life, when high fever is often excited by trivial lesions. There is much less sweating, the urine is more nearly normal, and the anemia is especially marked. The differential possibilities are *acute osteomyelitis* (causing monarthritis), pyemia, gonorrheal arthritis, Barlow's disease, syphilis and hemophilic joints. *Endocarditis* may not directly follow the polyarthritis, but develops more slowly, insidiously, later and twice as frequently as in adults. Watson knew but three instances of acute rheumatism, before puberty, which escaped severe heart disease. This is due to the greater functional demands on the child's heart and to the aorta being narrow at the isthmus. *Pericarditis* occurs later in what Cheadle terms the "rheumatic progression," after the valvular lesion has developed, and is almost always found in necropsies on children with valvular disease. Rheumatic heart disease at this epoch leads to greater hypertrophy because of greater nutrition, to more marked dilatation because of greater tissue elasticity, and to more frequent recurrence. In the diagnosis, functional murmurs are not frequent in children. The pulse is often irregular; the precordium more readily bulges; and left-sided pleurisy is more frequent. Oppression over the heart, pain, cyanosis, dropsy, hepatic congestion, pulmonary infarction, dyspnea and fever are infrequent. Rheumatic children are more nervous. More marginate, papular and urticarial erythemata and more purpura develop in children. Large subcutaneous nodes (rheumatismus nodosus) are serious prognostically and suggest severe intercurrent valvulitis. Chorea is prone to develop with arthritis, heart disease, nodes or erythemata.

2. Rheumatoides.—Rheumatoides or *pseudorheumatism* are symptomatic arthritides resembling rheumatism. The nomenclature of Quincke, who speaks of *polyarthritis gonorrhoica, scarlatinosa, etc.*, is the least objectionable. (*a*) Polyarthritis *gonorrhoica* (*q. v.*) resembles rheumatism very closely and most frequently. (*b*) Polyarthritis *scarlatinosa* often most closely resembles rheumatism (*v.* page 265). With it have been observed endopericarditis, nodes, erythema and chorea. (*c*) Polyarthritis may complicate many other infections; *e. g.*, measles, smallpox, erysipelas, typhoid, angina, appendicitis, diphtheria, syphilis, pneumonia, etc.

3. Severe Types.—In grave types the visceral findings are least confusing when secondary in development to the arthritis. In the primary visceral type the findings (*e. g.*, cardiac) antedate the arthritis, but diagnosis is uncertain, until the joints are involved.

4. Gout (*q. v.*). 5. Arthritis Deformans (*q. v.*). 6. Tuberculous Polyarthritis (*q. v.*).

The *number of joints* involved is highly suggestive, but not diagnostic. Rheumatism and gonorrheal rheumatism are often polyarticular; diagnosis of the latter in women is often difficult for notable reasons. Puerperal infections and an acute monarticular osteo-arthritis in pregnancy or lactation may cause difficulty.

Treatment.—1. Diet.—Biot's results, with a strict milk diet, were extremely good; arrow-root, gruels, etc., may be given.

2. Clothing.—The patient should wear a flannel gown and blankets are more comfortable than sheets on account of the sweating. Maceration of the skin by the drenching sweats can be avoided by dusting talcum powder in the axillæ, etc., and sponging with warm water and alcohol.

3. Local Treatment.—Hot boric fomentations and salicylic salve are useful (℞—Acidi salicylici ʒj, adipis lanæ hydrosi ʒj). Cantharides blisters are indicated for tardy involution.

4. Rest.—Fixation in the most comfortable position lessens pain, fever, pulse and respiration-rate. The absolute dorsal decubitus for three weeks must be enforced because of the weak heart. Muscular atrophy may result from the use of plaster-of-Paris casts.

5. Salicylates.—Buss (1875) remarked their antipyretic affects, and Strickler and Maclagan (1876) discovered their remarkable action in rheumatism.

(*a*) *Effect on the Arthritis.*—The pain and swelling are usually relieved after three to five full doses—within twenty-four hours. Salicylates are the best analgesic in rheumatism, and morphine is rarely necessary after the first twelve hours. Salicylates are thought to cause articular hyperemia.

(*b*) *Effect on the Temperature.*—The fever falls coincidently with relief of the articular symptoms, and in the great majority of cases the acute stage is over within three days. In hyperpyrexia less influence is noted.

(*c*) *Effect on the Endocarditis.*—The weight of opinion is that endocarditis occurs as frequently with the use of salicylates, *i. e.*, salicylates are only analgesic and antipyretic. The writer believes that the salicyl-

ates are practically specific and that heart complications are lessened by shortening of the acute stage.

(*d*) *Relapses.*—Relapses are not more frequent with the administration of the salicylates than with the alkaline treatment. They are less frequent under the use of salicylates for two weeks after the initial symptoms.

(*e*) *Physiological Action.*—The smallest doses produce cinchonism, as fulness of the head or roaring in the ears. After larger doses dulness, headache, giddiness, a dusky and moist skin, disturbed hearing, and tremor develop. Toxic doses also produce eye-muscle paralysis, pupil dilatation, amaurosis, deafness, increased reflexes, restlessness, delirium, dyspnea, disturbed circulation, skin eruptions and green urine (indican and pyocatechin). Postmortem, visceral congestion and serous ecchymoses are seen.

(*f*) *Administration.*—Sodium salicylate is the most prompt, certain, soluble and least irritating preparation. Where it fails, other preparations are usually unsuccessful. All salicyl preparations are absorbed as sodium salicylate, produced by the alkaline intestinal juices. Grains xv of the natural or synthetic acid—as both are equally good—or grains xx of the sodium salt, are given every three hours until *salicylism* appears, *i. e.*, until slight *dulness, dyspnea* and *deafness* develop; ʒij–iiss are given the first day, when the interval between doses is lengthened to six hours. The effects are better in proportion as the disease is more acute, the inflammation greater, the number of involved joints larger, and the fever higher. It usually can be given by mouth in fluid, if the taste be disguised by wintergreen oil, syrup of ginger or syrup of orange; even then it is frequently nauseating.

℞—Sodii salicylatis		ʒvj
Sodii bicarbonatis		ʒiv
Syrupi zingiberis		℥j
Syrupi aurantii		℥j
Aquæ	q. s. ad.	ʒviij

M. et S.—Shake. Two teaspoonfuls every three hours.

Capsules may irritate the stomach, but powders are well tolerated, taken in starch wafers. In case the stomach is intolerant, salicylates may be administered by rectum in peptonized milk.[1] Inunctions are uncertain. *Aspirin* (acidum acetylsalicylicum) is much less irritating to the stomach than salicylate of soda, less antipyretic and less analgesic but more sudorific; it is given in doses of twenty grains at the same intervals. Aspirin rarely occasions an angioneurotic edema of the face, lids or oral mucosa and urticarial, scarlatiniform or pemphigus eruptions. Salol (phenylis salicylas) is given in the same dose at the same interval, but the kidneys must be carefully watched, because of the phenol component; it is far less efficacious.

The claim that the *alkaline treatment* decreases the heart complications and shortens the disease, is not proved, yet in the above prescription, the sod. bicarbonate lessens the gastric and renal irritation of the salicylates.

[1] Hypodermic injections of the salicylates in rheumatism are advocated (10 c.c. of a 20 per cent. sterilized solution of sodium salicylate to 100 pounds of body weight, fifteen minutes after a cocaine injection, every twelve hours).

Baginisky thinks the administration of gr. xv–xl of potassium iodide daily produces better results in children than do the salicylates.

(*g*) *Unusual Results.*—1. *Nervous.*—Delirium has been observed in chlorotics, neurotics and alcoholics. It may be violent and protracted. Convulsions are infrequent.

2. *Cardiac.*—Small doses stimulate the heart, and ʒij per diem seldom affect the heart, even with old valvular lesions. If the heart becomes weak or irregular, acute myocarditis is probably developing and the heart muscle must be stimulated by coffee, strychnine or camphor. Slight irregularity without weakness is no contra-indication. Thorne has observed collapse from aspirin.

3. *Respiratory.*—With antecedent or concomitant bronchitis or tuberculosis, the lungs should be explored for possible congestion due to the remedy. Cardiac depression, sudden respiratory failure and violent expiratory dyspnea are observed in a few cases.

4. *Renal.*—Caution is necessary in old nephritics, because the salicylates are excreted by the kidneys as salicylic or salicyluric acid; however, albuminuria or nephritis directly due to the rheumatic virus, indicates, rather than contra-indicates, the salicylates.

5. *Alimentary.*—Vomiting and diarrhea may result from salicylic irritation or inhibition of the digestive ferments.

Diagnostic Therapy.—Senator claimed that salicylates are specific in true rheumatism and inert in pseudorheumatism; there are certain exceptions—some rheumatics resist salicylates, and salicylates are of some value in the arthritic type of grippe, but usually are useless in other arthritides.

Convalescence.—Absolute rest must be enforced for three weeks to save the heart. Antecedent tonsillitis indicates tonsillectomy.

DENGUE.

Definition.—An acute specific infection, extremely contagious, and characterized by fever, prodromal eruption, violent articular and muscular pains, and a secondary polymorphous eruption.

Etiology and Epidemiology.—The actual cause is uncertain. Graham found, in the blood cells, an organism resembling the Plasmodium malariæ, but small and unpigmented. It is transmitted by the bite of the Culex fastigans. Contagion is disseminated by indirect infection, fomites, bites, etc. It is carried by pilgrims, sailors and emigrants. The disease spreads with remarkable rapidity. Its home is in the tropical and subtropical zones and it prevails chiefly in the hot months. It is remarkable that the coast ports are chiefly invaded, and closely contiguous inland towns and higher places are exempt. The first epidemic, in 1779, in Java, was described by Brylon, and Benjamin Rush wrote of the Philadelphia epidemic in 1780; it was called "Front Street Fever," on account of the squalid condition of the houses along the river front. It has invaded Europe, Asiatic Turkey, South America and the United States. It has reached Savannah, New Orleans, Ohio and Virginia.

No immunity is conferred by one attack, and it may recur two or even four times.

Symptoms.—Incubation lasts one or two days.

1. Febrile Stage.—The fever *rises quickly* to 102°, 104° or even 107°, with a chill, severe frontal headache and intense pains in the back and limbs, especially the knees, which incapacitate the patient. The names "break-bone fever," of Rush's description, and "dandy fever," given because of the mincing gait which results, bring out this salient feature. The pulse averages 100. Many *joints* are *swollen*, one after another. The *muscles* are *painful*, the *face is suffused* and swollen, the conjunctivæ are injected and the flow of tears is increased. Total anorexia, coated tongue, and great thirst develop; vomiting is occasional and constipation the rule. The *prodromal hyperemic rash* lasts one to five hours, and is frequently absent. Prostration, delirium, convulsions and coma may develop. This stage lasts three days ("three-day fever") and ends with profuse sweating. Fever is absent in the lightest cases.

2. Exanthematous Stage.—The polymorphous eruption on the fourth day on the face, hands, forearms and chest, is scarlatiniform, morbilliform, urticarial, erysipelatoid, vesicular or even pustular. It is present in 80 per cent. of cases, lasting three days, and followed by desquamation, itching, sometimes by falling of the hair. An inflamed throat, lymph-gland enlargement, parotitis and orchitis occasionally occur. The temperature varies from subnormal to high. The pain recurs often. Herpes, splenic tumor, otitis, albuminuria and leukocytosis are absent.

3. Convalescence begins in seven days. There is prostration, emaciation and anemia. In no other disease of equal duration is such marked constitutional disturbance noted.

Complications.—Gastric hemorrhage, endocarditis, nephritis, pneumonia, pleurisy or ulcers very rarely develop.

Prognosis.—Though the morbidity is enormous, the highest recorded death-rate is 0.5 per cent. (10 deaths in 100,000 cases in Smyrna). Relapse may occur in one or two weeks.

Diagnosis.—The geography and the presence of an epidemic are significant. The exanthemata, rheumatism, yellow fever (*q. v.*) and epidemic *erythema exudativum multiforme* may be suggested. *Influenza* is differentiated by its occurrence in all climates, seasons and inland as well as on seaboard, its usual respiratory catarrh and complications, its more severe nervous, intestinal and cardiac symptoms, and the greater frequency of herpes, splenic tumor, otitis and albuminuria. In the rare *acrodynia*, observed in France, Belgium, Turkey, Persia and Mexico, Scheube gives the following differential points: Digestive disturbance; facial swelling; pains in the extremities, sometimes with anesthesia; spastic symptoms; afebrile course; and erythema multiforme.

Treatment.—*Isolation* and quarantine are necessary. *Pain* is the prominent indication, for which acetphenetidinum and salicylates serve fairly well, though they are much inferior to opium.

YELLOW FEVER.

Definition.—A specific infection, transmitted by the mosquito, endemic in the American tropics and subtropics, and characterized pathologically by degeneration of the liver and kidneys, and clinically by fever, icterus, albuminuria, adynamia, and hematemesis.

Etiology.—Transmission by the mosquito (*Stegomyia calopus*) was suspected by Carlos Finlay, in 1881, but was demonstrated by the investigating board composed of Walter Reed, James Carroll, A. Agramonte and J. W. Lazear. Dr. Carroll recovered from a severe attack, but Dr. Lazear and Dr. Myers, of the English Commission, died from inoculation. The Stegomyia breeds not only in stagnant street pools, but also in cesspools and sewers, whence its special danger in unhygienic tropical towns. Voluntary inoculations by mosquitoes, which twelve days previously had bitten yellow-fever patients, produced yellow fever in nonimmunes; Finlay failed to prove his suspicion because he allowed the mosquitoes only three to six days in which to develop the germs which they had ingested, whereas they must live twelve days before they can convey infection. The mosquito carries infection only when it bites the patient in the first three days of the disease. Transfusion of blood of an infected person, taken in the first three days of the disease, also conveys infection. The virus is filterable. As in malaria, the tropical foci, the hot summer and autumn seasons, the invasion of low altitudes, river courses and coast towns, humidity, and exposure to night air, are merely conditions suitable to the mosquito. Frost stops infection because it kills the mosquito. Direct contagion does not occur. Nurses and physicians are not infected from contact with yellow-fever patients. Persons going from the City of Mexico to Vera Cruz may acquire the disease, but on coming home with it they do not cause its dissemination, because the mosquito is not present. Reed's committee exploded the theory that fomites, clothing, etc., were infective. Volunteers remained in rooms occupied by yellow-fever patients and slept in their soiled linen without contracting the disease. During the American occupation, Col. Gorgas stamped out the disease in Havana, but 405 cases developed in 1905–8, from the New Orleans epidemic. No cases developed since December, 1908.

Predisposition.—The lighter races are more prone to infection. Immunes are merely those who have had light and unrecognized infections. Yellow fever is kept alive in endemic foci by mild cases, especially among the Creole children.

Foci and Epidemiology.—The habitats of yellow fever are (1) Mexico, the Antilles, Mexican Gulf and Caribbean Sea; and (2) Africa, from Senegal, through Sierra Leone, to the Gold Coast. The first clear description of yellow fever was in 1686, but yellow fever was known before the discovery of America. It reached this country in the sixteenth century and has extended as far north as Boston, St. Louis, Quebec and Chicago. In Philadelphia (1793, 1797–1798), 4041 died in a year; in New York, 2080; and in New Orleans, in 1853, 8000 died. In New

Orleans, in 1905, there were about 9000 cases and 1000 deaths. Europe is seldom, but South America is frequently invaded.

General Clinical Description.—Incubation averages three days. Prodromal symptoms last less than a day; they are malaise, fatigue, headache, dyspepsia or colic. The disease is divided into the following stages:

I.—The *stage of congestion* begins with frontal or retroöcular headache, the earliest and most prominent symptom, sometimes a chill, and flushed face; the conjunctivæ are injected and sometimes icteric; the eyes are sensitive to light, the lids puffed and the expression denotes pain, restlessness and confusion, comparable to the alcoholic facies; backache radiating into the calves, fever rising to 104°, and a full, tense pulse of 100 to 110. The tongue is slightly coated dorsally. The febrile urine is very early albuminous. The skin is intensely red and dry. The epigastrium is tender early and there is marked gastric intolerance of food, water and medicine. This stage averages two days.

II.—The *remission stage,* or deceptive stage, occurs in some cases and is so-called because of the subsequent severe symptoms. The fever, pulse and the pain remit. Slight jaundice may appear.

III.—*The Reaction Stage.*—Slight temperature, the "black vomit" (*vomito negro*) of blood, hemorrhage into the nose, intestines, uterus or skin, hiccough, epigastric pain and tenderness, liver enlargement, and icterus mark this period. The urine becomes scant and laden with bile pigment, or its secretion is suppressed; the pulse becomes weaker, and insomnia, prostration and restlessness may appear.

Various *issues* are possible: Gradual amelioration; fatal anuria; profuse hemorrhages, which end in collapse, and rarely in recovery; profound icterus, which results usually in death; or death from cardiac insufficiency.

(*A*) *Variations in intensity* include (1) abortive, ambulatory, atypical forms, especially in children; (2) the usual severe forms; and (3) the foudroyant type, rapidly lethal in one to two days.

(*B*) *Visceral Forms:* (1) The hepatic type, with two weeks of the status typhosus, often with normal temperature and sometimes with hemorrhages; (2) the uremic type; (3) the hemorrhagic; (4) the cardiac; and (5) nervous or atactic type.

Special Symptomatology.—1. Fever.—Fever is highest in the first stage, and averages 104°. It may fall on the second or third day to nearly normal in benign cases or in the ominous uremic type. High fever after the second day is a poor prognostic. In the second stage fever is usually 99° to 100°. In the third stage a high antemortem rise is frequent. In convalescence it is subnormal.

2. Nervous Symptoms.—Nervous symptoms are often absent in both benign and malignant cases. Headache is common in the congestive period. Delirium is infrequent, and in 80 per cent. of cases, is uremic. In children, alcoholics and in the debilitated the status typhosus or meningo-encephalitic symptoms (convulsions, unequal pupils, Cheyne-Stokes's breathing and coma) are possible. Pathologically, few changes are found except the slight degenerations common to the febrile state, punctate hemorrhage or bile tinging.

3. Circulatory Symptoms.—(*a*) The pulse in the congestive stage is 100 to 110 and tense; on the third day, 70 to 80; in the second stage it is still lower in benign and more rapid in severe cases (110 to 140) and is small; in convalescence it is slow (50 to 60, or even 30). Faget considers slowing of the pulse with maintenance of the fever almost pathognomonic. (*b*) In severe forms the first tone of the heart becomes weak and embryocardia is found. The heart is rarely irregular or intermittent. In the third stage, cardiac dilatation is common (in 70 per cent. of autopsies), due to myocardial degeneration. Cardiac action is exaggerated when uremia is present. (*c*) There is no anemia. The white cells are decreased; the polynuclears are relatively increased.

4. Respiratory Symptoms.—Respiratory symptoms may be due to intercurrent, or more often to cardiac or renal, complications.

5. Digestive Tract.—Hiccough, epigastric pain, tenderness and anxiety may be symptomatic of cardiac distress, premonitory of the black vomit, or indicative of disturbed circulation in the medulla.

6. Icterus (Yellow Fever, Typhus Icteroides).—Jaundice usually develops on the third to fifth day; its pathogenesis is obscure. It is frequently absent in children, in early death from uremia and in 50 per cent. of cases which recover.

7. Liver.—The liver is slightly enlarged and tender. Pathologically, it has a light cooked, fatty and anemic appearance.

8. Urine.—*Albuminuria* occurs in 88 per cent. of cases in the first three days. Leucin, casts and bile pigment are common. The kidneys, anatomically, are pale or yellowish, and, microscopically, show great fatty degeneration of the tubules, which in severe cases are choked by desquamated epithelium and casts. The glomeruli show few changes. *Uremia* causes 33 per cent. of fatalities; it is marked by delirium, nervousness, convulsions, decreased or suppressed urine (fatal in 97 per cent. of cases), rapid or Cheyne-Stokes's breathing, and a tense pulse. Death in coma may occur on the third day. Uremia is rare in children.

9. Hemorrhages.—They result from fatty degeneration or actual rupture of the bloodvessels. Hemorrhages may occur from the mouth, stomach or intestine. The *black vomit*, gradually transuded blood altered by the gastric acid, occurs on the fourth to sixth day, the "coffee-grounds" vomit; the stomach at autopsy shows the black contents and dark discoloration, epithelial desquamation and fatty change in the muscularis. Hemorrhage from the small intestine is equally frequent. *Epistaxis* is often profuse. *Uterine hemorrhages* are severe and in pregnant women cause abortion. Less frequent are skin petechiæ and muscular hemorrhages.

Complications and Sequels.—These include secondary infections such as endocarditis, pericarditis, pneumonia, erysipelas, parotitis, arthritis, suppuration and gangrene; coincident typhoid, malaria, influenza and insolation; and postfebrile psychoses.

Diagnosis.—The diagnosis is readily made from (1) the flushed, semi-intoxicated facies; (2) icterus; (3) the early albuminuria—the most valuable finding in doubtful cases; (4) black vomit, gastric intolerance and epigastric tenderness; (5) hemorrhages, and (6) the slow pulse, which

is absent in some severe, but therefore clear, cases. Pernicious *malaria*, with icterus, is distinguished by the blood (plasmodium and anemia), enlarged spleen (always absent in yellow fever), the infrequency of early icterus, albuminuria and hemorrhages (except hematuria). This form of malaria is rare in cities, whence the constant suggestion of yellow fever, when more than sporadic cases are reported. *Weil's disease* and acute yellow atrophy are readily distinguished.

Prognosis.—The mortality of epidemics varies from 10 to 85 per cent. The death-rate averages 30 per cent. The mortality, according to age, is 33 per cent. (one to three years), 14 (four to eighteen years) and 37 per cent. (nineteen to forty years). Unfavorable events are hepatic, renal or cardiac insufficiency, the typhoid state, high fever with hepatic symptoms and hemorrhages.

Treatment.—1. PROPHYLAXIS.—No greater triumph of hygiene exists than the stamping out of yellow fever during the American occupation of Havana. It illustrates not only the efficiency of the investigating commission, but also the possibilities of medicine in administrative capacity with unlimited discretionary power. In Havana the death-rate was 500 yearly, until Gorgas cut it down to *nil*. The patient should be isolated, not to prevent direct personal contagion but to keep mosquitoes from the infected person. Healthy individuals avert infection by the mosquito by the same measures employed in malaria, *e. g.*, screens, and extermination of breeding foci. The Stegomyia is found outside the habitual yellow-fever foci, and after biting patients from abroad, may inoculate other persons.

2. SYMPTOMATIC TREATMENT.—(*a*) In the *first stage*, fluidextractum aconiti, ℥iij, and sodium salicylat., gr. xx, may be given every two to four hours, until the congestion and pains are relieved. Coal-tar products are depressant. Calomel, gr. ¼ every hour for five doses is followed by castor oil. Overmedication is to be avoided because of the gastric irritability. Violent cerebral, uremic or pulmonary symptoms are sometimes treated by phlebotomy. (*b*) In the *last stage*, the black vomit contra-indicates any fluid, food or medication by mouth; warm peptonized milk and normal salt solution are given by rectum; morphine by mouth is dangerous. (*c*) Digitalis is especially good, and is beneficial even when *cardiac failure* is absent; saline solution, by rectum or subcutaneously, is better than intravenous transfusions. The patient must be kept absolutely quiet. (*d*) *Uremic* symptoms are treated in much the same way. Water is given freely by the bowel. (*e*) *Nervous* manifestations necessitate cool spongings, bromides, morphine and hyoscine, as in typhoid. (*f*) Vomiting is relieved most efficaciously by chopped ice. (See TYPHOID.)

HYDROPHOBIA (LYSSA; RABIES).

Definition.—An acute specific infection, peculiar to carnivora, the bites of which convey infection to other animals and man. The disease is characterized by tonic and clonic spasms of the esophagus, respiratory and other muscles and by a fatal evolution. It was described in ancient Jewish, Egyptian and Indian writings and by Democritus in the fourth

and fifth centuries, B.C. The first adequate account was by Youatt (1828).

Etiology.—In Germany, from 1887 to 1894, only 16 cases occurred because of the strict laws. In Russia, France and America it is far more common. The disease has been eradicated in England, Denmark, Norway, Sweden, Switzerland and Australia. Dogs are most frequently affected and the chief cause of dissemination (90 per cent.); next come wolves, cats and cattle; and finally skunks, horses, deer, pigs, rabbits and even fowl may be inoculated. The *virus* is found *in the nervous tissues most abundantly, in the saliva* and also in small quantities, in tears, sputum, semen, urine and milk, and in the adrenals. Its propagation is along the nerve trunks, thus reaching the salivary glands (not by way of the blood stream). Noguchi cultivated small granular pleomorphic bodies which produce the disease on inoculation. Inoculation occurs from bites of infected animals, which transmit the disease even during incubation. Punctured or deep wounds and those in the nerves or muscles are most dangerous; 10 per cent. of those bitten by mad dogs and 62 per cent. of those bitten by rabid wolves and cats die. In exceptional cases, rabies results from being licked by an infected dog, kissing an infected person or dissecting an infected body. The disease prevails chiefly in warm weather in the "dog days" and most laws are based on this assumption; but it is also common in cool weather. In Paris most cases occur in March and May. Sixty per cent. of cases occur in males and 40 per cent. under fifteen years of age.

The *incubation* period varies, averaging 40 days: (*a*) It is shorter in children; (*b*) if the wound is on the face or head, incubation is very short and the symptoms very severe; (*c*) it is longer and the symptoms less intense when the point of infection is on the body, because the clothing tends to clean the virus off the teeth.

Symptoms.—Three somewhat arbitrary stages are described:

First or Prodromal Stage.—The scar may become painful. Psychical alteration is expressed by depression, irritability or extreme anxiety. Headache, hyperesthesia, anorexia and some elevation of the temperature and pulse-rate are usual. Sometimes the voice is hoarse, the pupils are somewhat dilated and premonitions of dysphagia are experienced. This stage lasts one or two days.

Second or Excitation Stage.—(*a*) The *excitement* is more *motor* than psychical. The cutaneous and deep reflexes are increased. There is such hyperesthesia that the least sound or light, the slightest breath of air, or food and water in the throat, induce violent reflex spasms, whence the patient's dread of these excitants (photophobia, aërophobia, hydrophobia). Tonic spasms, following attempts at swallowing affect chiefly the muscles of the mouth, the hyoid elevators and the laryngeal and respiratory muscles. Even without laryngeal involvement the respiration is labored, with jerky, deep or sighing inspirations, during which the shoulders are lifted and the epigastrium protruded. The spasms may extend to the trunk and limbs. (*b*) The *psychical* excitement is marked at the time of the spasm by restlessness and later by horror or even mania. Delirium tremens may be simulated. The mind is usually

clear between the spasms. The patient rarely attempts to injure attendants, although such an impression may be made by the peculiar growling sounds occasioned by pharyngeal and laryngeal spasm and the "frothing at the mouth," due to inability to swallow the increased flow of saliva. Less constant symptoms are fever, salivation and lachrymation; tremor, erection and ejaculation; increased pulse-rate; scanty, albuminous urine (25 per cent.), often with casts. The excitation stage lasts one and a half to three days.

Third or Paralytic Stage.—This stage, less common in human rabies, in animals may be marked from the beginning and the other stages may be lacking or rudimentary—the "dumb" rabies as contrasted with the furibund type. The spasms and excitement cease, the pulse becomes fast, small and irregular, the pupils are wide and irregular, sweating is profuse, swallowing becomes impossible and paralyses develop, mono-, para- or hemiplegic, or of the acute ascending type of Landry. The patient dies, unconscious, of cardiac collapse, rarely of asphyxia or convulsions. The third stage seldom lasts more than eighteen hours.

Autopsy Findings.—Pathologists are not unanimous in regard to specific findings. Minute hemorrhages and special congestion of the medulla and cord characterize all convulsive states. Babes's *rabic tubercles* are aggregations of leukocytes around the vessels and nerve cells. The latter may degenerate. Rapid diagnosis of rabies may be made according to von Gehuchten and Nélis, by examination of the sympathetic and cerebrospinal ganglia, under the capsules of which are found *accumulations of epithelioid and lymphoid cells,* in animals, only, which die of the disease. The bodies described by Negri and Pavia (1903) in the cell processes may be protozoal parasites or artefacts.

Course and Prognosis.—In twelve hours to ten days—on the average two to four days—all untreated cases die, 10 per cent. in the first day and 60 per cent. in the first three days. Högyès's is the only recovery verified by inoculations of the saliva. Paltauf's autopsies, on persons under Pasteur treatment and dying from accidental causes, indicate that 9 out of 10 persons bitten destroy the virus in the central nervous system, for the inoculated medulla gives rabies to guinea-pigs; and that the tenth case fails to destroy it and therefore dies. Tonin alleges a cure under salvarsan.

Diagnosis.—After bites by supposedly rabid animals, microscopic examination of the plexiform ganglion of the vagus can be made and also subdural inoculations in rabbits, which die in two or three weeks with paralytic symptoms. *Tetanus* of the cephalic and hydrophobic type is distinguished by the invariable trismus. *Hysteria* (pseudohydrophobia, lyssophobia) in one bitten by a dog may at first cause suspicion, but rarely any protracted doubt; the initial spasmodic dysphagia and irritability are not followed by other symptoms of hydrophobia, and the clinical evolution alone is distinctive.

Treatment.—Prophylactic.—Pasteur, in 1881, found the rabic virus in the nervous tissues. "The virus of the street," as Pasteur called it, varies in virulence and subdural inoculations from the brains of

rabid dogs, into rabbits, killed them in periods varying from fourteen to twenty days. By inoculating a series of rabbits, the incubation period is progressively shortened to a fixed period of seven days; this "fixed virus" is attenuated by desiccating the spinal cord of the rabbit with caustic potash. Animals were inoculated with virus which had been desiccated for two weeks, then with virus a little stronger, until finally immunization was secured against inoculations which previously would have been fatal. In 1885 human beings bitten by rabid beasts were thus treated by two inoculations daily for fifteen days and immunization was secured, for the slow rabic incubation gives ample time for treatment. Statistics show that the former mortality of 80 per cent. in untreated bites in the head or face has been reduced to 1.25 per cent.; the mortality of 60 per cent. in those bitten in the hands is reduced to 0.75 per cent.; the mortality of 80 per cent. in those bitten in the covered parts of the arm and trunk, and of 15 per cent. in those bitten in the leg, is reduced to 0.25 per cent. *The total mortality has been reduced to 0.5 per cent.* Treatment requires two weeks. As Park says, "it is a contest of speed between the passage of the virus from the wound to the brain by way of the nerves and the development of immunity by absorption of vaccines through the circulation;" if "the patient escapes infection up to the end of two weeks, few, following the full course, develop the disease (2 or 3 per 1000)." As sequences of Pasteur's treatment, neuritis, bulbar or spinal, and Landry's paralysis are very rarely found (Babes, 1905). One-half of those treated by Pasteur's method are treated unnecessarily. W. H. Park formulates the *indications* thus: (i) Keep the animal and if it has rabies, give the treatment. (ii) No treatment is necessary if at the end of the week, the dog has not developed rabies. (iii) If the animal has escaped, judge as far as one can by its conduct (giving the patient advantage of any doubt). (iv) If the animal has been killed and no Negri bodies are found, give the treatment, as microscopic examination is negative in 3 per cent of cases. (v) If the dog has been killed and no microscopic examination made, give the treatment if rabies prevails.

2. Primary Wound Treatment.—The wound may be sucked by the victim, but by no other person. It should be washed with strong antiseptics, burned with nitric acid (the best remedy), formalin, pure phenol, etc., and exposed surgically, treated with the live cautery and packed with gauze, so that healing may be delayed for over a month.

3. Late Treatment.—In developed cases the mortality is 100 per cent., whence curative therapy is out of the question. The treatment of the *convulsions* is similar to that of tetanus, and nutrition is maintained by nutritive rectal enemata. Morphine and inhalation of chloroform are better than chloral and bromides and should be given until they show their full physiological effects. Cocainization of the mouth and pharynx may overcome the pharyngeal spasm incident to swallowing milk and water and to medication. Tracheotomy is useless for the respiratory spasm, which reaches further than the larynx and involves the muscles of the chest and diaphragm.

FEBRICULA.

Definition.—A fever of short duration. If only of twenty-four hours' duration it is called *ephemeral fever;* when it lasts a few days, it is called *febricula;* when attended by herpes, *febris herpetica;* or when accompanied by gastric disorder, *febris gastrica.*

Febricula is no morbid entity, but embraces (*a*) benign or abortive forms of various infections, such as rheumatism, typhoid, paratyphoid, malaria, scarlatina, tonsillitis and pneumonia; infections with the Bacillus coli, proteus or pyocyaneus; and (*b*) febrile dyspepsias, especially those in children. Gastric fever or febrile gastritis is possibly produced by toxins in the food. Sewer gas never causes gastro-intestinal fever.

Symptoms.—The three common symptoms are (*a*) chill or chilliness; (*b*) sudden rise of temperature; and (*c*) a slow crisis. The onset is abrupt, usually without prodromes. The fever rises quickly to 101° to 103°, or higher in the young, with the usual signs of toxemia, headache, coated tongue, anorexia and febrile urine. Herpes is frequent. Bronchitis, acute splenic tumor, albuminuria and in children, cerebral toxemia, are sometimes present. The term ephemeral is inappropriate, as the average duration is three days.

Diagnosis and Treatment.—The diagnosis is by exclusion, and the treatment is expectant. Rest in bed is advisable as a precaution against some longer and insidious malady; the bowels should be unloaded, the diet restricted and a drop or two of tincturæ aconiti and spts. ætheris nitrosi ℥ss given every three or four hours.

GLANDULAR FEVER.

Definition.—An acute infection, characterized by pharyngeal infection, short, high fever and painful cervical lymphadenopathy. It was described as *Drüsen fieber* by Pfeiffer, in 1889. Its *etiology* is unknown but it is evidently an infection and frequently epidemic. It is usually seen between five and eight years of age, and during the colder months. Park West observed an epidemic of 96 cases in Bellaire, Ohio. Hochsinger and Kretz maintain that the adenopathy is the first obvious sign and sequel to nasopharyngeal infection.

Symptoms.—The symptoms begin without prodromes, with fever of 101° to 103°, pain in the head and neck, redness of the throat, dysphagia, nausea, vomiting, pain in the limbs and abdomen. On the second day the cervical lymph glands become swollen and tender bilaterally, especially back of the upper part of the sternomastoid. The inguinal and axillary glands are involved in 75 per cent. of cases. The glands may reach the size of an egg. After ten to twenty days the swelling subsides, having lasted longer than the fever, which, after a few days, falls by crisis. In some instances a persistent, dry cough suggests involvement of the bronchial glands, and umbilical pain of mesenteric localization (in about 40 per cent.). The spleen is enlarged in 60 and the liver in 90 per cent. Suppuration is exceptional. Acute nephritis, endocarditis, herpes and otitis media are occasionally observed.

Prognosis and Treatment.—The prognosis is good. The absence of a rash excludes the adenopathies of varicella, scarlatina and measles, and the generalized glandular swelling excludes the local swelling due to simple pharyngitis. The treatment is expectant.

MILIARY FEVER.

Definition.—An acute infection, marked by fever, sweating, miliary eruption, nervous symptoms and precordial anxiety. It first appeared in England after the Battle of Bosworth (1486) and was known as the "English sweats" (*sudor Anglicus*); its mortality reached 50 to 80 per cent. In 1529 it caused 3000 deaths in Dantzig. There have been 200 epidemics between the epidemic in Picardy, in 1718, and that in Poitou, in 1887. It is highly contagious. Short epidemics have occurred in Italy, Switzerland, Germany and Austria, with a death-rate of 5 to 25 per cent. In 1906 an epidemic of over 6256 cases prevailed in northern France.

Symptoms and Treatment.—In mild outbreaks the symptoms are fever, profuse periodic sweating and miliary eruptions. The eruption may be erythematous or morbilliform (miliaria rubra), or without redness of the skin (miliaria alba); desquamation follows. In severe cases there are marked cerebral toxemia, rapid pulse, precordial distress, palpitation, splenic tumor, prostration and hemorrhages into the skin; death results in a day or two. Its duration averages ten days. Convalescence is tardy and treatment supportive and expectant.

WEIL'S DISEASE (ACUTE FEBRILE JAUNDICE).

Definition.—An acute infection, characterized by fever, jaundice, herpes, enlarged spleen and liver, sore throat, muscle pain and albuminuria. It was especially described by Weil (1886). Nicolaysen describes 63 epidemics in Norway and Sweden in the last fourteen years; the morbidity was 3 to 28 per cent.

Etiology.—Its etiology is unknown, whence it may be a morbid entity or may include several diseases. It occurs in 90 per cent. in men between twenty-five and forty, especially in butchers and alcoholics, in the warmer seasons. The Proteus fluorescens has been found, but the *intravitam* findings are usually negative. Some cases have occurred in this country; the author saw 9 cases in Chicago.

Symptoms.—The symptoms begin suddenly with fever, chill, headache and severe *pains* in the extremities. The *fever* reaches 103°; it remits and falls by crisis or lysis after five to eight days. The nervous symptoms may resemble typhoid. *Jaundice* develops on the third day, due to catarrhal angiocholitis; bile appears in the urine and the stools are often acholic; the author has twice seen deep jaundice without bile obstruction. *Muscular pain* and tenderness are intense, especially in the calves. Articular pains are not uncommon. *Herpes* is very common. The *rapid pulse* contrasts sharply with the slow pulse of simple catarrhal icterus. In 2 cases the author observed blood-stained bronchitic

sputum. Digestive symptoms are common, as furred tongue, vomiting, epigastric pain, abdominal distention and diarrhea. The *liver* is enlarged and tender in more than 50 per cent. of cases, the *spleen* in over 66 per cent., and the *lymph nodes* are frequently affected. The urine is albuminous in over 50 per cent.; acute nephritis is frequent; hematuria occurs occasionally and uremia exceptionally; the urea and the amount of urine are decreased during, and increased after, the seizure (Chauffard's nitrogenous and polyuric crises).

Diagnosis.—Weil's disease must be differentiated from recurrent fever, sepsis, typhoid, suppurative angiocholitis (*q. v.*) and acute yellow atrophy (*q. v.*).

Prognosis.—Convalescence is usually established in two weeks; the mortality may reach 11 per cent. Recurrence in 40 per cent. may be anticipated if the splenic tumor persists; renal or hepatic insufficiency, pregnancy and hemolysis render the prognosis less favorable.

Treatment.—The treatment is directed to (*a*) the intoxication, for which calomel and salines are indicated; cold-water enemata increase the renal flow, and, with it, the excretion of toxins; (*b*) the jaundice, for which salines are given to lessen duodenal catarrh; (*c*) the albuminuria, for which a milk diet is indicated; and (*d*) muscular pains, for which heat and massage with chloroform liniment, are most beneficial.

MILK SICKNESS.

Definition.—A disease acquired by man from the milk or meat of cattle suffering from the "trembles." Cows with the disease, after being driven too rapidly, suffer from anorexia, injection of the eyes, vertigo, trembling and convulsions ending in death. Young horses and sheep may also acquire the disease. Milk sickness occurs chiefly in newly settled countries, as in the Southwestern states, and it is still seen in North Carolina. Its origin and pathology are unknown. Jordan and Harris describe a Bacillus lactimorbi.

Symptoms.—In man the nervous symptoms are frequently conspicuous, as restlessness, convulsions or coma, which appear after a few days of malaise. The tongue is swollen and heavily coated, the breath fetid, the stomach painful and irritable, the fever low, high or sometimes typhoidal. The issue is either fatal, in acute forms, within a few days, or recovery may result after weeks. Flesh, butter, cheese and milk, from infected animals may, even when boiled, cause death when given to dogs.

ROCKY MOUNTAIN FEVER.

It occurs in the Bitter Root Valley of Montana and also in Wyoming, Nevada and Idaho. Howard Ricketts found the disease was inoculable and he and King found the medium of infection was the wood-tick (Dermacentor occidentalis). Ricketts (1909) discovered a bacillus like that of hemorrhagic septicemia. Cases develop in the early spring, and are marked by chill, fever lasting one or two weeks, rapid pulse, epistaxis, albuminuria, rapid breathing, bronchitis, splenic tumor, rapid anemia,

moderate leukocytosis, slight icterus, muscular pains and a widely diffused macular rash, which develops on the third day, disappears on pressure at first, but afterward persists on pressure. The liver and spleen are enlarged. Quinine may be beneficial in some cases. The death-rate may reach 70 to 90 per cent. in Montana, yet in Idaho it runs between 2 and 3 per cent. T. B. McClintic died a martyr to the disease, as Ricketts died of typhus.

FOOT-AND-MOUTH DISEASE.

Definition.—Epidemic stomatitis, aphthous fever, or aphthæ epizooticæ is one of the zoönoses. Loeffler and Frosch have shown that the virus is ultramicroscopic, passing in full virulence through porcelain filters. The disease, first described by von Valentini, in 1695, chiefly affects cattle, sheep and pigs, but sometimes also goats, horses, dogs and even fowl. In *cattle* there is fever, swelling of the mouth, salivation and the formation of small, clear vesicles on the tongue, gums, lips, udders and hoofs. These vesicles soon become opaque, and bursting, form small ulcers; emaciation is rapid, and the milk becomes yellowish and mucoid. Embolism or fatal apoplexy may result. Recovery is usual in ten days. The disease spreads very rapidly. It is observed in Hungary, Germany, France, Switzerland, England and America, Chicago being the centre of a wide epidemic in 1914.

Etiology and Symptoms.—*In man* infection occurs from diseased milk (as proved on himself by Hertwig), butter and cheese, but not by diseased meat. Open wounds may be directly infected. Cattle have been inoculated from man. The incubation is three to five days. There are fever, salivation, aphthous ulcers, angina, nausea, vomiting and vesicles on the skin—which have caused confusion with vaccinia. Hemorrhages occur in severe epidemics. Ebstein suggested that foot-and-mouth disease may be kindred with the ordinary aphthous stomatitis of nurslings or pregnant women. Children are more endangered than adults. The death-rate is 2 to 8 per cent. Recovery is usual in three weeks.

Treatment.—Prophylaxis concerns the boiling of suspected milk, the segregation of diseased animals or human subjects, and vaccination, as discovered by Loeffler. A dram of 5 per cent. solution of potas. chlorate, t. i. d., should be given, and the aphthæ should be touched with borax or silver.

RAT-BITE DISEASE.

Rat-bite fever, known as Sokudu in Japan, closely resembles recurrent fever and in some ways, lues. The bite heals, and after two to five weeks, inflammation, even ulceration develops at its site, with intermittent fever, chills, headache, vomiting, an erythematous eruption, eosinophilia and local and general lymphadenopathy. After a fever-free interval of three to five days, the symptoms recur; the mortality is 10 per cent., usually from nephritis. Crohn (1915) collected 52 cases. Salvarsan is recommended.

PSITTACOSIS.

Psittacosis is a disease of birds, characterized by loss of appetite, general weakness, diarrhea, convulsions and death. It is communicable to man, infection being commonly acquired from parrots. The disease occurs in England, Germany, France and Italy. It is characterized by atypical pneumonia, attended by profound and usually fatal toxemia.

METAZOAN INFECTIONS.

DISEASES CAUSED BY CESTODES.

TAPE-WORMS were known to the Egyptians, Hindus and probably the Jews. They may cause symptoms by the presence of the mature parasite in the bowels or of the larval form in the viscera.

I. Tenia Solium.—The Tenia solium or pork tape-worm (Linnæus, 1752) exists as the cysticercus cellulosæ in hogs and rarely in dogs, rats or deer, as "measles." The adult form is peculiar to man. Cysticerci (the larvæ), found in the intermuscular tissue, particularly of the undersurface of the tongue, in the masseters, shoulders, neck and diaphragm, are easily seen with the naked eye and very easily with a low-power lens, as small, opaque, white bodies. They are vastly more frequent in Asiatic and German than in American pork. Man is infected by eating raw or partly cooked "measley" pork; the cysticerci develop in the upper ileum into the tenia ("band or ribbon-like") solium (referring to its usual solitary appearance, though 2 or 3, and even 20 or 41, have been found). The worm measures 2 to 8 meters in length. Its head (*scolex*) is round, often gray or black in its upper part, is smaller than a pin-head, and is attached to the upper ileum; it has 4 sucking disks and 20 to 30 hooklets, arranged in an anterior, more numerous row, and a posterior row, whence the name "armed" tape-worm; it has no real mouth. Its neck is thin and not jointed, to which the segments, "*proglottides*," are attached. These number 800 or 900; the segments measure 10 mm. in length by 5 or 6 mm. in width; those near the neck are narrower and shorter. About a yard from the neck they become squarer, and lower down they are smaller again. Each proglottis is hermaphroditic, containing male and female reproductive elements. The uterus is central and vertical, with 7 to 15 lateral branches; only a few of them mature, chiefly those in the lower links; the ova (31 to 36μ) are very numerous in each maturing segment and each one shows a shell and an embryo armed with six hooklets. The tenia reaches its full length of two to four yards in three or four months, when the lower segments are detached and voided; if the worm breaks and a yard or so appears in the feces, segments do not reappear in the feces until three months later. An ovum ingested by a hog or man loses its shell in the stomach and the freed embryo passes into the tissues, where, encysted, it becomes again the cysticercus cellulosæ (or larva).

Symptoms of the Tenia Solium in the Human Intestine (Intestinal Teniasis).—Many, possibly most, cases present no symptoms; they may first develop after passing segments are noted by neurotic subjects. In other cases headache, itching, pupillary inequality, emaciation and dyspeptic disturbances arise, as anorexia (or less often ravenous appetite), nausea, abdominal pains, constipation or diarrhea. Stiles, in an experimental infection on himself, thought that he could feel the parasite move. Several feet of tape-worm have been vomited; in these rare instances the individual suffers from both the intestinal and larval stages. Anemia is uncommon. In children reflex convulsions are possible. The parasite may live for 10 to 15 years. The *diagnosis* is determined by the segments and ova and the *prognosis* is good.

Treatment.—As to *prophylaxis:* segments of the tape-worm should be burned; meat inspection should be general; "interstate" meat is well inspected; pork should always be thoroughly cooked. Hogs obtain ova from human feces.

1. To cure the disease a light and chiefly *fluid diet* should be given for two days, when food is withheld for eighteen hours; this often causes the parasite to release its hold on the mucous membrane; a meal of onions and herring may be given just before drugs are administered.

2. A *preliminary cathartic* cleanses the bowel, allows more direct access of the vermifuge to the parasite and prevents its regaining its hold when once detached.

3. *Anthelmintics:* Of these *pomegranate* (granatum) ranks first; a decoction is made of 3 ounces of the root with 8 ounces of water, reduced to 4 ounces by evaporation and taken in divided doses within three or four hours; its active principle is the expensive pelletierine, which is given in doses of 3, 5 to 10 grains and is followed by a purge in an hour (pelletierinæ tannas). *Male fern* (felix mas) is almost equally effective; it is given as the oleoresina aspidii, or extr. felicis maris ether., ʒiss–iiss; it may be given in syrup; both are followed by a saline purge in three or four hours. Castor oil is never used, because it promotes absorption of the male fern, which causes toxic symptoms, as icterus, claudication or amaurosis; Sidler collected 78 cases of poisoning with 15 per cent. mortality and lasting blindness in 25 per cent.; Leichtenstern advises that more than 10 gm. (ʒiiss) of the ethereal extract should never be given to adults (ʒj or less to children); that it never be given on an empty stomach or given two days in succession. The drug decomposes easily, whence reliable preparations must be obtained. Combinations may be made by giving after the decoction of pomegranate, spts. chloroformi (♏xv) for four doses or croton oil (♏ij) for one dose; thymol (*v. i.*) is effective. Search should be made for the head of the parasite, without removal of which treatment is unsuccessful. If it is not found another trial should be made after a few weeks.

℞—Oleoresinæ aspidii,
Tr. vanillæ . . . . . āā gtt. xlv
Acaciæ,
Sacchari albi . . . . . āā ʒj
Aquæ . . . . . ʒj
M. et fac emulsum.
S.—Take in one dose.

Symptoms of the cysticercus cellulosæ (somatic teniasis) result when ova of the Tenia solium are taken into the stomach; ova may reach the stomach from the intestines by antiperistaltic movements during vomiting or indirectly by handling worms voided from the bowels. Cysticerci in the hog produce few symptoms. In man, symptoms depend largely on the number and localization of the larvæ. (*a*) In *the muscles and skin,* a few cysticerci cause no symptoms; numerous cysticerci cause pain, tenderness and difficult movement. Diagnosis is possible only on excision of the subcutaneous nodules. In Stiles's collection of 155 cases the localization was muscular in 20 per cent. and cutaneous in 3 per cent. (*b*) In the *brain* (in 75 per cent. of Stiles's series) symptoms depend on their location; they are often absent; if the location is in the cortex, Jacksonian epilepsy is frequent, and if in the medulla, diabetes may develop. (*c*) *Other localizations.* They may be found clinically in the eye (v. Graefe) and at autopsy in the lungs, kidneys or liver. There is no *treatment* except surgery.

II. Tenia Saginata (Mediocanellata).—This form (Goeze, 1782) is the most frequent of large tape-worms in America and Europe. It is the "fat," "unarmed" or beef tape-worm. Its *cysticercus bovis* is found in masseters of beef seven times as frequently as in other muscles or in the heart or brain. They are not so readily seen with the naked eye as the cysticerci in pork. Ingestion of rare beef containing cysticerci produces the Tenia saginata in man, in whom alone the adult worm is found.

The intestinal parasite differs from the T. solium in the following points: (*a*) its *head* is larger, measuring 2 mm. and is square; it has no hooklets but four sucking disks, which are larger, more forward and are surrounded by pigment. (*b*) Its neck is much shorter. (*c*) Its *proglottides* are longer (16 to 22 mm.) and broader (5 to 10 mm.); even 1000 segments have been found. (*d*) The *uterus* contains 15 to 35 branches, which are less dendritic than pronged or forked; in diagnosis the uterus is more important than the ova or size of the segments, which are hard for the practitioner to distinguish (Stiles). (*e*) The *ova* measure 30–40 by 20–30μ. (*f*) The *parasite* is larger and longer, measuring 4 to 10 meters. Its symptoms and treatment are those of the Tenia solium. Ingestion of the ova or segments from human feces produces the cysticercus mediocanellata, which is common in beef but extremely rare in man.

III. Other Teniæ Occurring in Man.—The *Dibothriocephalus latus* (Linnæus, 1782), or the Russian tape-worm, is found chiefly in Russia, Poland, Norway, Sweden, Switzerland and Japan. It is rarely found in this country unless imported (30 cases reported, Stiles), but fish in Lake Superior have recently been infected. Its cysticerci are found in the muscles and peritoneum of the pike, trout, salmon and perch and in man's intestines develop into the dibothriocephalus. Its head is flat or ovoid, possesses no hooklets and attaches itself to the intestines by two lateral grooves (suckers). Its neck is long and its segments broad, square and may number 3000 to 4200. It is long, measuring 8 to 10 yards. It may produce a profound anemia which in severe cases closely resembles the pernicious type in its general symptoms and blood findings.

The *Tenia elliptica* or cucumerina (Dipylidium caninum) is a form, the larvæ of which develop in the lice of dogs and the adult form exists in the intestines of dogs, sometimes in those of cats and rarely in man.

The *Tenia flavo-punctata* (Hymenolepsis diminuta), the larvæ of which exist in beetles and Lapidoptera, exists in rats and 12 human cases are recorded (Rausom).

The *Tenia nana* (Hymenolepis nana) is the dwarf tape-worm. It measures but ½ to 2 inches in length. Its cysticercus stage is in the intestinal wall of the rat and its adult stage in the rat's intestine. Stiles decided that it is much more frequent in man than previous accounts would indicate and in some places is the most common type. Its head contains four suckers and one row of hooklets. It may occur singly or in myriads. Aspidium alone is of therapeutic benefit.

Tenia Echinococcus (Echinococcus Disease) (Zeder, 1803).—We may first consider the *adult parasite as inhabiting the intestine of the dog* chiefly and rarely of the wolf, fox or jackal. The head is small and is provided with four suckers, and from 30 to 50 hooklets arranged in a double row. The segments number only three or four, and only the end one matures; it measures but 0.6 by 2 mm. but contains even 5000 ova. The worm is small, measuring but 4 to 5 mm. in length, is very delicate and white, so that it readily escapes detection. When the ovum reaches the stomach of another animal, the hog and ox chiefly, and less often the horse, sheep or man, it loses its shell and migrates to the various organs. The *geography* of echinococcus disease and mode of infection is as follows: In Iceland, where dogs and men live together, every seventh person becomes infected. In Australia both men and sheep are often infected. In Europe the malady is much more common than in America, where the reported cases in 1901 numbered 241 (Lyon); most of these were foreigners. Icelanders brought the disease to Manitoba in 1874.

Infection occurs by licking of the hands by the dog, the tongue and anus of which often come in contact. Infection by contaminated drinking water or vegetables is less common.

General Symptoms and Pathology.—The small embryo, freed of its shell, penetrates the intestinal wall and wanders into the muscles, peritoneum or radicles of the portal vein or cava, so that any tissue or organ may be infected, and stops when it reaches vessels too small for it. Once lodged, its hooklets disappear and a cyst develops, which is small at first but gradually reaches large or enormous proportions; cysts may contain 30, even 70 pints of fluid. Its layers consist of a granular endocyst and an outer finely lamellated structureless capsule; a fibrous layer develops outside from reaction of the tissues to the foreign body. After a while a number of "daughter" cysts arise from the endocyst; these are first "buds" and then miniatures of the original mother cyst; from these daughter cysts, which number about a dozen, "granddaughter" cysts arise by an identical process. Allen in one case found 8000 daughter cysts. Finally from the endocyst of the maternal and daughter cysts there develop "brood capsules," in which small buds arise, which gradually become scolices (the heads of which will be intestinal teniæ in the animal which ingests them). Some cysts do not develop scolices and

are called sterile. In animals the daughter cysts may grow outward (exogenous cysts). The well-grown cyst contains fluid (*v.* Liver Echinococcus); it is clear and neutral, has a specific gravity of 1005–15 and contains succinic acid, inosite (possibly sugar) and much sodium chloride but no albumin, unless the parasite dies and cystic inflammation develops, then the fluid may become buttery, gelatinous, purulent, brownish or grumous; the cysts disappear as well as the membrane, though the hooklets remain a long time. When the parasites are alive a toxin is present, for collapse often develops when the cyst ruptures or is punctured (anaphylaxis).

The parasite lives for years, in one instance for twenty years; after a variable time, unless suppuration intervenes, the cysts become cheesy, perhaps calcified or ossified.

The parasite may rupture into the serous sacs, bloodvessels (causing embolism), air passages, urinary tract or externally (see pages 580 and 665); in 50 per cent. it is fatal within five years.

Visceral Localization.—In 1912 cases (the total reported by Davaine, Finsen, Mosler, Neisser and Cobbold) the liver was involved in 51 per cent., genito-urinary organs in 10, intestinal canal in 9, lungs or pleuræ in 9, brain or cord in 7, bones in 3, heart and vessels in 3, and other organs in 8 per cent. Vegas and Cranwell reported 970 Argentine cases of which 64 per cent. were hepatic. Thomas, in 809 Australian cases of single hydatid cyst, found the lungs involved in 16.5 per cent. Hooklets have been obtained by lumbar puncture. There are reported 142 cases of renal echinococcus, 78 cases with localization in the bones and 23 in the pelvic bones, 25 in the pancreas, 80 in the spleen and 55 cases with cardiac or pericardial localization; 23 thyroid cysts have been reported.

Echinococcus of the Lung.—Many cases produce no symptoms and are unexpected findings at the postmortem. Dieulafoy described three clinical stages: (*a*) the initial, (*b*) the tumor stage and (*c*) the suppurative or rupture stage. (*a*) The *initial stage* often has no symptoms, but may be marked by scanty hemoptysis, cough, emaciation and possibly fever, all of which may closely simulate incipient phthisis. (*b*) In the *tumor stage*, there are bronchial (or weak) breathing, increased fremitus and circumscribed dulness, which occur in the lower lobes and twice as frequently on the right side as on the left; it sometimes causes bulging of the chest wall. There is a sharp transition between it and the note of the sound contiguous lung. Dislocation of the heart and compression of the lung may occur. The *x*-rays often confirm the physical signs. The dulness curves upward, but is quite different from the curve of pleuritic effusion which is concave upward. Tumor of the lung gives a larger, harder flatness. Puncture reveals a clear, aseptic fluid (*v. s.*). (*c*) In the third *stage*, as in liver echinococcus, rupture may occur into the pleura (in which primary disease is 25 times as rare). Pneumothorax may result. Far oftener rupture into a bronchus occurs, with pain, dyspnea, collapse and sudden "mouthful" expectoration of pus, small vesicles, lamellated cyst wall and hooklets. At the time of rupture a curious urticaria develops (Finsen) which is probably toxic, as the fluid

from echinococcus cysts produces it when inoculated into animals. Profuse hemoptoë is common. If it comes from the liver, the sputum is ochre-yellow from undissolved bilirubin, even when there is no direct connection with the bile vessels; cholesterin is often present. The vesicles or cyst wall may cause suffocation. Rupture may also occur into the pericardium, stomach or peritoneum. In this stage hectic fever is usual.

There is an unusual form of echinococcus, the *multilocular form*, probably due to a separate worm, the clinical features of which are described under hydatid disease of the liver, to which it is almost exclusively confined. Virchow first described it in 1856. It occurs in Bavaria, Würtemburg, Switzerland, Austria, Austrian Tyrol and Russia; 6 imported cases have occurred in America. It is unknown where the ordinary form is most common. It is rare, few more than 100 cases being recorded. Sixty per cent. occur in males between 20 and 50 years of age. The ova are spheroidal and measure 100 to 170 micromillimeters. The great difference from the ordinary form is the outward (exogenous) growth of the cysts. The irregular and diffuse cystic proliferation is an unfavorable feature. The cysts contain a gelatinous material (sometimes simulating colloid cancer), surrounded by intersecting fibrous strands, which gives the liver a porous appearance, like a sponge or cheese with air cavities. In some vesicles there is cheesy material, with some resemblance to the granulomata. The vesicles sometimes suppurate.

Echinococcus cysts belong to surgery, though spontaneous recovery is not infrequent; the operative mortality is 10 per cent., the expectant, 60 per cent.

DISEASES CAUSED BY NEMATODES.

I. The Ascaris Lumbricoides.—This most common intestinal parasite in man, described by Linnæus, 1758, occurs oftenest in children and with relative frequency in the insane. Unlike the cestodes, no intermediate host is required. The female measures 7 to 12 and the male 4 to 8 inches in length; it is smooth, pointed at both ends and has transverse rings and four longitudinal ridges, a white one dorsally and ventrally and two brownish ones laterally. The oval reddish ova measure 0.075 x 0.058 mm. and have a thick capsule; 60,000,000 eggs may develop in a single female. Its mode of infection is obscure. Though they usually occur singly or in pairs, in the upper small intestine, they may exceptionally develop in such numbers that they obstruct the intestine. The ascaris may rarely rupture through an intestinal ulcer. It has passed through a perforated appendix. With or without vomiting, ascarides may pass from the stomach into the esophagus, nose, bronchi, lungs, middle ear, the common or hepatic duct, causing asphyxia, gangrene of the lung, etc.

Symptoms are not usually pronounced, though anorexia, salivation, offensive breath, constipation, colic and diarrhea occur in some cases. Malnutrition is sometimes marked. The parasite secretes irritating, volatile aldehydes and fatty acids, which may cause constitutional symptoms. Irritability, grinding the teeth, muscular twitchings, con-

vulsions or even meningeal symptoms may develop in nervous children. The author doubted the existence of the *typholumbricosis* of Chaffard, Marie and Tauchon, until in one adult case, with fever and nervous toxemia, instantaneous subsidence of all symptoms occurred when the parasites were removed.

Treatment.—After a fast of one-half to one day, *santonin* should be given, gr. ss–j, for a child, and gr. ij, iij or v, for an adult, followed by a saline cathartic (never by castor oil); if unsuccessful it may be repeated on two or three successive days, though toxic symptoms sometimes develop, as xanthopsia (yellow vision), collapse, urticaria (which may result from the parasite itself), vomiting or convulsions. The *fluid-extractum spigeliæ* (℥j) may be given with or without the santonin or calomel. These remedies do not kill the parasite, but merely render it uncomfortable.

II. Oxyuris Vermicularis (Seat-worm, Pin- or Thread-worm).—The female in length measures 10 and the male 4 mm. Ingested ova develop in the small intestine, where the worms mature and cohabit; afterward they exist chiefly in the lower colon and rectum. The eggs remain in the mother until the worm is expelled, though she occasionally "aborts" in the bowel. Infection occurs in those who are the most uncleanly, as in children and the insane. Infection may occur from water and green vegetables, and reinfection is not uncommon from scratching the anus during sleep and thus crushing the parasite; Eichhorst frequently found ova in the minute fecal particles about the anus and Zenker repeatedly demonstrated their presence under the finger nails. As the parasites migrate at night, the symptoms are greatest at that time; they may be found on the bed sheet. The anal itching and rectal irritation are often excessive; periproctal suppuration may be excited. If a vaginal discharge moistens the perineum (and only then) they can wander into the vagina, causing irritation, sexual stimulation or masturbation. Disturbance of sleep and appetite, nervous symptoms and anemia may result. Diarrhea is due to large numbers of parasites.

Treatment.—Santonin may be given in obstinate cases, but local measures are usually sufficient, as high enemata containing small proportions of carbolic acid, quassia or turpentine. They should be repeated daily for two weeks. Itching is palliated by application of 0.5 per cent. carbolic salve to or within the anus. A thymol salve on the anus kills worms and eggs.

III. Trichina (Trichinella) Spiralis; Trichinosis.—Though Tiedemann (1822), Hilton (1832) and Paget (1835) saw the parasite, Owen (1835) first fully described it. Leidy saw it in the hog in 1847. Zenker (1855–60) first described its pathological and clinical bearings; he found 4 cases in 136 autopsies.

Swine are infected probably from eating offal. From the clinical and etiological stand-point, the (*a*) *muscular or larval stage in hogs* should be considered first. The small encapsulated, "hair-like" (trichina) worm lies in the abdominal, diaphragmatic, psoas, laryngeal and other muscles. It lives for a long while, even twenty to twenty-five years,

without causing symptoms; calcification rarely occurs in swine. *Man is infected* by eating smoked, "cured" or insufficiently cooked pork; animals may be accidentally or experimentally inoculated, especially the rat, guinea-pig, rabbit, less often the cat and rarely the dog. Relative to its alleged presence in fish or worms, the trichina has been confused with other parasites. (*b*) The *adult or intestinal stage in man* is the next phase. When eaten by man, the small capsules (larval form in swine) are digested and the worms liberated; in the small intestine they mature (the females measure 3 or 4 mm., and the males are half as long) and the females are fecundated in two or three days. The female trichinæ produce 1000 to 2000 embryos at once or possibly in succession, which stage requires another week (from fecundation of the female to birth of the embryos). At the end of the second week the embryos migrate, entering the lymph vessels and then the veins, by which they reach the voluntary muscles, which they seek for their glycogen. They lodge between the muscle fibers, enter them and attain (*c*) *the mature larval form,* which is identical with the larval stage (*a*) in hogs. They measure 0.5 to 1 mm. in length. As a result of tissue reaction against the foreign bodies, a capsule develops in about six weeks, which surrounds one or more parasites. The capsules at first transparent, grow opaque and, after months, calcify and later the trichinæ calcify also.

Frequency.—Though trichinæ are about 20 to 40 times as frequent in American as in German swine, the disease is more common in North Germany where raw sausage, Westphalian ham and smoked ham are freely eaten. Seymour reports infections in Boston among Italians eating raw ham (prosciutto). Fränkel denies that German trichinosis results from American pork. Williams of Buffalo, in 505 autopsies, found trichinosis in 5 per cent. Müller of Dresden found it in 0.98 per cent. of 1939 postmortem examinations. Other statistics usually show lower figures than Müller's, and like the above, are taken from accidental postmortem findings. The disease occurs sporadically or epidemically. Perhaps a thousand epidemics occurred in America. In Germany the epidemic at Hedersleben numbered 337, and that at Emmersleben 250 cases.

Symptoms.—Symptoms may be absent or slight, particularly in sporadic cases, and with moderately severe infection, as evidenced by accidental autopsy finding of encapsulated parasites.

In clear cases the symptoms are divisible into *two stages:* (*a*) The preliminary, *gastro-intestinal* stage, which begins in two or three days after eating the diseased meat; the symptoms are not constant either in sporadic or severe epidemic cases; they are anorexia, nausea, vomiting, colic and sometimes diarrhea, which may resemble the evacuations of cholera. (*b*) The *invasion symptoms* begin in one or two weeks and correspond to the parasitic migration. (i) *Fever* is usual, but seldom rigors. It results from some toxin the parasites elaborate; it may rise to 102°, 104° or 106°, its course is remittent and intermittent, it lasts a few days to even two or three months, and falls by lysis. Symptoms attendant on any fever may be present, as headache, febrile urine or

quickened pulse. In severe cases, as in Zenker's, typhoid may be suggested by delirium, epistaxis, dry tongue, status typhosus, diazo reaction (in 80 per cent.), bronchitis, albuminuria and kindred toxemic symptoms. Typhoid and trichinosis coexisted in the cases of Zenker, Fischer and McCrae. (ii) *Acute diffuse myositis* is always most suggestive. Its intensity varies from mild forms of "muscular rheumatism" to the most severe and characteristic involvement; there are pain, tenderness, swelling, flexion to relieve muscular tension, and *edema* (25 per cent.) in the face and other locations. The worms most curiously select only the striated muscles, so that the edema observed in the abdomen and thighs, for example, never invades the scrotum or vulva. The eyelids are often affected greatly and early. Invasion of the eye muscles causes pain on moving the eyes; pain during mastication, phonation and deglutition marks penetration of the masseteric, laryngeal and pharyngeal muscles by the parasite; dyspnea (25 per cent.) results from involvement of the diaphragm and intercostal muscles. W. G. Thompson records frequent corneal hemorrhages. The tendon reflexes may disappear. (*c*) *The blood* presents two striking features: first, the *leukocytosis* (15,000 to 30,000) and second, the *eosinophiles*, normally 0.3 to 4 per cent. of the leukocytes, rise as high as 30 or 86 per cent. and though they increase in the other parasitic diseases, this rise is most peculiar if not pathognomonic. The eosinophilia averaged over 20 per cent. in more than half the cases (Thompson) and is greatest at the time when the trichinæ enter the muscles. (See Plate XVI, Fig. 5.) In an experimental study Graham observed the embryos in the arteries of rats and in hemorrhagic lesions in the lungs, and Stäubli found them in the heart's blood and in the blood from the ear of infected guinea-pigs. His method consisted in laking the blood with 3 per cent. acetic acid, centrifugating and examining the sediment. W. W. Herrick and T. C. Janeway (1909) using Stäubli's method, found the parasites in the human blood in 2 cases of trichiniasis. The blood is best obtained from a vein at the elbow by means of a small syringe. They thought it is certain they also migrate along the connective-tissue spaces. (*d*) *Profuse sweats*, polyuria, paresthesia, urticaria and anemia are inconstant manifestations. On the 24th day a second edema appears.

Diagnosis.—This is based on (*a*) the possibility of infection, as by eating poorly cooked pork, (*b*) the discovery of trichinæ in the blood, the uneaten portions of suspected meat or perhaps in the feces or cerebrospinal fluid; (*c*) their detection by removal of small slivers of the biceps or pectorals for microscopic examination, especially the parts near the muscular insertion; (*d*) the myositis with edema; (*e*) signs of insufficiency, *e. g.*, in the ocular or diaphragmatic muscles and (*f*) leukocytosis with eosinophilia.

Prognosis.—This depends on the number of parasites ingested. Choleraic and typhoidal symptoms, dyspnea and pneumonia are most ominous. Moderately severe early diarrhea is favorable. Children die less often than adults. Of the 15,000 recorded cases, 830 resulted fatally. According to Stiles, the German mortality averages 5.6 per cent. In the epidemic at Weimar there were no fatalities in 108 cases. In the epidemic at

Hadersleben 30 per cent. died. In certain epidemics the mortality was 70 to 100 per cent.

Treatment.—(*a*) *Prevention* comprises the feeding of hogs with clean food, government inspection of meat, and thorough cooking (160° F.). (*b*) In the *preliminary stage*, active and repeated purgation should be ordered, for moderate and early spontaneous diarrhea is prognostically favorable; calomel should be followed by salts and castor oil; male fern, santonin (*v. s.*), thymol gr. v, t. i. d. benzene ℥ij–v, t. i. d. and oil of turpentine ℥v–x are recommended; glycerin is now discarded. (*c*) In the stage of *muscular invasion*, the fever is treated as in other infections, the myositis by local applications of ice and by narcotics, and the general nutrition by food and stimulants.

IV. Ankylostoma (Uncinaria).—A disease, due to the hook-worm, whose development is favored by warmth and filth; attacking those coming in contact with damp earth or water infected with the larvæ; and characterized by the ova in the stools, progressive anemia, weakness, and alimentary, developmental and other disorders. The uncinaria duodenalis (ankylostoma duodenale) exists in two forms, that of the old and that of the new world. Ankylostomiasis was first well described by Piso (1648) in Brazil and the parasite was recognized in Milan by Dubini (1838). It occurs chiefly in the tropics and subtropics, including the Philippine and Sandwich Islands; according to Thornton, it is the most dangerous of all tropical diseases. In Egyptian chlorosis it was found by Bilharz and Griesinger, and is said to be present in nearly every autopsy made there. It is endemic in Italy ("bricklayers' anemia") and also in Belgium, France, England, Germany, Hungary and the Balkans. In India it is found in even 80 per cent. of the healthy coolies and in 300 autopsies in Assam it was present 299 times. Stiles (1902) described the new-world type, caused by the Uncinaria (or Necator) Americana, which is also found in Africa and East Indies. Thirty per cent. of Porto Rican deaths result from uncinariasis, and Ashford and King state that 90 per cent. of the rural and 50 per cent. of the urban population have the disease; during six months in 1905, 18,865 cases were treated in Porto Rico, of which 33 per cent. died. Capps in 1903 collected 50 American cases of the European type. It is possibly the most important disease of our Southern States. Dock and Bass estimate that over a quarter of the Southern, apparently healthy, population, suffer from this malady.

In the Westphalian mines, Tenholt himself treated over 4000 cases. It was the cause of the "tunnel anemia," observed in building the St. Gothard tunnel. It is disseminated by Italian and Polish workmen. Infection comes from the food, drinking water and earth which are infected by dejections from persons suffering from the disease. Looss and C. A. Smith proved that it may enter by the skin, producing the "ground itch," common in the Southern States. Dock and Bass believe the skin to be the sole mode of infection. The larva penetrates the skin in a few hours and, by way of the heart, lodges in the lungs and *bronchi*, whence it is expectorated or swallowed; in the latter case the larva reaching the intestine, sheds one skin and acquires a buccal

capsule by which it adheres to the mucosa; from the mucosa it draws blood and sheds its last skin. Subjects, harboring the hook-worm, void feces laden with ova of the parasite. In localities, most severely affected, the privies are commonly so exposed that swine or fowl scatter the ova from the dejecta over the ground; even more frequently, outhouses are not employed, but the infected persons defecate in the barnyard, bushes, garden, fields or in the mines. The ova develop into larvæ in the moist ground, shallow water or the soil of a mine, and attack those running bare-foot, enter the hands or reach the mouth.

The male worm is 8 to 10 and the female 10 to 18 mm. in length; the mouth has hooks by which it adheres to the mucosa of the duodenum. The ova measure 30 to 60 micromillimeters; they are oval, are covered with a translucent shell and differ from the flat oxyuris ova. They show 1 to 6 yolk segments and may number over 4,000,000. They develop into the larvæ.

Symptoms.—Some subjects resist the parasites they carry and present no symptoms. Despite their immunity, these "carriers" disseminate the disease. *Mechanisms* of the symptoms: (1) The parasites *suck blood;* Ernst and Treichtenstern describe dying worms ejecting blood, as a locomotive puffs smoke. (2) They also *bore into the intestinal mucosa* but destroy it and the submucosa which Looss pictures drawn into their esophagus and even into their intestines. (3) Their *bites become infected* (A. J. Smith). (4) It is also possible that they secrete a substance which degenerates the bloodvessels, hinders hemostasis or operates as a hemolytic. *Types:* (i) The symptoms may be only mild dyspepsia, slight anemia (hemoglobin 80 or more), dull headache, etc. (ii) Moderate intensity; with hemoglobin 60 to 80 per cent., epigastric pain, poor development in the case of children and some apathy. (iii) The severe type, with hemoglobin 30 to 60 per cent., great dyspnea and abdominal pain, vomiting, anasarca and other symptoms (*v. i.*). (iv) The severest form, with hemoglobin below 30 per cent., and all symptoms extreme.

The *ground itch* represents the atrium in most cases and a history of it is generally obtained. Around the toes, or less often on the hands or buttocks, an itching, very painful eruption develops, first of vesicles which loosen the skin and contain the larvæ, then of pustules, oozing and fissures. In a few days the larvæ reach the air passages (*v. s.*) and cause sore throat, bronchitis, cough and expectoration, which latter, being swallowed, allow the larvæ to reach the bowel.

Alimentary Symptoms.—The tongue exhibits ink-like patches. The appetite may be increased in cases of mild or moderate intensity but is lacking in the severest types; it is often perverted and resin-chewing, eating of hair, earth (geophagy), clay or pebbles, inordinate or early chewing of tobacco, etc., are frequently observed. Ashford and King find that epigastric pain or uneasiness is the most constant, suggestive and clear symptom. Nausea, vomiting, diarrhea alternating with constipation and colic are common. Occult or obvious blood in the stools is the rule. In the Panama case which the author saw in the County Hospital, there was great nausea, pain and exquisite abdominal tenderness; the stools contained blood, eosinophile cells, Charcot-

Leyden crystals and ova. Stiles suggests a ready test for blood, by which parts of the stool are placed upon blotting paper; after an hour or so a rusty stain develops about the stool. Children become pot-bellied. In the subjects coming to autopsy, the feces show Charcot's crystals, eosinophiles, much mucus, ova, worms and blood. Reddish areas may be seen through the unopened gut. The bowel may appear red or chocolate color; Peyer's patches and the solitary follicles are often swollen; ecchymoses, submucous cysts and erosions are common; and moist red areas are noted, either elevated or bitten by the worms, and eosinophilic infiltration of the mucosa, submucosa and even the muscularis. Most of the parasites lodge in the jejunum but also in the duodenum and upper ileum. Before the death of their host, many leave the body.

Blood Findings.—An anemia develops which is first of the secondary type and later resembles pernicious anemia; it is due to loss of blood and possibly some hemolysin from the parasite. The hemoglobin averages 67 per cent. (Dock and Bass) or 42 per cent. (Ashford and King); it is more reduced than the red cells (low color index). The red cells range between $2\frac{1}{2}$ and $3\frac{1}{2}$ millions; there are polychromatophilia, normoblasts and microcytes. The white cells are usually not increased; in advanced cases there is leukopenia; the eosinophiles average 15 to 25 per cent., but may run higher (30 to 72 per cent.). The skin is sallow, pasty, even lemon-color or waxy, and finally edematous. The bone-marrow is pale—perhaps lymphoid, gelatinous or fatty; nucleated cells indicate blood regeneration.

Many, perhaps most, of the other findings are anemic—the extreme dyspnea, rapid pulse (over 100), pulsating vessels, edema of the skin, serous sacs and brain; dulness, somnolence, headache, inertia (laziness), weakness and headache are common and in severer infections the vertigo is extreme; pains develop in the joints and sternum; marked paresthesia, abolition of the patellars and weakness develop, to the simulation of paralysis; the pupils dilate and the eyes present a fishy look; there is retinal clouding, anemia and hemorrhage; and extreme apathy, anxiety or melancholia intervenes. Cardiac hypertrophy and dilatation and arteriosclerosis are frequent. The urine is copious, of low specific gravity, either free of albumin or highly albuminous and contains no casts. The heart, liver and kidneys are fatty, degenerated and enlarged; the spleen is not usually enlarged (save in Egyptian autopsies). The temperature is subnormal, sometimes with irregular runs of fever. Malnutrition and maldevelopment mark the pronounced types. Children grow poorly and reach puberty late and incompletely. Men of twenty may appear ten years old. Some are dwarfs. The pubic hair is scant, the beard comes slowly, the mammæ are small, and in males, impotence, and in women, amenorrhea are common.

Diagnosis.—The diagnosis is suggested by the anemia, dyspnea, dilated heart, epigastric pain, maldevelopment and history of ground itch, but is made certain only by examination of the stools for ova or worms.

Prognosis.—The outlook depends on the stage of the malady. Uncinariasis greatly reduces the resistance to other diseases. The eosinophilia

measures the degree of resisting power. The mortality is 10 to 30 per cent. Much depends on early therapy.

Treatment.—1. PREVENTION.—Drinking water should be boiled and the hands should be cleansed before eating. The stools should be received in water-tight closets and disinfected; careless defecation in mines has caused wide dissemination of the disease. Mines should be disinfected with the milk of lime. Prevention means (i) the extermination of the mature worms in man, particularly in "carriers;" (ii) preventing the growth of ova into larvæ; and (iii) guarding against infection by already developed larvæ.

2. MEDICATION.—*Thymol* is the most efficacious remedy. After a preliminary purge of sodium sulphate, the patient is kept abed and as the peristalsis decreases, gr. xx of thymol are given in capsules and repeated twice at three-hour intervals. The thymol is first well triturated with an equal amount of sugar of milk to prevent its cohering and followed by a saline purge two hours afterward. The first treatment evicts 80 to 90 per cent. of the parasites, 5 courses cure 95 per cent., and persisted in, "it always succeeds." As toxic symptoms (nervous excitation, smoky urine, dizziness, fainting, abortion or death; 11 cases in America) may develop, no solvents of thymol should be given, as alcohol, chloroform, ether or castor oil. This treatment should be repeated once a week, as long as ova or Charcot-Leyden crystals appear in the stools. Very edematous patients do not tolerate thymol. *Male fern* is considered the best remedy by Tenholt and other Westphalian writers.

V. Filaria.—Demarquai (1863) first found the embryos of the filaria; Wücherer (1866) found them in the urine and Lewis (1872) in the blood.

1. **Filaria Nocturna.**—(Bancrofti, Cobold, 1877). Its distribution is largely tropical and subtropical. It occurs in 30 per cent. of the inhabitants of the Samoan and Friendly Islands. In the United States it has been found in the South, New York, Illinois and Pennsylvania. (*a*) *Infection of man* occurs by mosquito bites or possibly by water, contaminated with mosquito eggs. In the mosquito, the embryos change to the young adult form. (*b*) The *next stage* is the presence of *adult filaria in the lymph channels,* where they cause lymph stasis and lymphangitis. The female filaria measures 155 x 0.7 mm. and the male 83 x 0.4 mm. (*c*) The *embryos enter the blood current* from their birth place, the lymph channels; they are very numerous, measure $\frac{1}{8}$ inch in length and the diameter of a red blood cell. Under a low-power lens their active movements are easily seen. They appear in the blood only at night, usually near midnight, and during the day retire to internal organs, as the lungs, but if the patient sleeps by day, they appear in the daytime only. (For Stäubli's method of detection see page 310.)

Symptoms are absent in the majority of cases of human and animal infection and are due to the parent worm, not to the embryos. (*a*) *Chyluria* or hematochyluria is due to plugging by the worm or its ova of the lymphatic vessels in the peritoneum, pelvis, vesical and perirenal tissues. Lymph stasis, ectasia and lymphangitis result, with intermittent rupture into some part of the urinary tract, and the passage of milky, chylous urine, containing molecular albumin and fat. The amount of

the urine varies; it may be normal or increased. There is usually also some blood. If the blood clots, vesical disturbance results. The embryos are often found in the blood and sometimes in the urine; there is also a non-filarial chyluria. The affection may last for years without impairment of health. The very rare rupture into the peritoneum causes chylous ascites. (*b*) The *lymph scrotum* is due to lymphatic obstruction by the adult worm. The scrotum may weigh 50 to 200 pounds. Rupture into the tunica vaginalis causes chylocele. The labia may be similarly involved. The embryos are often missing from the blood, being apparently confined to the local involvement by the lymphangitic thickening, whence the difficulty in stating the relative percentage of parasitic and non-parasitic cases of lymph scrotum and elephantiasis. (*c*) *Elephantiasis* usually develops in the legs, scrotum and labia, less often in the arms, breasts or trunk. In forms (*b*) and (*c*) there occur intermittent exacerbations with fever and nervous disturbance.

2. **Other Forms of Filaria.**—The exact status of the *Filaria diurna* (Manson, 1891) is not known; *F. perstans* (Manson, 1891), which causes pustular eruptions (craw-craw); *F. loa* (the adult stage of Filaria perstans) which inhabits the conjunctiva; *F. labialis;* and *F. oris* and *bronchialis* are other forms.

Treatment.—(*a*) For *prevention,* mosquitoes and infected water should be avoided. (*b*) No parasiticide is known. (*c*) *Chyluria* is treated by cinchonization, salvarsan and *x*-rays. (*d*) *Lymph scrotum* and *elephantiasis* may necessitate castration or amputation.

VI. Dracunculus (Filaria) Medinensis.—This is found on the West African coast, the Gold Coast, in Abyssinia and South Egypt; on the Caspian Sea and Persian Gulf; in the East Indies, parts of India and South America. Guinea-worm disease (drachontiasis) developed in persons who had always lived in Philadelphia and Fortress Monroe. Man is infected by a small water flea (cyclops) which contaminates the water with the larvæ (embryos). These embryos, measuring 0.6 mm., enter the stomach, bowel and probably the mesentery, where they copulate; the male, of which little is known, is either discharged from the bowel or dies in the mesentery. The female is cylindrical in form, measures 50 to 80 x 2 mm., has a triangular mouth, eight papillæ and a blunt tail; its uterus occupies almost its entire length. The female, which is usually solitary, wanders downward in the subcutaneous and intermuscular tissues to the thighs, legs and in 66 per cent. of cases reaches the feet where, near its head, vesicles, abscesses and ulcers develop. Its downward course occupies a year, during which it may be felt subcutaneously. When the skin breaks the parasite discharges her embryos and then leaves the body, to die.

Treatment.—Treatment by excision, or by 1 to 1000 bichloride injections, seems most rational. If the worm is ruptured in efforts to extract it, high temperature, alarming nervous symptoms and sepsis result from migration of the embryos liberated in the tissues.

VII. The Trichocephalus Dispar or Whip-worm.—This nematode is found most often in southern Italy and France and but seldom in this country; one case occurred in the Cook County Hospital. It is also

called trichuris trichura. It requires no intermediate host, enters with the food and drink and is found in the cecum and large gut. The male is slightly shorter than the female, which is 4 or 5 cm. in length. The anterior portion of the worm is thin; the posterior two-fifths is thick, in the male round and in the female pointed. It bores into the mucosa and is nourished by the blood and not by the alimentary contents. The eggs measure 0.05 mm., are oval and are marked by a button-like projection. It causes few symptoms; occasionally there is severe abdominal pain. Three fatal cases are recorded. Male fern and thymol internally and benzene ʒj to Oij of water as an enema are recommended.

VIII. The Strongyloides Intestinalis.—This includes a number of nematodes, as the Anguillula intestinalis or stercoralis, rhabdonema, etc. They are found in the endemic diarrhea which occurs in Cochin-China. Strong reports cases from Manila and W. F. Thayer reported 3 cases in America. A few parasites cause no symptoms. Thymol is almost specific.

Enstrongylus Gigas.—The Enstrongylus gigas which occurs frequently in dogs and wolves, is very rare in man. It is a nematode whose ova appear in the urine. It causes pyelitis, hematuria, colic, tenesmus and sometimes chyluria.

DISEASES CAUSED BY TREMATODES (DISTOMIASIS).

1. *Bilharzia disease* (endemic hematuria) is due to the blood fluke (Schistosomum hematobium). Bilharz described the disease in 1851. It is an African and Japanese affection; it has prevailed for many centuries in Egypt, where Bilharz considered that half of the lower classes was affected. It is equally frequent in Uganda. Importation has occurred into India, the Mediterranean islands and America. Only 12 cases have occurred in the United States.

Children are more predisposed than adults to infection, which occurs through the drinking water or possibly through the skin. The embryos enter the stomach, penetrate the alimentary mucosa and mature in the veins of the pelvis, spleen, mesentery, liver, kidney and bladder. The male worm measures 4 to 15 x 0.6 mm., the female 15 to 20 x 0.3 mm. The male is rolled up like a leaf, forming a groove in which the female lies during copulation. Many ova are found in the urine; they are oval, carry a spine and contain a ciliated embryo. They die in the urine in a day but live for some time in water, where their life-history is still imperfectly understood.

Hematuria is the most common and often the only symptom; it may be macro- or microscopic and occurs chiefly at the end of urination; examination shows many ova and eosinophiles. Cystitis occurs in severe infection. Stones are not uncommon, particularly in Canton, China. Suppuration may develop fistulæ. These clinical symptoms result from massive accumulation of ova in the submucosa, which leads to papillomatous hyperplasia, hemorrhage, suppuration, necrosis, salt deposits (calculi) and rarely to malignant degeneration. Similar changes in the rectum cause pile-like papillomata, dysenteric evacuations and tenesmus.

As complications, pyelitis, prostatitis, urinary fistulæ and changes in the liver (thickening of the periportal connective tissue), uterus, vagina, seminal vesicles and renal pelvis, resembling those in the bladder, may develop. The eosinophiles are increased to 10 to 50 per cent.

Schistosomum Japonicum (Cattoi). This fluke is found in China, the Philippines and Japan. Its ova are smaller and have no spine. Living in the bloodvessels of the digestive tract, it induces a severe anemia, dysentery, ascites and cirrhosis of the liver and spleen. Epilepsy may develop. In the intestines, other flukes may be found—the Gastrodiscus (Amphistoma) hominis and Distoma (Fasciolopsis) Buskii, in India and the Mesogonimus heterophyes in Egypt and Japan.

Treatment.—Change of climate is advisable to avoid reinfection. The parasites usually die, though slowly, and as no local or systemic remedies modify their life-history, treatment must be expectant.

2. *Lung flukes* cause the *endemic hemoptysis,* observed by Bälz chiefly in North China, Corea, Japan, the Philippines and Formosa. The parasite is named Distoma pulmonale (Paragonimus westermani); it measures 10 x 6 mm. and is found largely in the lungs, but sometimes in the brain and skin. In young males dyspnea, anemia, lung induration and a chronic cough develop with bloody or prune-juice sputum, in which are found blood, dark brown ova measuring 0.08 to 0.1 x 0.05 mm., eosinophile cells and Charcot-Leyden crystals. The hemorrhages, which are sometimes profuse, result from small infarcts and cysts of ova in the subpleural lung tissue. Epilepsy and hemiplegia may result from brain embolism. The course is usually favorable, though chronic, and is not influenced by treatment.

3. *Liver flukes* (Fasciola hepatica, Opisthorchis sinensis and at least four other species) enter like the above forms through contaminated water. The fluke is found in the upper small intestine and the biliary or pancreatic duct. It measures 10 to 20 x 2 to 5 mm. and its ova 30 x 15μ. It is found in Japan and certain provinces show a mortality of 20 to 72 per cent. (Bälz and Inouye). White reported 18 cases in San Francisco, and Brayton and Darling cases in Panama. The liver is enlarged and tender; icterus results from biliary stasis, for the parasites lie in the walls of the biliary ducts and gall-bladder; diarrhea develops, and the dejections contain ova and blood; anemia, ascites, anasarca, adynamia and emaciation develop and death invariably results. There is no treatment beyond the symptomatic support of the heart. Prophylaxis: avoidance of drinking or of swimming in canal water, and thorough cooking of fish and mussels have stamped out the plague in one Japanese province (Inouye).

SECTION II.

DISEASES OF THE CIRCULATION.

DISEASES OF THE HEART MUSCLE.

INTRODUCTORY PHYSIOLOGY.

Although convention has sanctioned the classification of heart diseases into those of the myocardium, endocardium and pericardium, besides its nervous disturbances, nearly all cardiac disorders are referable—on the last analysis—to failure of the heart muscle. A review of the more salient affections of the heart relegates many of them to a secondary place. Thus, valvular diseases seldom attract attention, until the overstrained heart muscle becomes more or less "incompetent." In pericarditis, the fresh effusion presses on the heart and embarrasses its diastole; or, when older adhesions firmly anchor the organ, the heart's systole is impeded. The heart muscle and the vasomotor system sustain the brunt of the acute infections and chronic intoxications. Angina pectoris is a myocardial malady, arising from disease of the coronary vessels, which bathe the myocardium with fresh blood. Arteriosclerosis works its gravest injury on the cardiac musculature. The fatty heart, chronic myocarditis, the senile heart, heart-block, and many of the arrhythmias or "neuroses" of the heart, are but instances of failing competency in some segment of this hollow contractile muscle.

Until recently it was maintained that the heart mechanism was regulated by nervous influences (the neurogenic theory), but now the source of cardiac action is held to reside in the muscle itself (the myogenic theory—which is possibly overstrained in our swing from one extreme to another). Gaskell enumerates the functions of the myocardium as five: "The muscular fibers of the heart are capable of rhythmically creating a stimulus (*rhythmicity*), of being able to receive a stimulus (*excitability*), of responding to a stimulus by contracting (*contractility*), of conveying the stimulus from muscle fiber to muscle fiber (*conductivity*), and of maintaining a certain ill-defined condition called tone (*tonicity*)."

The *normal* (or sinus) *rhythm* of the heart consists of the successive contraction of the sinus, auricle and ventricle. The *stimulus* to contraction arises in the remains of the sinus venosus (the sino-auricular node of Keith and Flack) at the mouth of the superior vena cava; it is called the pace-maker of the heart. In the wall of the right auricle near the

coronary sinus is Tawara's auriculoventricular node, the origin of the auriculoventricular bundle of Kent and His, by which impulses are *conducted* from the auricle to the ventricle. (In pathological rhythm, stimuli may arise from other points than the sino-auricular node and initiate extrasystoles.) *Contractility* is so regulated that each systole is forcible (to the full capacity of the heart's muscle); each beat is followed by a period of rest—a refractory period in which contraction cannot be elicited. *Tonicity* holds the heart in "tone," keeps its volume less than it would be if the myocardium completely relaxed, and prevents dilatation. Tone is increased, *e. g.*, by digitalis, and lessened by severe toxemias and cardiac overstrain. The vagus inhibits or decreases the rate, conductivity, irritability and contractility and the sympathetic increases the rate, etc. Throughout this section, repeated reference to these functions will be made, as the myocardium is the key to cardiac conditions.

In the balanced circulation, besides the heart and its nerves, the bloodvessels are important; without their elasticity and contractility and their vasomotor control, the circulation could not be maintained; and in vasoparalysis, we see the circulatory failure of certain severe toxemias. Respiration aids in charging the heart with blood and the coronary flow is directly stimulating. We are just gaining, also, a small conception of the part played by the organs of internal secretion, *e. g.*, the chemical action of the secretion from the suprarenal glands and the pituitary body.

DILATATION OF THE HEART.

Definition.—Dilatation means *distention and diminished tonicity* of the chambers of the heart. Tonicity is that function of the heart which sustains a slight degree of contraction even during the diastole. Hypertrophy is thickening of the walls of one or more chambers. The two conditions are separate in themselves, though frequently combined; dilatation is the incentive to hypertrophy. Lancisi (1706–1728) described hypertrophy and dilatation.

Etiology.—1. Mechanical Causes.—The normal heart may possibly yield to increased blood-pressure or a weak heart to normal pressure. Rarely, if ever, is dilatation an independent condition. (*a*) *Valvular disease, e. g.*, dilatation of the left ventricle in aortic insufficiency, or of the left auricle and right ventricle in mitral stenosis. Hypertrophy is inversely proportional to the dilatation. In combined aortic and mitral insufficiency, the heart is enormous—the ox heart, *cor bovinum, bucardia.* (Two types of dilatation are recognized: the accommodative, in which it is compensatory and the dilatation of stasis). (*b*) *Causes raising arterial blood-pressure,* as stenosis of the aorta, arteriosclerosis and nephritis produce hypertrophy and dilatation of the left ventricle. Workers in caissons may suffer from dilatation. (*c*) *Causes raising blood tension in the lesser circulation,* as spinal deformity, chronic disease of the lungs and pleura, which produce hypertrophy of the right ventricle; of these emphysema is the most important; abdominal lesions are thought to reflexly raise the pulmonary blood-pressure. The lung's

vascular area must be reduced to 25 per cent. in order to affect the right heart. When there is pulmonary obstruction the heart stands pneumonia, bronchitis and other infections poorly. The size of the heart varies much physiologically; during severe exercise, the systole is incomplete and right-heart dilatation—even tricuspid regurgitation and marked venous stasis—may follow. Physical training increases the power and reserve strength of the right heart, the "wind" and endurance. An equal effort in an individual out of training, or with poor nutrition, "breaks his wind" and produces cardiac exhaustion. Air embolism also produces dilatation.

2. NUTRITIVE DISTURBANCES.—Dilatation may be induced (*a*) by acute infections, leading to cardiac degeneration or acute myocarditis; (*b*) by anemia or loss of fluids; (*c*) by chronic myocarditis, fatty heart or coronary atheroma, possibly following sudden heart strain; by endo- or pericarditis invading the heart muscle by contiguity, pericardial adhesions, etc.; (*d*) by poisons, alcohol or tobacco.

Pathology.—Dilatation occurs (*a*) with thinning of the walls, *simple dilatation,* or (*b*) with eccentric hypertrophy (*q. v.*). It is most common in the thin-walled auricles and less resistant right ventricle. The muscle is lax and the heart, when held inverted by the basal vessels, may in extreme instances fall flabbily like a cap over the hand; the endo- and pericardium may almost touch.

The papillary muscles and trabeculæ are flattened (pressure atrophy) and frequently thickened (pressure sclerosis). Microscopically the heart muscle may be normal, or show fatty or cloudy degeneration or myocarditis. There is much blood in the heart's chambers, cavæ and other veins. In dilatation occurring during the death agony from acute lung disease, suffocation, etc., the right heart is chiefly affected and on removal of the postmortem clots the chambers seem nearly of normal size.

Physical Signs.—1. DILATATION OF THE LEFT VENTRICLE.—(*a*) *On inspection,* the *apex beat* is dislocated downward, to the left and is diffuse, undulating and weak, if not absent. (*b*) *On palpation,* the apex may not be felt even though visible; in other cases the apex and heart shock are snappy and feel strong to the inexperienced hand, yet contrast sharply with the weak, rapid and often irregular radial pulse. (A palpable impulse may be due to active diastole of the auricle, to passive distention of the ventricle by blood or to closure of the auriculoventricular valves.) Albertini (1761) distinguished hypertrophy and dilatation by palpation. (*c*) *On auscultation,* the *heart tones* are weak, though sometimes snappy. The first apex tone is more valvular. The second aortic sound is weak because of poor arterial tension. In insufficient hearts, the heart tones sound alike, resembling the fetal heart tones (embryocardia). An apical *systolic murmur* is sometimes due to relative insufficiency of the mitral valve whose normal circumference of three and one-half inches may be stretched to five. Decreased tonicity accounts for the dilatation and murmur also; these signs may be relieved by rest and digitalis. *Gallop-rhythm* consists of three tones like the rhythm of the hoofs of a galloping horse; according as the extra tone falls at the end, beginning or middle

of the diastole, we distinguish presystolic, protodiastolic or mesodiastolic gallop-rhythm. The presystolic type is heard in strongly beating hearts, being due to strong auricular contraction and is likewise heard in weak hearts, being due to the ventricle beating later after the auricle than usual, from delayed conductivity. The other two types originate in an extra tone developed in the ventricles, probably based on decreased tonicity. The protodiastolic (third) sound is heard in the majority of normal young individuals. Gallop-rhythm is heard in the failing hypertrophied hearts of nephritics chiefly, in valvular lesions, atheroma, emphysema and in acute diseases; it is a sign of great danger. (*d*) *On percussion*, the heart extends to the left and downward, giving a more oval area of dulness.

2. Dilatation of the Right Ventricle.—This is more common. (*a*) On *inspection*, a diffuse beat is seen to the right of the sternum or in the epigastrium; and (*b*) it may be *palpated*. (*c*) *Auscultation* shows weak heart tones, and (*d*) *percussion* outlines dulness to the right of the sternum. Extreme dilatation increases the tricuspid orifice from four and one-half to five or six and one-half inches, causing relative tricuspid insufficiency.

3. Dilatation of the Auricles.—Because of the deep location of the left auricle physical signs of dilatation are seldom obtained. The changes usually ascribed to the left auricle are due to enlargement of the conus of the right ventricle. The distended right auricle is recognized by undulation to the right of the sternum, above the right ventricle, by dulness and sometimes by a presystolic wave.

Dilatation of all the chambers gives the heart a trapezoid conformation. The symptoms and physical findings of dilatation may develop gradually or abruptly after violent exercise, especially when the heart's nutrition is altered.

Symptoms.—Subjectively, the consequences of dilatation are loss of reserve power of the heart (see Symptoms of Valvular Disease). Cardiac insufficiency is evidenced by breathlessness or dyspnea, by cyanosis, cough, syncopal attacks, precordial anxiety, and, when the systole ceases to expel completely the blood from the dilated chambers, the liver enlarges and becomes tender, the lungs reveal crepitant rales along their bases, the legs swell and the urine, the surest gauge of cardiac competency, becomes less abundant.

Diagnosis.—The diagnosis depends on the etiological factors, the *weak tones*, the *increased dulness* and evidences of *cardiac failure*. The *absolute heart dulness* in middle life is bounded by the left border of the sternum, the fourth or fifth rib and the left parasternal line. Percussion should be made lightly, using the finger-to-finger method and applying the passive finger firmly against the chest wall; observance of this technique gives as accurate results as does the fluoroscope. According to Moritz, the normal heart measures 3.5 to 4.5 cm. to the right of a midsternal point and 7.5 to 8.5 cm. to the left. The *relative dulness* reaches from the third or fourth rib above to the apex beat and to a line drawn obliquely from the third left costal cartilage across the sternum to the fifth right costal articulation. Percussion is difficult in distorted, fatty

or edematous chests. Various functional tests[1] are suggested, yet none is satisfactory except the test where a given effort causes it to decrease in size, stimulation inducing tonicity, or to increase in size, decreased tonicity on overstraining. Differentiation concerns the same conditions discussed under *effusive pericarditis* (*q. v.*). Rest and digitalis benefit dilatation, but not pericardial effusion. *Consolidation of the adjacent lung* margin can be recognized by the altered breathing and rales. *Mediastinal tumor* and *aneurysm* are suggested by pressure symptoms. If the margins of the *lung are adherent* from pleural adhesions or *emphysema* obscures the outlines of the heart, dilatation is diagnosticated by the relative, more than by the absolute, dulness. Palpatory percussion, very lightly made, is most valuable and the *feel* of the increased dulness has greater diagnostic value than the sound elicited. *Encapsulated pleurisy* is differentiated by careful puncture, if other signs fail. The fluoroscope is frequently of aid. In short chests and in 12 per cent. of women, the apex is seen normally at or even beyond the nipple. In *spinal curvature* the topographical relations are disturbed by visceral luxation, and the irregularity of the sternum and ribs makes percussion uncertain. In *fat individuals*, dulness may result from adipose deposit in the mediastinum. Dilatation must not be mistaken for dislocation due to distention of the abdomen.

Prognosis.—The prognosis largely depends on the etiology.

Treatment.—(*a*) The cause must be treated. (*b*) The *dietetic* and *special indications* of *valvular disease* (*q. v.*) must be carried out. (*c*) *Oertel's treatment* consists of (i) *limitation of fluid ingested*, and stimulation of its excretion by sweats and deep inspirations; beer must be interdicted. Many nervous subjects tolerate limitation of water very poorly. (ii) *Reduction of the body weight* (see OBESITY). (iii) *Stimulation of the circulation by exercise.* Mountain climbing is said to stimulate the heart, develop the respiratory muscles, increase the lung capacity and widen the vessels. Reasonable exercise may prove beneficial, but rest in bed gives better results; exercise is injurious when the heart muscle or coronaries are diseased, or when endocarditis, pericardial adhesions or Bright's disease exists. (*d*) The *Schott method* consists of (i) *baths* in springs containing sodium and calcium chloride, iron carbonate and carbon dioxide. The temperature should be a few degrees below body heat, and they should be given once daily for five to twenty minutes, for two or three days, and then intermitted. They may be given artificially, using 40 gallons of water, with 3 to 10 pounds of salt,

[1] In Schapiro's test, the normal decline of 7 to 15 beats in the heart's rate, when the patient lies down, is absent in decompensation. *Gräupner's test:* Normally the pulse and blood-pressure rise after exertion, such as running upstairs, and if the effort is prolonged and arduous, the figures fall to normal or below it, but the pulse rises and falls before the blood-pressure begins to rise and fall; in weak hearts the blood-pressure rise is delayed, decreased or absent. In *Hertz's test*, the patient sits and very slowly flexes and extends the right forearm, directing all his attention on the act but avoiding any firm contraction of the muscles; the physician supports the patient's elbow with his left hand and directs the movement with his right, but without aiding the movement; there is no influence on the pulse in normal conditions, while in pathological conditions, it is slowed 5 to 20 beats. *Marey's* and *Levy's test* consists of digital compression of both femoral arteries; in normal conditions, within three minutes the blood-pressure rises 5 to 15 mm. Hg.; in hypertrophy, even more and in weak hearts, no rise (this test is not valid in neurotics).

10 ounces of pure calcium chloride and carbon dioxide generated in a vessel from sodium bicarbonate and hydrochloric acid. The temperature is then reduced to 90°, and the amount of carbon dioxide increased. The bloodvessels are contracted and later dilated. The pulse becomes slower and fuller, dilatation decreases, stasis is lessened, diuresis is increased and the metabolism of the muscles and heart is improved. (ii) *Movements* by the patient, *resisted* by the operator, dilate the vessels. Active exercise constricts the vessels. The Nauheim treatment has been unduly exploited.

Each exercise is made against slight resistance applied by the physician.

1. The arms are extended in front of the body at the level of the shoulder, with the palms touching. The arms are then moved slowly outward till they are in a line with each other; they are then brought back to their original position.
2. The arm and hand hanging down with the palm turned forward, the forearm is flexed upon the arm (which is kept still) until the fingers touch the shoulder. The forearm is then extended to its original position.
3. The arms, hanging down as in No. 2, are raised outward until the thumbs meet over the head; they are then returned to their original position.
4. With arms dependent, the fingers, at the first phalangeal joints, are pressed together, and the arms are then raised until the hands are above the head, after which they are brought back to their original position.
5. The arms, hanging in the position of "attention," are advanced forward parallel to each other until they are elevated to a vertical position; they are then brought back to where they were before.
6. Same as No. 1, but with fists clenched.
7. Same as No. 2, but with fists firmly clenched.
8. The arms, starting from the position of "attention," describe a circle by moving forward and upward until they are raised vertically; then each palm is turned outward, and the arms descend backward to their former position.
9. The body is bent forward, and then brought back to the erect position.
10. The body is rotated, without any movement of the feet, first to the right and then to the left, and then back to its original position.
11. The body is flexed laterally, as far as possible, first to the one side and then to the other, and afterward restored to its original erect position.
12. The patient, standing with the feet side by side and supporting himself by leaning with one hand upon any object, flexes the opposite thigh as far as it is possible, and afterward extends it until the feet are again side by side; then leaning on the other hand, he carries out a similar movement with the other thigh.
13. The patient, supporting himself by one hand, as in 12, and the knee being kept straight, each leg in turn is raised as high as possible in front of the body, and then in the same way behind.
14. Supporting himself by placing both hands in front on the back of a chair, the patient first flexes one leg and then the other upon the thigh as far as he can.
15. Each leg in turn is abducted as far as possible, the knees being kept straight, the patient resting on one or other hand the while.
16. The arms, held horizontally outward, are rotated forward and backward at the shoulder-joint.

Contra-indications are aneurysm, angina pectoris, acute cardiac insufficiency and tendency to hemorrhage or embolism.

HYPERTROPHY OF THE HEART.

Etiology.—*Hypertrophy* of the *left ventricle* results from (*a*) *valvular lesions* wherein dilatation precedes and incites hypertrophy; (*b*) *renal disease;* usually from a chronic interstitial nephritis (mechanical obstruction to the flow of blood or retention within the blood of waste substances which excite arterial spasm); (*c*) *arteriosclerosis* (*q. v.*); (*d*) *congenital*

narrowness (*hypoplasia*) *of the arterial system,* or of the *aorta at the isthmus;* (*e*) *dilatation of the aorta,* diffuse or aneurysmatic; (*f*) *pericardial adhesions,* (*q. v.*); (*g*) *myocarditis* (*q. v.*); (*h*) *pregnancy;* the placental circulation or increased intra-abdominal pressure increases the heart's work. The high diaphragm also apposes the heart to the chest wall and tips the apex outward so as to simulate hypertrophy; (*i*) *overexertion;* the "irritable heart of soldiers" is caused by forced marches, cold and mental excitement; the so-called *idiopathic* hypertrophy occurs in hard workers, and especially in Munich from excessive use of beer and from the resulting high blood-pressure; hypertrophy results from continued hard work, while dilatation follows sudden cardiac strain; (*j*) *nervous or toxic causes,* as exophthalmic goitre and tobacco, especially strong Havana cigars, which cause rapid irregular heart action; (*k*) *reflex* hypertrophy is said to result from abdominal growths, brachial neuritis, etc.

Hypertrophy of the right ventricle arises from (*a*) *left-heart disease,* from the intimate connection between the muscular fibers of both sides; (*b*) *right-heart valvular lesions* and *congenital heart disease;* (*c*) *increased pulmonary pressure* from disease of the pulmonary artery or pressure upon it; (*d*) *emphysema, chronic bronchitis, spinal deformity,* pulmonary induration, extensive *pleural adhesions,* etc.

Hypertrophy of the auricles always coexists with dilatation.

Pathology.—The muscle fibers increase even tenfold in size (*hypertrophy*) and increase in number (*hyperplasia*). Hypertrophy may exist alone—*simple hypertrophy,* but more frequently with dilatation—*eccentric hypertrophy. Concentric hypertrophy* is rarer and some regard it as a postmortem condition; the ventricle may be firmly contracted in death from violence, cholera or hemorrhage, but it can be distended by the finger. The size of the normal heart is that of the fist (Laennec), and the normal weight is 300 gm. The heart is sometimes increased in weight, even 1980 gm.—the *cor bovinum.* The left ventricle, normally 9 to 10 mm. in thickness, may be more than doubled in hypertrophy; the right ventricle measures 3 or 4 mm.; the left auricle measures 3 and the right 2 mm. In the fetus the ventricles are equally thick. Up to the eighth year, the left ventricle is relatively thicker than in adults, because of narrowing of the aorta at its isthmus.

Symptoms.—Hypertrophy of the left heart may be symptomless, or there may be palpitation, cardiac oppression or cerebral congestion—tinnitus, headache or vertigo. There is frequently a tendency to hemorrhage—epistaxis, cerebral hemorrhage, excessive menstruation. Polyuria results from high arterial tension. The causal nephritis, valvular disease, arteriosclerosis or emphysema presents its own symptoms. Right-heart hypertrophy is attended by brown induration of the lungs, hemoptysis, cyanosis or bronchial catarrh.

Physical Signs.—Much confusion in the clinical description prevails because the signs of hypertrophy are very often confounded with those of dilatation, with which it frequently coexists.

Hypertrophy of the Left Ventricle.—1. *Inspection.*—This discloses (*a*) a *strong,* sometimes heaving, diffuse *apex;* its dislocation

indicates dilatation, because thickening of the heart muscle alone gives no appreciable increase in the size of the heart. Rotatory retraction of the apex beat is sometimes seen. (*b*) *Precordial prominence;* (*c*) *forcible pulsation* of the *vessels.*

2. *Palpation.*—Palpation confirms (*a*) the strong *apex beat,* detects (*b*) the *palpable second aortic tone* due to high tension or atheroma, and sometimes (*c*) a *systolic apical thrill* (transmission of the strong first tone). (*d*) The *pulse* is regular, full and hard in simple hypertrophy; in the eccentric form it is softer and more rapid; it is sometimes weak or almost absent in the arms, due to loss of vessel tonus. (*e*) *Thrills over the peripheral vessels* from high tension vibration may be felt.

3. *Percussion.*—Enlargement means dilatation or an atheromatous relaxed aorta which allows sinking of the heart.

4. *Auscultation.*—This brings out (*a*) the *loud first tone* over the apex (but not in aortic leakage); (*b*) the *accentuated,* even, metallic, *second aortic tone;* (*c*) *systolic arterial bruits* from tension and vibration of the *walls;* (*d*) the *gallop-rhythm* in failing hypertrophy, especially in nephritics; (*e*) a *peculiar* clink, sometimes heard to the right of the apex; (*f*) an *apical systolic murmur,* frequently of the cardiopulmonary type. Seitz and Gendrin recorded murmurs resembling the pericardial rub, due to violent muscular contraction.

Hypertrophy of the Right Ventricle.—(1) *Inspection* shows a *strong, systolic impulse under the lower part of the sternum* which is (2) *palpable,* as is the *strong second pulmonic tone.* (3) *Auscultation* brings out the accentuated second pulmonic; and (4) *percussion* is negative, unless there is coincident dilatation.

Diagnosis.—In *left-sided hypertrophy,* the cardinal signs are (*a*) the tense pulse, (*b*) the strong apex beat and (*c*) the *accentuated* second aortic tone; in *right-sided hypertrophy* they are (1) the strong second pulmonic tone and (2) substernal pulsation.

In nervous overaction of the heart, the apex is not heaving. Retraction of the left lung exposes more of the left ventricle, but the heart's outline enlarges upward and to the left. In narrow chests with poor lungs, the heart's dulness is wide, but hypertrophy is lacking. At puberty the heart is nervously unstable, the chest is thin and the heart normally wider. Abundant casts and blood in the urine usually indicate *nephritis,* but, if there are other evidences of stasis, such as congested liver, they are compatible, in the minds of some clinicians, with stasis alone. Nephritis may cause hypertrophy; nephritis and hypertrophy may be due to a common cause; or a failing heart may produce renal stasis. (See Nephritis, Differential Table.)

Prognosis.—The immediate outlook is good because hypertrophy is usually a compensatory process; the ultimate outlook is bad, as the reserve power is exhausted. Symptoms appear gradually or suddenly from intercurrent fevers, malnutrition and mental or physical strain. Hypertrophy caused by tobacco or athletics may regress. Gallop-rhythm is ominous, as is the occurrence of the first and second apical tones close together.

Treatment.—The therapy is eminently causal and dietetic; alcohol, tea, coffee, tobacco, cold baths and constipation are to be avoided. Treatment of decompensation is that of valvular disease (*q. v.*).

ATROPHY OF THE HEART.

Atrophy of the heart is chiefly of pathological interest.

Etiology.—The heart is at times congenitally weak and small. It may then be associated with small vessels, chlorosis (Virchow) or with hypoplasia of the genitalia (Rokitansky). Pressure atrophy, coronary sclerosis, pericarditic adhesions, the senile involution and cachectic diseases are the most frequent causes. *Anatomically*, the heart is reduced in size and weight (even to $1\frac{1}{2}$ ounces), the subpericardial fat disappears, the pericardium wrinkles, the coronary arteries are tortuous and the heart muscle is pigmented. The valves, especially the aortic, may waste. The atrophy may be partial, as that of the left ventricle in some cases of mitral stenosis.

The *symptoms* are indeterminate, and the *diagnosis* impossible.

FATTY HEART.

Etiology and Pathology.—Fatty heart is no distinct pathological or clinical entity. Two forms are recognized: (1) fatty infiltration and (2) fatty degeneration.

1. Fatty Infiltration.—*The Lipoma cordis capsulare, Virchow*, consists of invasion of the heart muscle by the subpericardial fat, normally found about the heart, in its furrows, at the tips of the ventricles and around the basal vessels. The fat extends in parallel lines between the muscle fibers, which it may wholly replace. The fatty infiltration was once considered primary, but it is secondary to muscle atrophy or some nutritive disturbance. The fat develops most in the outer layers of the myocardium, but may reach to the endocardium. Fatty infiltration is most common in obese males over forty years of age. It may occur after the menopause or in sterile women. High living, abuse of alcohol and heredity are factors. The heart is reduced 25 per cent. compared with the body weight.

2. Fatty Degeneration.—This form is caused by deficient oxygenation which is either (*a*) *general*, resulting from acute infections, old age, cachexia, anemia, poisons, etc.; or (*b*) *local or cardiac*, from pericarditis; coronary disease, which most frequently produces myofibrosis; and from cardiac failure following hypertrophy. Most degeneration occurs in the left heart. The fatty heart is flabby and yellowish-brown—the "faded leaf" color of Laennec; the splashes and streaks of fatty degeneration give the variegated "tiger" or "tabby-cat" appearance; oil globules in rows in the muscle fibers may replace them in part or entirely. Frequently the muscle fibers show albuminoid degeneration, the first step in fatty degeneration. The striations of the muscle disappear later, its consistence is decreased and its substance is friable.

Parenchymatous and fatty degeneration are observed in those seg-

ments of the heart where the greatest work and the most nutrition are demanded, as in the papillary muscles of the mitral valves and where pressure is greatest, viz., the septum and left conus arteriosus.

Symptoms and Signs.—Extensive changes may occur without clinical symptoms; Leube details a case of pernicious anemia without cardiac symptoms in which the apex was completely fatty. In some obese subjects, cardiac embarrassment develops with dyspnea, palpitation, etc. The autopsy may reveal fatty heart, but far more frequently myocarditis, hypertrophy or coronary disease is found; *i. e.*, there is no uniformity in the pathological findings, nor parallelism between them and the clinical signs. Though percussion of obese individuals is difficult, the heart is found dilated. In fatty heart, the tones are weak and distant, the apex beat is weak, diffuse or absent, and sometimes an apical systolic murmur is heard, due to irregular systolic vibration. Gallop-rhythm indicates cardiac fatigue. The pulse may be rapid, small and arrhythmic. Low temperature, sweating and bronchitis are common. Much stress was formerly laid on the *Adams-Stokes's syndrome* (see page 342).

Prognosis.—The prognosis is grave when coronary disease, angina, lung edema or an irregular or very slow pulse intervenes. Sudden death is not uncommon from acute dilatation, pulmonary embolism, cerebral hemorrhage, "heart block," or rarely heart rupture.

Treatment.—The treatment is that of obesity in fatty infiltration, but distinction must be made between (*a*) *obesity with anemia,* in which the subject is pale, with cold extremities, sweating, scanty urine, weak muscles and dyspnea; and (*b*) *obesity with plethora,* in which the color is red, the urine increased and uratic, the heart stronger, the muscles fairly developed and exercise possible with little dyspnea. Schott's treatment is beneficial in the first type, and Oertel's method in the latter. Saline cathartics, total abstinence from alcohol and the use of potassium iodide are indicated. Cardiac insufficiency is treated as in uncompensated valvular lesions and dilatation. Morphine should be used with great circumspection.

ACUTE MYOCARDITIS.

Etiology and Pathology.—Acute myocarditis is of the greatest clinical and prognostic importance.

Acute parenchymatous myocarditis corresponds to granular and fatty degeneration (*v. i.*); the "mushroom" heart of Stokes, and the "softened heart" of Laennec and Louis.

Acute interstitial myocarditis has been most studied of late years, while formerly the degenerations attracted most attention. It was first discovered by Leyden in scarlatina; it is found in a large percentage of cases of diphtheria, typhoid, acute endocarditis and rheumatism. Round cells are found in the interstitial tissue, with or without muscular alteration, such as vacuolization, nuclear multiplication, pigmentary deposit, blood extravasation and waxy changes.

Nodes (*v.* page 284), inflammatory thickening of the intima in the cor-

onary radicles and perineuritis are also described. Probably inflammation is primary and degeneration secondary.

Symptoms and Signs.—During the progress of an acute infection the heart's strength decreases; its tones, especially the first mitral, become fainter. There is often a marked presystolic impulse; the less distinct apex is dislocated to the left and the pulse grows more weak and irregular. Vasomotor relaxation is a very important element in the heart failure. Gallop-rhythm and embryocardia develop, and there is usually a relative mitral leakage. Cardiac pain may be severe. These changes may develop during the typhoid, scarlatina or rheumatism; or occur two to ten weeks after the infection, as in diphtheria. Sudden death may occur during convalescence. Rheumatic myocarditis often leads to chronic fibrous myocarditis.

Diagnosis.—The etiology is important; acute myocarditis is distinguished with great difficulty from acute endocarditis (*q. v.*).

Treatment.—*Prophylaxis.*—The patient should have absolute rest well into convalescence, because sudden heart insufficiency or unexpected death may follow effort, difficult defecation or sexual intercourse. *Cardiants* are indicated as in valvular incompetency. (See therapy of TYPHOID, DIPHTHERIA and PNEUMONIA.)

I. Acute Circumscript Myocarditis.—This, the disease of gladiators (Galen), is a form of sepsis, and its embolic heart foci are usually multiple. It is usually caused by the pyogenic organisms. The issues are (*a*) resorption with fibrosis, (*b*) caseation or calcification and (*c*) rupture into the pericardium or into the heart cavity, thus producing "heart ulcers," embolism, heart aneurysm or abnormal communications between the chambers. Sudden valvular insufficiency is sometimes suggestive, though it is usually impossible to make a diagnosis. The treatment is that of the fundamental septicopyemia and the resulting cardiac insufficiency.

II. Fragmentation of the Heart Muscle.—There are two forms (*a*) segmentation or separation from each other of the heart fibers, and (*b*) fragmentation or rupture across the normal or degenerated muscle fibers. It also has been called segmentary myocarditis; it most frequently occurs in death from violence, but may have clinical significance in sudden or gradual heart weakness. (See MYOLYSIS, under DIPHTHERIA.)

CHRONIC MYOCARDITIS (CHRONIC FIBROUS OR INTERSTITIAL MYOCARDITIS, MYOFIBROSIS CORDIS).

Etiology and Pathology.—Chronic myocarditis is rarely a disease *sui generis*, but is a sequel of some retrogressive lesion—a secondary process. It compensates for destruction of heart tissue, is a measure of that loss and is more often a *fibrosis* than an inflammation. The longitudinal incision of the heart muscle discloses scar-like tissue often unseen in transverse sections. This tissue is seen most frequently in the lower two-thirds of the anterior wall of the left ventricle and the upper two-thirds of its posterior wall near the auricle, as glistening sunken areas under the pericardium or endocardium. It also occurs in the papillary

muscles or septum. The scars appear as streaks or stellate plaques, sometimes measuring three or four centimeters. The foci may be macro- or microscopic, circumscribed or general. *Gross changes in the heart* include:

1. Hypertrophy.—This is vicarious and is inversely proportional to the number of scars. The relation is twofold; (*a*) hypertrophy often compensates for moderate muscular destruction; or (*b*) fibrosis develops in tired, hypertrophied hearts.

2. Dilatation.—This follows extensive fibrosis and decreased tonicity. Fibrosis may neutralize dilatation.

3. Atrophy of the Myocardium.

4. Valvular Disease.—A relative mitral insufficiency may follow myocarditis in the mitral papillary muscles. Dittrich described a genuine heart-stenosis, a myocarditic contraction of the left conus arteriosus in the adult, or of the right conus in the fetus. Relative aortic insufficiency may follow dilatation and myocarditis of the left ventricle (personal observations).

5. Heart Aneurysm and Heart Rupture.—Partial *heart aneurysm*, a localized bulging of the heart wall, is seen largely near the apex (left ventricle, 68 per cent. of cases), where acute myomalacia cordis or chronic fibrosis occurs most frequently; it is seen more rarely at the base or septum which may bulge into the right heart; and most infrequently in the right heart. The aneurysm is usually small, but may equal the size of the heart; it is usually single (though four were found by Thurnam), and may communicate with the heart cavity by a neck-like constriction; its walls consist of connective tissue, which is rarely ossified, as in Corvisart's case. It may rupture or detach a valve. During twenty years ending in 1903, D. G. Hall found 112 cases recorded (26 had aneurysm in the valves, and 25 in the coronary vessels); Volker alone made a diagnosis. *Rupture* of the heart (*v.* page 332).

Pathogenesis.—The *most frequent cause is disease of the coronary arteries*, which are end arteries, and anastomose with each other only through their capillaries. The vessels of Thebesius, entering the muscle from the auricles and ventricles may carry on some collateral circulation when the coronary arteries are occluded. *Coronary narrowing* may be *gradual*, by obliterative endarteritis or by the occlusion of the vessel from an atheromatous plaque in the aorta; or it may be sudden and fatal by embolism or thrombosis in an already narrowed vessel; the most common location is in the left ventricle (anterior coronary artery which is relatively small) and in the septum. In acute coronary obstruction, the heart muscle becomes ischemic and opaque and the necrotic area swells (coagulation necrosis); a wedge-shaped anemic *infarct* is less common. The muscle fibers become fragmented, lose their nuclei and disappear; this process may lead to rupture of the heart or sudden death. Ziegler named it *myomalacia cordis*. If the subject lives, scar tissue develops—a *dystrophic sclerosis*. Huchard employs the terms, arteriosclerosis of heart and arterial cardiopathy, to emphasize the causal role of arterial disease. A less frequent cause is *actual chronic myocarditis* following *acute myocarditis* or *endarteritis* due to rheumatism, typhoid or diphtheria. Dehio holds that *myofibrosis is a result of dilata-*

tion, and occurs where the muscle is destroyed, as a necessary and protective process, conforming with Thoma's conception of arteriosclerosis (*q. v.*).

Symptoms.—(*a*) In the *latent type* there are no symptoms and sudden apoplectiform death occurs in apparently perfect health. (*b*) Most cases present chronic symptoms of *cardiac insufficiency*. There is venous congestion. The *pulmonary circulation suffers* especially, as is shown by cyanosis, large infarcts, hydrothorax, edema of the lungs, bronchitis and dyspnea. The cardiac symptoms are precordial oppression, palpitation, cardiac asthma or angina pectoris. Examination of the heart sometimes gives negative results, but *dilatation* and hypertrophy are usually present (cardiomegalia). The *heart tones*, at first often clear, become *faint and irregular*, so that in the resulting delirium cordis, the tones are disorderly and the phase of the murmur, due to relative muscular insufficiency or to arteriosclerotic dilatation, cannot be distinguished. The second pulmonic sound is accentuated or split and the second aortic tone is weak, because the arterial tension is low. The *pulse* is *usually irregular;* paroxysms of arrhythmia may indicate an extension of the sclerosis. Irregularity usually antedates cardiac insufficiency. A regular pulse may be present throughout, or a number of regular strong beats is followed by a wave of rapid, tumbling heart action. The pulse-rate, often slow at first, increases to 90 or 100, with ominous crises of tachycardia. In some instances the pulse is persistently slow and associated with syncope (*v. i.* BRADYCARDIA).

Edema is, as a rule, less common and intense than in valvular disease. Embolism may occur. Digestive disturbances are constant. Alimentary autotoxemia, from carelessness in diet, may cause paroxysmal cardiac symptoms. Potain and Barie hold that digestive disorders induce a reflex contraction of the lung vessels and thus lead to dyspnea. The urine is that of stasis in the later stages, but early in the disease polyuria may prevail from high blood-pressure.

Diagnosis.—There are five cardinal points: (*a*) In the majority of cases *arteriosclerosis* is most important; it is usually diffuse, but may exist in the heart and not in the radial arteries, and conversely. (*b*) The *heart is usually dilated* and hypertrophied. (*c*) *No valvular murmur exists* in uncomplicated cases. The systolic apical murmur of arteriosclerotic dilatation may be heard or the soft, variable bruit of relative mitral leakage. (*d*) *The pulse is fast and irregular.* A regular pulse does not absolutely exclude myocarditis. (*e*) *Dyspnea* and pulmonary stasis are suggestive.

Myofibrosis may be overshadowed by the more frank yet wholly identical fibrosis of the vessels, kidneys, liver and perhaps meninges or adrenals (polyvisceral scleroses), until one of the great trio of risks of diffuse degeneration intervenes—cerebral accidents, renal suppression or cardiac agony (angina with pain or angina with precipitate coronary death).

Differentiation is often so difficult that many clinicians limit the diagnosis to *muscular insufficiency;* fatty heart, hypertrophy and dilatation, or pericardial adhesions may be found at autopsy instead of the

anticipated myocarditis. Sometimes when myocarditis is suspected, the heart shows nothing microscopically or macroscopically (the causes of heart failure are often beyond detection by the microscope). A diagnosis is made by exclusion, and then is only a "probability diagnosis." In differentiation from the "heart of *renal disease*," long observation, the gallop-rhythm (more common in the *cor renale*) and uremic manifestations are helpful; myocarditis and renal disease are frequently associated. From *mitral stenosis:* both lesions cause irregularity and a weak radial pulse and apex beat; when the presystolic stenotic murmur is absent, the loud second pulmonic sound, the loud snapping apical tone and the wide right heart are most suggestive of mitral stenosis. "Cardiac apoplexy" may be mistaken for cerebral apoplexy or embolism; the autopsy in cases of sudden death often discloses coronary closure with myomalacia cordis or fibrous myocarditis.

Prognosis.—Since digitalis is often ineffectual and angina pectoris, heart rupture or coronary closure may occur, the outlook is most unpromising. Some patients, especially elderly subjects, may live a decade or more, until intercurrent pneumonia or another secondary affection causes their death. Early digestive symptoms are less favorable than early edema or congested liver.

Treatment.—The therapy is (*a*) that of the *causal factor*, arteriosclerosis (*q. v.*) being the most common cause. Potassium iodide should be given, freely diluted in milk or water—

℞—Potassii iodidi . . . . . ʒiiss
Syr. sarsaparillæ co. . . . . ʒiv
M. et S.—One teaspoonful in a glass of water after meals.

(*b*) *Dietetic*; stimulants should be used carefully and overexertion and excitement avoided. (*c*) *The Schott treatment* (*v. s.*). (*d*) *Cardiants.* Digitalis is beneficial in the early stages—less so in advanced cases, or when the pulse is slow. Strychnine is frequently valuable. For anginal seizures or hard vessels, nitroglycerin is given. Caffeine, camphor, and morphine should be administered for cardiac asthma. (See THERAPY OF VALVULAR DISEASE.)

RUPTURE OF THE HEART.

Etiology.—*Rupture through the altered heart muscle* occurs in myocarditis; heart aneurysm, in which it may even perforate the chest wall; myomalacia cordis; coronary aneurysm; fatty heart, abscess, gumma or neoplasm of the heart, ulcerative endocarditis, obstructive valvular disease and stenosis of the isthmus of the aorta. It occurs largely in men over sixty years of age. It may occur during sleep or may be caused by trauma, vomiting, great effort, excitement, delivery, coitus, or by the abuse of digitalis. Kroll collected 332 cases.

Pathology and Symptoms.—(*a*) *Rupture* of a valve induces sudden valvular insufficiency. (*b*) *Total rupture* is usually single, though five rents are recorded, and takes place in the left ventricle (80 per cent.) where the pathological factors come most into play. *Sudden death* may occur, or

death may result after a few hours to eleven to seventeen days. The symptoms are a sense of something giving way in the chest, great precordial pain, incoercible vomiting or diarrhea, irritation of the vagus from hemopericardium, collapse and death from pressure of the blood on the heart or venæ cavæ. Hemopericardium (*q. v.*) may be suspected because of greatly increased triangular dulness, faint heart tones and absent or weak apex beat within the left border of the dulness.

Diagnosis and Treatment.—The diagnosis is rarely made. It depends on the etiology, increased dulness, cardiac insufficiency and symptoms of internal hemorrhage. The outlook is bad and the treatment wholly symptomatic; absolute rest should be enforced.

ANGINA PECTORIS.

This affection is usually described under cardiac neuroses but it is a coronary cardiopathy and its effects relate to the myocardium. Angina pectoris (stenocardia, breast-pang) was first described by Heberden and then by Hunter. Edward Jenner first recognized the connection between angina pectoris and coronary disease. By far the best treatises are Osler's classic on *Angina Pectoris and Allied States*, and Neusser's *Ausgewählte Kapitel der klinischen Symptomatologie*, Heft 2.

Definition.—An arteriosclerotic cardiopathy, characterized by (*a*) paroxysms of excruciating cardiac pain, (*b*) mental anguish, a sense of dying and (*c*) frequently by sudden death.

Etiology and Pathology.—The chief and practically constant pathological finding is arteriosclerosis involving the heart muscle directly by coronary atheroma, or indirectly by plaques in the aorta at the coronary orifices. The aorta is often atheromatous without coronary involvement and coronary sclerosis is frequent without much change in the aorta. It is notable that atheroma is frequent and angina pectoris is rare. The etiology is that of arteriosclerosis (*q. v.*)—syphilis (aortitis), gout, alcohol, contracted kidneys, sometimes infections such as rheumatism or influenza. A history of its occurrence through several generations is sometimes elicited. It occurs chiefly in men (90 per cent.), usually after the fortieth year (although cases in the second decennium are reported) and in the well-to-do classes, professional men, hard workers and luxurious livers. Angina is far more common in private than in hospital practice.

Symptoms.—Mode of Onset.—(*a*) In some cases the *onset is gradual*, and is preceded by dyspnea, especially when dressing or undressing, lacing the shoes, emptying the bowels, sitting up or beginning to exercise; by short, deep respiration, precordial oppression, pulmonary edema, bronchial catarrh, strong beating in the second or third left interspace, and difficulty in moving the left arm. This "cardiac asthma" is followed by genuine angina; both are secondary to coronary disease. (*b*) In other cases *angina is the first important symptom* of cardiac disease. It is excited by muscular effort, as walking up hill; mental excitement, as anger or coitus; chilling or bathing; flatulent dyspepsia or overloading the stomach, which by way of the vagus, may induce the attack when

the patient is quiet or just going to sleep. (*c*) *Death* may be the first symptom.

Paroxysm.—The attack itself consists of (1) *sudden agonizing pain*, which is lancinating and feels like an iron hand squeezing the heart. The pain emanates from the cardiac plexus lying behind the aorta, and in the nerve branches from it to the coronary arteries, whence its reflection to other parts, mentioned later. During the seizure the *heart muscle is ischemic* from coronary obstruction or possibly from superinduced spasm of the vessel (Allan Burns, Potain). In man and in the horse, atheroma or thrombosis of the abdominal aorta or its branches to the legs, shuts off the blood necessary during exertion; with moderate exercise the collateral circulation is sufficient, but prolonged exertion produces tingling, cramps and transient paraplegia ("intermittent claudication"). The heart-pang is caused by an analogous cardiac ischemia when unusual stress is thrown on the diseased coronaries and the myocardium becomes exhausted (impaired contractility). The author observed thoracic and abdominal angina followed by claudication. The "unutterable" pain is usually *substernal* and *radiates* to the left shoulder, neck and arm (to the chin, ear, jaw, occiput, larynx, even to the gall-bladder and renal region, spine, stomach, left leg and testis). Pain *along the ulnar nerve* to the fingers may precede the angina, or perhaps is associated with motor weakness or ulnar atrophy (in primitive vertebrates, the nerves involved would have been over the heart). Hyperesthesia of the arm, chest or mammary regions is frequent, and may persist, due to reflex cord irritation. The pain produces vasomotor symptoms, such as great pallor, low temperature and profuse sweating. The patient may fall, perhaps in a fatal syncope, or more often may support himself for a few seconds or minutes until the paroxysm has passed.

(2) The *mental anguish*, the *angor animi* (Latham) is the second component of the seizure and consists of a sense of dying, with great anxiety, complete consciousness, immobile attitude with head and trunk extended, and perhaps inability to articulate.

(3) The *danger of sudden death* is the third element. Death may come without pain (syncope anginosa) in the first seizure; often in the second or third attack; years after the initial paroxysm; or after repeated attacks, maybe twenty a day (the *état angineuse*). Osler has called the anterior branch of the coronary artery the "artery of sudden death."

Other Signs and Symptoms.—The *heart is variable*, sometimes normal in rate and strength, and again tense or weak, slow or rapid or irregular. The left radial pulse may be smaller than the right. Dunin found the arterial pressure low in 45 per cent. and high in 20 per cent. of his cases. Delirium cordis, partial heart-block, gallop-rhythm, extrasystoles, pulsus alternans and shortening of the long pause are but signs of cardiac fatigue.

The second aortic tone is often loud and metallic; a rough or blowing systolic (and sometimes a diastolic) murmur is heard over the aorta, which is sometimes dilated; and the left ventricle may be dilated; the relative mitral leakage relieves the high blood-pressure inducing the angina and exhausting the left heart. These incidents are simply

arteriosclerosis and its sequences. *Constitutional disturbance* is very frequent, as loss of weight, color and endurance. *Dyspnea* is not a part of angina pectoris, but of heart insufficiency. The urine is often pale and abundant after the seizure. Dysphagia, singultus, air-swallowing or gastric symptoms are due to sympathetic, phrenic and pneumogastric participation.

Diagnosis.—When the attack is typical and severe, there is little difficulty in diagnosis. Mild or early attacks may be confused with cardiac asthma. *Angina involving the abdomen* (angina abdominis) or legs (angina cruris) is *easily mistaken* and may appear first or alternate with the more usual angina pectoris (see Arteriosclerosis). The classical picture, together with hardness of the arteries and atheroma of the aorta, makes the diagnosis practically certain. When the heart seems normal, severe precordial pain means angina.

Pseudo-angina (Latham and Walshe) is the most frequent cause of diagnostic error. Its varieties are (*a*) *nervous pseudo-angina.* (James Mackenzie regards all angina as of the genuine type; the anginal pain arising, *e. g.*, in worn-out women, is due to exhaustion of the heart as well as of the nervous system.) Huchard's widely quoted table is substantially as follows:

True Angina	*vs.*	Pseudo-angina.
Most common in middle-aged men.		Commonest in women, at any age.
Paroxysms induced by exertion, etc.; diurnal; few in number.		Spontaneous; often nocturnal and periodic; frequent.
Pain intense, of short duration, precordial, inarticulate; immobile attitude; sensation of cardiac compression.		Less severe, lasting hours; epigastric; agitation, activity; sensation of distention.
No nervous symptoms.		Neurasthenic or hysterical stigmata.
Vasomotor form rare.		Common.
Prognosis grave or fatal.		Never fatal.
Lesions: from coronary sclerosis.		From neuralgia.
Arterial medication.		Neuralgic medication successful.

(*b*) *Vasomotor angina* is attended by vasomotor spasm, coldness and numbness in the extremities, faintness and great pain in the heart (Nothnagel). It may be reflex in origin.

(*c*) *Toxic pseudo-angina* is due to coffee, etc., but especially tobacco. Vasomotor symptoms, amaurosis, dyspepsia, weak, rapid or irregular heart action, palpitation and anginal pain are common. Functional tobacco angina from coronary spasm heals when tobacco is withheld; but actual coronary disease, due to tobacco and other heart poisons, is not curable. (*d*) The crises of tabes and precordial pain due to brachial or vagus neuritis, gout, etc., may be difficult to diagnosticate.

Prognosis.—The prognosis must be given with great caution; one patient lived for forty years. Few recoveries are reported.

Treatment.—The treatment concerns (*a*) *the etiology*—arteriosclerosis. Gout and diabetes necessitate appropriate diet and therapy. Attention must be directed to the question of stimulants. The possibility of syphilis should always be considered; aside from syphilis, the iodides are valuable, given two-thirds of the time, for months and years. Small amounts are tolerated not as well as 10- or 20-grain doses. Care in their use is necessary in nephritis and even then well-diluted, gastric disorder

may result; Spts. glycerylis nitratis is indicated in the intervals, in doses of ♏ij, four or more times daily; its effects last less than an hour. It may cause headache, flushing or (in larger doses) cyanosis or pallor, muscular relaxation, dyspnea and irregular pulse; erythrol tetranitrate in doses of gr. ss. t. i. d. has a more lasting effect. (See ARTERIOSCLEROSIS.)

(*b*) *The causes precipitating paroxysms.* Prophylaxis concerns exertion, excitement, coitus, chilling, constipation and dyspepsia; foods favoring flatulency should be avoided, and a light evening meal should be taken. Rest is imperative, as whatever exhausts the heart, precipitates the paroxysm. Bromides and chloral may avert nocturnal attacks. The patient should be provided with aromatics to dispel flatulency:

℞—Spiritus ammoniæ aromatici . . . . . ʒss
Spiritus chloroformi . . . . . ʒss
Spiritus glycerylis nitratis . . . . . gtt. j
Spiritus ætheris compositi . . . . . ʒss
M. et S.—To be taken in hot water (as a single dose).

(*c*) The *paroxysm.* *Amyl nitrite* was advised by Brunton (1867) as an analgesic and vasodilator; the pearls contain three minims of the remedy. They often operate powerfully and instantaneously, even when the blood-pressure is normal; sometimes they are useless. It is better to have the patient carry the spts. glycerylis nitratis or amyl nitrite with him, for the physician rarely sees the attack. Some member of the family should be taught to give a hypodermic of morphine and atropine. Morphine and chloroform by inhalation are dreaded when brain trouble exists, but they must be used; large doses of morphine are required and tolerated. A full drink of whisky, is excellent, as first aid.

(*d*) *Cardiac weakness* is treated by ammonia, camphor, strychnine and digitalis; morphine is carried in one hand and stimulants in the other.

℞—Tr. digitalis . . . . . ʒiv
Tr. strophanthi . . . . . ʒij
Tr. valerianæ . . . . . q. s. ad. ʒiv
M. et S.—One teaspoonful after meals.

Coffee dilates the coronaries and prevents cardiac weakness and the paroxysms. *Pseudo-angina* is treated according to the type. Electricity and arsenic are valuable.

TUMORS OF THE HEART.

The heart is rather insusceptible even to neoplasms in its immediate vicinity. Link collected 91 cases (1909). Carcinoma and sarcoma are the most frequent, and lipoma, myxoma, fibroma and myoma infrequent. They are usually secondary. Cardiac insufficiency, valvular compression, anginal symptoms or embolism sometimes develop.

Parasites are most often accidental postmortem findings. Echinococcus occurs twice as often in the right ventricle as in the left; 55 cases are reported. Cardiac rupture, sudden death from occlusion of the pulmonary trunks, relative pulmonary insufficiency or cardiac incompetence may occur. The cysticercus, the pentastomum and trichina are of purely anatomical interest.

CARDIAC THROMBOSIS—THROMBOSIS CORDIS.

This usually occurs at the time of, or after, death.

Symptoms.—(*a*) Entire latency. (*b*) Thrombosis sometimes develops during life and leads to pulmonary or arterial embolism. (*c*) *Sudden cardiac weakness.* (*d*) *Sudden valvular* stenosis, a ball thrombus forming in the auricle, and occluding, *e. g.*, the mitral orifice; it is beyond clinical recognition, although frequently associated with gangrene of the lower extremities or ascites.

NEUROSES OF THE HEART.

This topic is closely related to affections of the myocardium, especially the arrhythmias and heart-block.

Palpitation of the Heart.—Palpitation (cardiopalmus, cardiogmus or hyperkinesis cordis) is purely a symptom, the patient experiencing a fluttering sensation; in health the patient is not conscious of his heart action; extreme irregularity, or the pounding action in aortic insufficiency, may escape the patient's notice, while the normal heart may palpitate after overwork or abuse of coffee. It is closely related to the hyperdiastole caused by vagus stimulation.

Etiology.—1. Nervous Causes (Hyperesthesia of the centripetal heart fibers).—Nervous excitability is common especially in medical students, whose hearts must be examined when they first study physiology or physical diagnosis. Peter Frank feared aneurysm while writing his *Diseases of the Heart.* It is common in school children, from nervous tension, fear of examinations and public exercises; it is more frequent in females at puberty, climacteric or menstruation. It occurs constantly in exophthalmic goitre and in 50 per cent. of neurasthenics; neurasthenia cordis is attended by turgid face, red eyes, contracted temporal arteries and angioneurotic edema. Exhaustion, anemias, acute infections, lactation, poor hygiene, incipient tuberculosis and sexual excess may produce palpitation. The "irritable" heart of soldiers, observed by Da Costa in our Civil War, associated with arrhythmia, diarrhea, emotional causes and physical strain and often placed under this head, is better classified as muscular insufficiency.

2. Reflex Causes.—The products of indigestion and mechanical gastric distention, constipation, intestinal parasites, gall-stones, renal calculi, prostatic, uterine or ovarian disease and floating kidney are possible causes.

3. Toxic Causes.—Palpitation may result from the use of coffee, tea, alcohol, tobacco, narcotics and from gout.

4. Cardiac Causes.—Palpitation may occur in organically incompetent hearts, viz., valvular disease, acute or chronic myocarditis.

Symptoms.—The patient complains of "feeling the heart beat," "goneness," "fluttering," forcible or intermittent action of the heart, dyspnea and anxiety. Intermittent epigastric throbbing and vasomotor symptoms, as flushing of the face, pale urine, etc., are not uncommon. Phrenocardia, described by Herz (1909), is a syndrome composed of pain below

the nipple, palpitation and difficulty in breathing, and ascribed to sexual repression.

Physical Examination.—Sometimes the heart is absolutely normal. Some authors describe palpitation under tachycardia. The heart's rate is often increased after exertion or fright to 100 to 150, with violent arterial throbbing due perhaps to stimulation of the accelerator nerves. The heart is rarely slowed. A series of rapid beats followed by a series of slower beats is somewhat characteristic. If the pulse is irregular when the patient is quiet it sometimes becomes regular after exertion. The first apex tone is often accentuated, is even metallic or may be heard by the physician at some distance from the patient's chest. The systole may sound short. The second tone at the base is more often strong than weak. A murmur at the base is usually functional.

Diagnosis.—(*a*) The *etiology*. (*b*) *Negative results* of examination of the heart during the attacks, but particularly in the intervals. Coronary disease, arteriosclerosis, myocarditis, etc., are usually excluded with ease, but may coexist with palpitation. Palpitation is held by some to lead to dilatation or hypertrophy, but this is confusion of cause and effect. (*c*) If pain be present in nervous palpitation, it is more often lower (an "*epigastric cramp*") than the pain of organic lesions which radiates to the shoulder or arms.

Prognosis.—The outlook is generally favorable. The old belief that fright could cause death is as unfounded as most popular conceptions, and few such instances are recorded (Bollinger, Laache).

Treatment.—(*a*) If the cause is *nervous, suggestion* is valuable and repeated examinations assure the patient and avoid error. Regulated exercise, tepid baths with active friction, light meals, regular habits and possibly the rest cure are indicated. It seems from personal experience that isolation often increases introspection. In anemic cases iron, arsenic and strychnine should be given.

℞—Ferri valeratis,
Quininæ valeratis,
Ammon. valeratis . . . . . āā gr. xx
M. et ft. in pil. xx.
S.—One pill after meals.

A few doses of veratrum and belladonna often quiet the violent throbbing and general nervousness, probably affecting the peripheral fibers of the vagus. Sexual hygiene must be strictly observed.

℞—Fluidextr. veratri . . . . . gtt. x
Fluidextr. belladonnæ . . . . . gtt. xv
Aquæ . . . . . q. s. ad. ℥j
M. et S.—One teaspoonful every half-hour for two or three doses.

(*b*) When the cause is *reflex*, flatulency and constipation must be treated, with bromides, given well diluted.

℞—Sodii bromidi . . . . . ℥j
Spts. ammon. aromat. . . . . ℥ss
Syr. zingiberis . . . . . ℥iij
Aquæ . . . . . q. s. ad. ℥ij
M. et S.—One teaspoonful in hot water every half-hour for four doses.

A light evening meal of non-nitrogenous food is beneficial in children afflicted with night terrors or palpitation. (*c*) *The toxic type.* (*d*) *In valvular disease* (*q. v.*), belladonna and the ice-bag are indicated. Digitalis is of little use in the nervous type.

Arrhythmia.—There are five major varieties: (1) Sinus arrhythmia; (2) extra-systole; (3) heart-block; (4) auricular fibrillation; (5) the pulsus alternans.

1. The *juvenile, sinus or respiratory* form is found in all children at some time. The normal (fundamental or sinus) rhythm or contraction of the heart is initiated at the mouths of the great veins in the right auricle; this arrhythmia is a sinus irregularity and the chief variation is changing of the pulse-rate with respiration and, if the heart is rapid, shortening of the diastole. The heart function concerned in this type of arrhythmia is the stimulus production, and the vagus is largely concerned also (whence atropine often controls it temporarily). Hygienic measures are usually efficacious.

2. *Extra-systole* is the most frequent irregularity, and the usual cause of intermittent pulse. Its causation is increased irritability of the myocardium which answers to some abnormal stimulus and initiates a "premature," "abortive," "futile" contraction during the diastole. The source of the contraction is at the auriculoventricular node, or perhaps in the auricle or ventricle, or some point between them. In one group of cases, there is no heart disease and the extra-systole is physiological, as in young people, women at puberty or menopause, or mental or physical fatigue; in a second group, the cause lies in poisoning (tobacco, alcohol, coffee, digitalis), in reflex alimentary origin or sometimes in infections where it is ominous; in the third group, there is organic heart disease, or increased arterial tension from atheroma or nephritis. If the extra-systole occurs early in the diastole, no change in the pulse is noticed, as the aortic valves are not opened and a muffled third sound is heard; if it falls later in the systole, the aortic valves are opened, two sharp sounds are heard and the pulse shows the extra beat (pulsus bigeminus, trigeminus, polygeminus). There is always a systolic tone. The compensatory (diastolic) pause is lengthened after each systole, because of exhaustion of the left ventricle, which becomes "refractory" to stimuli, and the next beat becomes delayed. The time occupied by the normal systole plus the extra-systole plus the compensatory pause equals the time occupied by two complete normal cardiac cycles. In many cases, the extra-systole is described by the patient as a thump, fluttering or stopping of the heart. What appears simple extra-systole may turn out after years to be arteriosclerosis with high blood-pressure. Compensation may remain normal or may break, depending on the specific cause (*v. s.*), upon which also hangs the prognosis and therapy. In decompensation digitalis often helps (though it is an occasional cause of this arrhythmia).

3. *Heart-block* (*v. i.* Bradycardia).

4. *Auricular fibrillation* is also called perpetual arrhythmia, absolute irregularity or nodal rhythm. All rhythm is lost and the condition is associated with loss of tonicity, occurring in severe, specially rheumatic

heart disease with dilatation, particularly in mitral stenosis and also in the last stages of exophthalmic goitre and in myofibrosis; its cause apparently is always intracardial, but rarely coming from the acute infections or the use of digitalis. The auricle contracts or flutters with extreme rapidity but ineffectually and the bundle of His is overwhelmed with impulses attempting to pass to the ventricle; consequently the ventricle beats as it lists, irregularly as to sequence, force, equality or rate; these changes show disturbance in conductivity also. The normal auricular wave in the jugular tracing, representing auricular systole, disappears, as the auricle weakens and the curve shows 400 to 900 fine undulations from auricular fluttering (fibrillation). This gives the delirium cordis (permanent rapidity), temporary rapidity like paroxysmal tachycardia, and rarely a slow rate of action. A positive venous pulse appears in the jugulars. Mackenzie described its *sudden onset* in mitral stenosis and Lewis demonstrated that auricular fibrillation was the cause; these authors estimate that it is the cause of 50 to 70 per cent. of irregularities of the heart. Symptoms may be absent and the patient goes on unaware of the change; again there is breathlessness and signs of a weak right heart; in mitral stenosis the presystolic murmur disappears and a positive venous jugular pulse is detected. Rest may eliminate the trouble and here, more than in any other condition, digitalis works its marvellous effects, increasing tonicity and blocking the abortive impulses sent through the bundle of His by the excited auricle. Atropine may be used with benefit in certain cases.

5. The *pulsus alternans* is a disturbance not of the rate but of the size of the heart beats; it shows in the sphygmogram as alternating high and small waves. It indicates lasting *depression of contractility* and is always serious, occurring in mitral lesions, angina, advanced arteriosclerosis, old rheumatic hearts, high blood-pressure and irremediable cardiac fatigue, with death in two or three years. In pulsus alternans, we may hear strong tones alternating with weaker ones. It is to be distinguished from the p. bigeminus, in which the second beat is due to an extra-systole and is followed by a longer pause.

Tachycardia.—Definition.—Tachycardia is the usual term for rapid heart action, although polycardia and pyknocardia are better etymologically, for tachycardia implies quick (not rapid) heart action like the *pulsus celer*. Tachycardia is only a symptom. Some hearts normally beat 90 to 100 (120 in the case of Congreve).

Etiology.—(*a*) *Heart and vascular disease;* coronary, valvular or myocardial affections. (*b*) *Nervous affections;* brain and cord disease, as of the medulla (vagus centre), tumors, hemorrhage, bulbar paralysis, etc.; vagus neuritis, compression of its trunk by tumors, irritation of the accelerator nerve, exophthalmic goitre, *neuroses* as emotional tachycardia and reflex tachycardia from uterine or ovarian conditions. (*c*) *General diseases* as fevers, chronic troubles (tuberculosis, cancer), and *convalescence, exhaustion* or *excesses*. (*d*) *Toxic* causes; tea, coffee, alcohol, lead, digitalis, atropine, amyl nitrite, etc. Some individuals by voluntarily moving the ear or skin muscles of the neck can raise the heart's rate, through the spinal accessory nerve and the vagus (Tarchanoff).

A special variety is the *tachycardia paroxysmalis* described by Pröbsting and defined by Nothnagel as a slumbering of the vagus centre. Paroxysms of rapid heart action occur abruptly, last from a few seconds or minutes to days or even weeks, and usually end abruptly. It is seen mostly in strong, hard-working men who indulge in excesses in alcohol, tobacco, etc.

The pulse becomes smaller and weaker, and its rate reaches 150 or even 280. It is usually regular, though arrhythmia may mark the beginning and end of the seizure. There are three types: In one the auricular beat precedes the ventricular, as normally; in a second, more frequent form, the auricles and ventricles beat synchronously (see nodal rhythm); in the third type, the ventricle beats independently of the auricle; the irritation is in the auricle, auriculoventricular bundle or in the ventricle, respectively. The heart often dilates. The pause of the heart is lessened, the systole incomplete and the diastole shortened. The systolic tone is often clicking. The vessels pulsate violently and the patient is anxious, depressed and late in the course, shows signs of venous stasis. The urine is of low specific gravity. An acute distention of the lungs like that observed in valvular disease, may be caused by vagus or phrenic irritation or tonic contraction of the diaphragm.

Diagnosis.—Permanent tachycardia suggests organic disease in the medulla or nerve trunks. The physiology of the vagus and accelerator nerves is still obscure from the clinical stand-point. It has been held that (*a*) increase in the pulse up to 120 indicates sympathetic irritation (accelerator stimulation), which is relieved by morphine; (*b*) increase from 120 to 180 indicates paralysis of the vagus, which is relieved by pressure on the vagus trunk; (*c*) increase over 180 is indicative of sympathetic irritation in addition to paralysis of the vagus. Sympathetic symptoms may prevail, as exophthalmos, wide pupils and interpalpebral increase. Evidences of centric vagus involvement may exist, such as respiratory, laryngeal or gastric crises, polyuria, etc.; sudden onset and cessation suggest a bulbar origin.

Prognosis.—The permanent is less favorable than the paroxysmal form. Bouveret holds that permanent cure is rare and reported 4 cases of sudden death. The infrequent autopsies (only 16 out of 135 cases collected by Hoffmann in 1900) show degeneration of the heart muscle.

Treatment.—Stimulation of the vagus is effected by faradization of the neck, holding of the breath, by Valsalva's experiment, by firm bandaging of the abdomen, pressure on the right vagus, the use of the ice-bag, swallowing ice, induction of vomiting by apomorphine and inverting the patient. Morphine, asafetida, valerian and bromides are frequently of value. The causal factors require consideration.

Bradycardia.—Definition.—Bradycardia, spanocardia and brachycardia are the usual terms for *slow heart* but *oligocardia* is a better designation. A *pulsus tardus* is normal to some individuals and certain families in successive generations. It is often accommodative, as in aortic stenosis. The apparent pulse-rate must be verified by cardiac auscultation. Paroxysmal bradycardia may occur with weak heart, palpitation, difficulty in breathing or gastric distress.

Mackenzie distinguishes: (1) normal bradycardia, where all the

heart's chambers beat slowly; (2) slow heart due to missed beats; (3) nodal rhythm (*v. i.*), where the auricle ceases to beat or beats synchronously with the ventricle; (4) heart-block; and (5) vagus slowing of the heart—stand-still of the whole heart.

Etiology.—The etiological factors are: (*a*) *Acute infections;* bradycardia is very common during or after typhoid, pneumonia, diphtheria and rheumatism, probably caused by acute myocarditis (lessened conductivity or heart-block) or by a vagus neuritis. (*b*) *Digestive disorders;* jaundice, constipation, gastric cancer, ulcer, distention or vomiting and gall-stones are the most frequent causes. (*c*) *Respiratory disease* is a less frequent cause; emphysema occurs frequently with a slow, strong pulse. (*d*) *Circulatory disease,* such as aortic stenosis, fatty heart, *coronary atheroma* or chronic myocarditis, lessens conductivity and contractility. Dehio uses atropine to distinguish between (i) disease of the heart muscle, upon which the drug has but little effect, and (ii) slow heart due to inhibition of the vagus, in which it brings the heart to, or above, the normal rate. (*e*) *Toxic conditions* such as poisoning from lead, alcohol, tobacco, digitalis and other drugs; in cholemia and in uremia it is unfavorable. (*f*) *General troubles,* such as diabetes, cachexia, anemia or exhaustion, lessen the irritability of the heart muscle. (*g*) *Nervous conditions,* apoplexy, meningitis, disease of the medulla or cervical cord, vagus stimulation by tumors, compression of the vagus trunk, are factors in its development. Jacob describes an angiospastic form (bradycardia with chills, wide pupils, cold skin, loud, second aortic tone, etc.).

Heart-block.—The *Adams-Stokes syndrome* was first described by Morgagni (1761) and Spens (1792) but more fully by Adams (1827) and Stokes (1846). It is due to *disturbed conductivity* through the *bundle of His,* who in 1893 found a band-like muscular bundle connecting the right auricle and the ventricles. It measures 18 millimeters in length, 2.5 millimeters in width and 1.5 millimeters in thickness, and courses posteriorly in the septum ventriculorum, whence it reaches the right and left ventricles and their valves. J. G. Wilson considers that the bundle is an intricate nerve pathway. According to Gaskell (1883), and especially Erlanger (1906), slight compression of His's bundle increases the interventricular pause; marked compression causes intermittency in the ventricular contractions; complete compression results in independent contraction of the auricles and ventricles, known as "heart-block." Its *causes* are various; mild, transient or incomplete heart-block may occur after infections, as typhoid, diphtheria, etc., in old rheumatic hearts, arteriosclerosis, from the use of digitalis or strophanthus, or possibly from nervous causes, *e. g.*, by the vagus. In the more severe and permanent cases, organic changes are discovered in His's bundle, as gummata, an endocarditic patch in Stengel's case, postdiphtheritic myocarditis (A. S. Dunn), acute myomalacia cordis, tumors and changes in the medulla.

Symptoms.—The complete syndrome consists of (1) bradycardia; (2) pulsation of the cervical veins, exceeding in rate the arterial pulsation; (3) cerebral disturbances, as syncope and convulsive seizures; and (4) accessory symptoms. The normal rhythm and stimulus to contraction

begin in the right auricle and pass over the bridge or bundle of His to the ventricles. Disturbance in *conduction* leads either to delayed contraction of the ventricles or to complete "blocking" of the stimulus, when the ventricles beat independently of the auricles (ventricular rhythm).

1. *Bradycardia.*—With the normal pulse of 70, one-fifth of a second intervenes between the auricular and ventricular systoles; if this interval is much increased, there is disturbance of conduction and the transmission of the contraction wave from the auricle to the ventricle is "blocked"; the auricular movements may be seen in the pulsating cervical veins, the fluoroscope or in polygraph tracings. When the ventricular contractions exceed 36 a minute, there is a definite relation between the ventricular beat and the auricular waves (seen in the pulsating jugulars), as 1 ventricular to 2, 3, or 4 jugular beats; if the ventricular rate is 30 or less, there is no definite ratio. An interval of one or two minutes may intervene between the ventricular contractions (or radial pulse beats). During the ventricular systole, a muffled sound, due to auricular contraction, may be heard and identified with it as one watches the pulsating jugulars. The pulse may fall to 6 beats.

2. *Jugular pulsations* are synchronous with the auricular contractions (*v. s.*) and the radial pulse with the ventricular.

3. *Cerebral disturbances* are not necessarily connected with dissociated action of the auricles and ventricles. Attacks resembling apoplexy are marked by coma, pallid or congested face, stertorous breathing, aphasia, etc., and are probably due to venous stasis in the brain. In other instances there is vertigo or epileptiform attacks ensue, from arterial anemia. In a personal observation the convulsive attacks came on with the eyes open, so that an incorrect diagnosis of hysteria had been made.

4. *Accessory manifestations* embrace angina, Cheyne-Stokes's respiration, cardiac asthma, etc.

Prognosis.—The constant is less favorable than the paroxysmal type.

Treatment.—The causal therapy alone is of value. Morphine must be given with care where there is disease of the central nervous system. Strong cardiants are usually contra-indicated and in the Adams-Stokes's syndrome are actually dangerous (see digitalis, page 374). Alcohol is injurious. Mercury and iodides help, maybe cure, syphilitic disease in His's bundle, unless the gummata are sclerosed. Atropine may be of service in partial heart-block.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Definition.—Inflammation of the endocardium. Its clinical importance attaches to the fact that chiefly the valves are involved. Two varieties are distinguished, the *acute* and the *chronic*. Bouillaud, in 1840, first placed the disease on a scientific basis.

I. Acute Endocarditis.—This is a *secondary, general* and *mycotic* disease. Its clinical varieties—(*a*) malignant or ulcerative endocarditis, and (*b*) benign or verrucose endocarditis—*differ only in degree;* the term benign is relative, as most endocarditides entail serious sequels. Litten divides endocarditis into (*a*) the *benign* forms produced by rheumatism, chorea, etc.; and (*b*) *malignant,* (i) non-suppurative and (ii) suppurative or septicopyemic.

(**A**) **Malignant (Ulcerative or Septic) Endocarditis.** — Etiology. — The heart lesion is either (1) *secondary* to some clearly causal infection, or (2) *cryptogenetic,* when the causal infection is not apparent; care in examination and at autopsy usually reveals the cause. The atrium, bacteriology, mode of extension and symptoms are described under Septic Infections.

The disease is more frequent in women than in men, even excluding puerperal infections, and between the years of twenty and forty. Its frequency is 2 per 1000 patients. *Seventy-five per cent.* of cases *develop upon an old valvular lesion* by which the local physiological resistance is lessened. The literature contains 11 cases developing at a patent ductus Botalli (Hart, 1904).

Pathology.—Malignant endocarditis in 86 per cent. involves the *left heart* which is far more frequently the seat of previous lesions and its arterial blood favors the growth of microörganisms; the right heart is involved more frequently (12 per cent.) than it is in other types of endocarditis; both sides are diseased in 2 per cent.

The process may involve the walls (parietal or mural endocarditis), or more frequently attacks *the valves* (valvular endocarditis), which are often already indurated by previous simple endocarditis and are subject to greater mechanical activity. The valvular localization is embolic. The endocardium becomes opaque, and irregular deficits occur on the undersurface of the aortic or the auricular surface of the mitral valves; later there is injection of the endothelial coat and a grayish deposit. Exudation is more abundant on the mitral than on the aortic or pulmonary valves. *Thrombi* and excrescences develop on the erosions, whose removal discloses subjacent *ulceration.* The thrombi, especially in pneumococcic and gonococcic infections, consist of lamellated fibrin and bacteria. The *vegetations* may attain the size of a walnut and often extend from one valve to another by contact infection; some thrombi are purely mechanical deposits of fibrin, but *endocarditis is always mycotic.* Heiberg first found microörganisms (1869) and Weichselbaum first obtained pure cultures. The following bacteria have been found: streptococcus, staphylococcus, pneumococcus, less frequently the gonococcus, colon, diphtheria, influenza, tubercle and typhoid bacillus, meningococcus and, very exceptionally, other bacteria. The same organism may produce either the benign or malignant form. *Experimental endocarditis* has been produced by inoculations of various organisms, usually after wounding the valves by a sound introduced through the cervical vessels, but also without trauma. The pyogenic organisms are said to attack more frequently the mitral, and the pneumococcus rather elects the aortic valves. The mitral valves are more often involved than are the

aortic. The process may invade the aorta or the pulmonary artery. Destructive ulceration through one leaf of the valve may produce acute *valvular aneurysm*, which, when aortic, projects toward the ventricle, and when mitral, projects toward the auricle. If the necrosis destroys both lamellæ of the valve, the perforation leads to valvular insufficiency. Ulceration of the chordæ tendineæ or papillary muscles also produces valvular insufficiency; necrosis of the parietes results in a "heart ulcer," which sometimes leads to communication between the ventricles or to rupture of the heart. Dislodgement of tissue particles, thrombi or bacteria occurs frequently, usually in the arterial circuit. The effects vary with the location of the *infarct* (see SYMPTOMATOLOGY) and with the character of the microbes; *i. e.*, emboli act mechanically, infectively or in both ways. Streptococci are said to produce anemic infarcts, while staphylococci result in suppuration. Loehlein described typical glomerular lesions due to the Streptococcus viridans; their epithelia swell, proliferate and desquamate; necrosis sets in, with ultimate organization. Pathological changes *in other organs* are those of (*a*) general *sepsis*, such as parenchymatous degeneration or splenic tumor, (*b*) *embolism*, (*c*) *stasis* when the heart fails, and (*d*) the primary disease.

SYMPTOMS.—The clinical features vary greatly, according to the basic disease, embolic involvement and virulence of the microörganisms. The onset may be violent or most insidious. In the majority of cases the septic element prevails. The *various types*, artificial, though convenient for description, are:

1. *The Typhoid Form.*—The toxemic symptoms, such as apathy, delirium, dry tongue, distended abdomen, diarrhea and splenic tumor are suggestive of typhoid. The resemblance may be heightened by hemorrhage of the bowels, rose-like spots and the diazo reaction. Dilatation of the right heart and cardiac murmurs, so important in endocarditis, are not uncommon in typhoid. But at this point certain variations arouse doubt; there is no Widal and no typhoid bacillemia and certain septic symptoms or cardiac signs come to the front. In the *skin* or upper air passages, *emboli* appear as spots with white or yellow centres and hemorrhagic peripheries, or as pemphigus, gangrene or suppurative foci. Infarcts sometimes occur in the conjunctiva, and in the *retina*, simple hemorrhage or actual infarcts with yellow centres are frequently detected. The entire eye-ball may suppurate. The frequent *splenic infarction* is characterized by sudden pain, tenderness, enlargement and sometimes perisplenitic friction. Embolism is often accompanied by fever, chill and vomiting. *Renal infarction* is very frequent, is attended by pain in the loins, hematuria, albuminuria and sometimes anuria or dysuria. Severe nephritis is common. *Cerebral embolism* usually occurs in the left Sylvian artery and results in the usual stroke and hemiplegia with aphasia. *Pulmonary infarcts* are caused by lesions of the right heart or by auricular thrombi, and occasion sudden pleuritic pain, dyspnea, hemoptoë, temperature and possibly a small area of crepitant rales, pleural friction or consolidation. Embolism also occurs in the liver, in the mesenteric artery (with enterorrhagia and peritonitic symptoms), in the muscles and joints, testes and parotid; in the heart

and various mucosæ; in the meninges, pleuræ, pericardium, peritoneum and in the peripheral arteries, sometimes causing gangrene or embolic aneurysm. The pneumococcus is often the cause of this type.

The *course* may be peracute, acute or chronic. Death occurs from sepsis, embolism or cardiac failure.

2. *The Septic or Intermittent Type.*—This form runs an irregular fever-curve, with chills, drenching sweats and enlarged spleen. Heart symptoms may be absent or the relaxation of the right heart and the murmur are called functional. The paroxysms are more irregular than in malaria and no response to quinine is obtained. Sepsis is suspected but the localization is not immediately determined. The streptococcus is often causal. Cardiac findings, embolism or clearly septic manifestations may establish the diagnosis. The course is usually several weeks but may cover several months to over a year.

3. *The Visceral Forms.*—In these cases one organ is especially involved. In the *cardiac form*, the heart murmur seems hemic, until a typical valvular lesion develops, or, if one already be present from older valvular disease, until another appears, as a diastolic following a systolic murmur. In the *cerebral form*, meningitis, apoplexy, coma or psychoses may dominate the clinical picture. The symptoms referable to any organ which is the seat of embolism may seem to indicate a primary disease; thus acute yellow atrophy, pernicious anemia, Banti's disease, tuberculosis, nephritis, rheumatism, etc., have been incorrectly diagnosticated.

Special Symptoms.—(1) All types of *fever*, from intermittent to continuous, are noted, although it may be absent for long intervals. (2) The *heart* is *always somewhat disturbed.* Its action is more frequent and variable, now being rapid, again slow and often irregular. The heart seems to beat harder and the pulse may throb, although the irritable overaction is attended by low arterial tension. Palpitation is frequent. Cyanosis is not common. (3) *Respiration* is *always increased*, from toxemic centric stimulation, pain or complications as septic pneumonia, infarcts, pleurisy, etc. (4) The *digestive tract* may be affected, as nausea, vomiting and diarrhea from the general sepsis. Embolism has been mentioned. The liver is enlarged, perhaps tender or the seat of friction. Icterus is ominous and referable to duodenal catarrh or oversecretion of bile (polycholia). (5) the *spleen* is almost always palpably enlarged from sepsis or embolism. (6) The *urine* shows evidences of the sepsis or stasis by albuminuria, with or without cylindruria, and of the "blood dissolution" and embolism by hematuria. The course may resemble that of subacute nephritis, present in 33 per cent. of cases. (7) The *nervous system* is irritated or dulled by the sepsis toxins; or embolic meningitis or encephalitis may intervene. (8) The *muscles* are frequently tender and inflamed and may undergo suppuration. The bones are often tender, particularly the sternum, and the *joints tender* or *swollen*, although the swelling is more frequently periarticular than intra-articular; bacteria are seldom found, whence the occasional resemblance to rheumatism. (9) The *skin* may be the seat of embolism (*v. s.*) or of toxic sudamina, erythema, *purpura* or *polymorphous septic rashes.* Bullæ, gangrene, phlegmons or bed-sores are also observed. (10) Optic neuritis is frequent.

(11) The *blood* exhibits marked anemia; without heart findings, the anemia and splenic tumor have been confused with pseudoleukemia. The leukocytes are not often increased. Bacteria may be cultivated from the blood (*v.* pages 19 and 20).

Bacteriological Forms.—(*a*) *Streptococcic Endocarditis.*—The streptococcus is the usual cause of rapidly fatal ulcerative endocarditis, with severe repeated chills and high, irregular fever; chronic infective endocarditis is almost invariably streptococcic, especially the S. viridans of Jochmann and Schottmüller (see page 21); this type is often latent, chronic and is attended by slight if any leukocytosis, great frequency of positive blood cultures (Libman and Jochmann, 95 per cent.) and the absence of suppuration; Schottmüller designates this type endocarditis lenta. (*b*) *Staphylococcic endocarditis* is less frequent. The course is acute and attended by high fever, pustules in the skin, disseminated hemorrhages and septic infarcts, especially in the kidneys. (*c*) *Pneumococcic endocarditis* is usually acute but may last over one year; frequently follows pneumonia (*q. v.*), less often cholecystitis; begins with gradual fever rise associated with chills; and is often attended by suppurative meningitis. (*d*) *Gonococcic endocarditis* (Ricord, 1847) may result from Neisser's coccus or from mixed infection. Cases of recovery are instanced.

Diagnosis.—Bamberger's remark still holds: "The diagnosis of acute endocarditis is seldom easy, usually difficult and often impossible." Without physical changes in the heart, the diagnosis is only presumptive. A proper etiology, malaise, weakness or emaciation, the presence of an old valvular lesion, fever or other evidences of sepsis, signs of cardiac insufficiency, such as irregular, weak pulse with apparently strong cardiac shock, and dilatation are strongly suggestive and may be confirmed by continued observation, embolism, change in the valvular murmur and positive blood cultures. The difference between malignant and benign endocarditis is that the former is septicemic (bacillemic) or pyemic. Actual hypertrophy of the heart not only requires time for development but adequate nutrition, of which the acuity of the disease seldom admits. The misleading visceral forms have been discussed (*v. s.*). In 37 per cent. of cases, the heart is negative, in 44 per cent. the murmur is systolic and in 5 per cent. diastolic. Absolute certainty rests upon our ability to diagnosticate by physical signs, the presence of a mitral regurgitation (*q. v.*) which is the most frequent form, or of an aortic regurgitation (*q. v.*), plus the *signs of sepsis* of which the disease is a part ("arterial pyemia"). The valvular murmur may disappear after embolism. (See Typhoid, differential table.)

Diastolic murmurs are very rarely functional and their appearance is therefore especially suggestive. Acute myocarditis may produce a systolic murmur, but is usually attended by marked early muscular insufficiency, weak heart shock and irregular pulse, weak systolic murmur and a moderately accentuated second pulmonary tone; fatty and cloudy degeneration of the myocardium is the rule in acute endocarditis, besides which the muscle may suffer from coronary closure or embolism, stasis and direct involvement, by contiguity, through inflammation or ulceration.

Prognosis.—Satterthwaite's figures show 5 per cent. of recoveries. It depends on the bacteriology, the number of infarcts and their location, the variability of the heart murmurs (which may indicate danger of embolism), and the intensity of the sepsis. Recovery from the gonorrheal form is relatively frequent. Musical murmurs with fever are usually ominous. Apparently mild endocarditis may become severe. The course may be rapid or severe, of a few days' duration, or a slower evolution of over a year.

Treatment.—(See Benign Endocarditis and Septic Infections.)

(B) Acute Verrucose or Benign Endocarditis.—This variety is more frequent, constituting 1.5 per cent. of clinical cases, and is always secondary. It differs from the malignant type in degree of virulence only.

Etiology.—(*a*) *Rheumatism* (*q. v.*), its most frequent cause, accounts for 60 to 85 per cent. of the cases. Over 20 per cent. of rheumatic patients develop endocarditis; in children it is very frequent, for in them the heart "resembles an articulation." Muscular rheumatism, angina or erythema nodosum may occasion endocarditis. (*b*) *Chorea:* 30 per cent. of clinical and 82 per cent. of fatal cases are complicated by endocarditis. (*c*) Of other *specific infections*, scarlatina ranks first, causing 10 per cent. of endocarditides, and active or latent foci in the tonsils, or elsewhere, rank not far behind. Endocarditis is far more uncommon after typhoid, diphtheria, measles, erysipelas, gonorrhea, variola, syphilis, grippe, tuberculosis, etc. (*d*) Occasionally it is associated with trauma, burns, cancer, Bright's disease, gout or diabetes. (*e*) It is most common in males between the years of twenty and thirty.

Pathology.—The vegetations are verrucose, wart-like, papular or even pedunculated, are usually small and occur at or above the lines of closure of the valves, whereby their function is impaired and valvular insufficiency or stenosis results. At times the vegetations are cauliflower-like or condylomatous. They are grayish, gelatinous and transparent; they become whiter and firmer with age and consist of two strata, the deeper being granulating endocardium and subendocardial tissue and the superficial layer consisting of thrombi from the coagulable elements of the blood. The signs of active inflammation, such as redness or swelling, are usually lacking, redness being sometimes confused with postmortem imbibition of hemoglobin from the blood. Vegetations must not be confused with Albini's nodes, seen in infants and children. The vegetations are at an early stage, cellular from leukocytic exudation and tissue proliferation and usually contain bacteria (see Ulcerative Type); the inflammation is probably primarily embolic in the myocardium, and affects the valves secondarily. The outgrowths becoming detached, *emboli* are released and produce mechanical hemorrhages or anemic infarcts, not suppurative infarcts as in the ulcerative type. The fate of the vegetations is (*a*) *embolism;* (*b*) *organization* with bare possibility of restoration to normal; (*c*) more often, *organization with valvular lesions* resulting from retraction, induration and calcification; (*d*) being a *locus resistentiæ minoris, endocarditis* may be *recurrent;* (*e*) the inflammation may lead to malignant endocarditis.

Localization.—The vegetations are largely on the same surface of the same valves as in the first type, and in the left heart; conditions in fetal life predispose to endocarditis on the valves of the right heart, or on fetal openings or defects. The mitral valves are most affected (50 per cent. of the cases alone or 80 per cent. when combined with other lesions), next the aortic (13 per cent. alone or 40 per cent. when combined with other lesions), and far less frequently, the tricuspid and pulmonary valves and the walls, chordæ or parietes.

Symptoms.—The symptoms are not characteristic unless the valves are considerably involved. Simple endocarditis therefore is found far more frequently at autopsy than at the bedside; *i. e.*, it is very often *latent.* An increased pulse may indicate palpitation, a valvular lesion or a myocarditis, though the latter is the more likely cause. Daily examination of the heart in such diseases as rheumatism may reveal a gradual involvement of the endocardium, with moderate fever, cardiac unrest, oppression, palpitation or insufficiency. Pain is generally pleuritic or pericarditic. A valvular bruit often follows muffling or irregularity of the cardiac sounds, and is still later followed by the signs of mitral or aortic insufficiency (*q. v.*). In marantic causation, as cancer and tuberculosis, murmurs are generally absent. The infrequent diastolic murmurs are more distinctive than the systolic, for they are rarely functional. *Embolism* is very important, as in the first type, but mechanically rather than bacteriologically.

Diagnosis.—Conservatism in diagnosis is necessary and acute benign endocarditis is much too often diagnosticated. The most conspicuous symptom is *fever,* though it is not characteristic and may blend with that of the causal disease; the most important findings, objectively, are the signs of valvular heart disease; for these findings one must often wait, because in many cases the evolution alone is final. More stress should be placed upon the results of percussion than on the murmur itself. The murmurs are mistaken for functional murmurs accompanying febrile relaxation of the heart chambers or *myocarditis,* which also always accompanies endocarditis.

Recurrent endocarditis shows not only the fever and other symptoms caused by the fresh lesions, but also the *hypertrophy* of the older valvular lesion. An aortic diastolic or a mitral presystolic murmur is more likely to indicate an *old lesion.* With repeated examinations fresh murmurs may be heard in addition to the old ones. (See Mitral Insufficiency and Ulcerative Endocarditis.)

Prognosis.—(*a*) *As to life,* the outlook is generally good, unless (i) emboli lodge in important structures, such as the basilar or coronary arteries, or (ii) the process becomes malignant. (*b*) *As to complete recovery,* (i) valvular lesions are usual which are anything but "benign"; (ii) fresh recurrent endocarditis is very common; and (iii) complete recovery is uncommon, though not unknown.

Treatment.—The treatment of the two forms of endocarditis is considered together. (*a*) *Prophylaxis* resolves itself into the treatment of the causal acute infection, such as rheumatism or sepsis. The administration of salicylates in rheumatism shortens the rheumatic attack.

(*b*) *Rest* spares the heart and probably lessens the percentage of cases involved. Psychical, physical and cardiac rest is the prime essential, and requires that the patient lie, not sit, in bed for many weeks after temperature and cardiac weakness have wholly disappeared. Should relative recovery occur, the general care of the heart's strength must be insisted upon as in chronic valvular disease and, with intercurrent affections or with the appearance of temperature, the patient must be confined to bed. Senac (1749–1783) recognized the causal importance of inflammation in heart disease, the necessity of avoiding strain and of regulating the amount of fluid and food ingested, the relations of liver and stomach disturbance and the advantages of mental quietude. (*c*) The *sepsis* is treated by moderate doses of quinine, with three ounces of red wine every four hours; bichloride of mercury and other antiseptics are futile; there may be some virtue in hexamethylenamine. (*d*) The *irritable heart* may be quieted by the ice-bag. Sinapisms sometimes act favorably but blisters are to be avoided. Cardiac stimulants should be employed with caution; digitalis is only indicated when tumultuous heart action threatens to induce embolism. (*e*) *Symptomatic treatment:* For nervous symptoms, sodium bromide is indicated. Sulphonal and trional are to be avoided when the heart weakens. Hydrotherapy, aside from simple sponging, is inadvisable, for the joints and muscles are very tender and movement may provoke embolism. Diarrhea of septic origin should not be checked at once, because it is nature's effort at elimination. A light diet should be given because of the fever. Potassium iodide is often administered as a resolvent after the acute stage has passed. (*f*) Vaccines and serums injure more often than they help.

II. Chronic Endocarditis. Chronic Valvular Disease.—Etiology.—Valvular disease constitutes 5 per cent. of all diseases. It most often develops from (*a*) *acute verrucose endocarditis* and in 60 per cent. the cause is rheumatism. One-half the cases of valvular disease are in children and over one-third occur between 20 and 30 years of age. Some apparently primary cases are seen mostly in young or adolescent subjects and in women, but doubtless some minor or forgotten infection explains this group. (*b*) *Atheroma* is an important cause; it is promoted by overexertion, as in the working classes, by advanced years or by chronic nephritis, gout, diabetes, *syphilis*, alcoholism and lead poisoning. It is more common in males. Chronic valvular disease may develop from other causes than actual endocarditis, *e. g.*, from (*c*) *rupture of the valves;* this occurs most often in the aortic valves during supreme physical efforts and is favored by previous valvular lesions, although sometimes observed in normal valves. Healing is rare. (*d*) In *relative valvular insufficiency* the normal valves are unable to close their orifices because the heart is dilated; it is most commonly tricuspid and mitral, and more rarely aortic and pulmonary. Relative stenosis is not clearly established. (*e*) *Other rarer causes* are aortic aneurysms, neoplasms, valvular aneurysms and cardiac thrombi.

Pathology.—Small grayish-red swellings are sometimes seen in the early stages. Foci of endocardial thickening and retraction (*endocarditis retrahens*) develop, in which calcification, and even ossification, of the

valves may be present. The valves become opaque, rigid, unable to unfold, adherent to each other or distorted by their own thickening and retraction or that of the chordæ or papillary muscles. Acute exacerbations may occur and then the ordinary vegetations appear. Embolism occurs in 30 per cent. of cases. The myocardial changes are hypertrophy, dilatation and myocarditis. The valves most often involved are the mitral (78 per cent.), especially in adolescence, then the aortic (20 per cent.), especially in syphilis and arteriosclerosis, and finally the tricuspid (1 per cent.) and the pulmonary valves. Combined valvular lesions occur in 33 per cent. of cases. The process often extends by continuity of surface from the mitral to the aortic valves. When one valve is but moderately involved, the others of the same valvular orifice may compensate by stretching to cover the defect, especially in aortic disease. In fetal cases the right heart is more frequently involved.

Valvular insufficiency is caused by (*a*) thickening and retraction or even calcification of the valves or the chordæ tendineæ and papillary muscles; (*b*) massive vegetations; (*c*) growing together of the valves, or of the valves with the wall of the heart or aorta; or adhesions between the papillary muscles and tendons; (*d*) valvular aneurysm, perforation, rupture or tearing of the tendons or muscles. *Valvular stenosis* (stenosis of the orifice) results from (*a*) adhesions between the valves, (*b*) retraction, calcification, fibrosis of valves or ring of insertion and (*c*) vegetations. Combined valvular insufficiency and stenosis are very common.

Symptoms.—The symptoms of valvular disease are (*a*) the *cardiac findings* peculiar to each lesion, which will be first considered, and (*b*) the *general symptoms* more or less common to all forms of cardiac insufficiency, the description of which will follow the individual lesions.

Aortic Insufficiency (Corrigan's Disease).—Aortic regurgitation was described by Cowper (1705), Vieussens (1715) and Hodgkin (1829), but the first full description was Corrigan's (1832).

Etiology.—1. *The endocarditic form* explains 27 per cent. of cases. The sinuses of the valves may fuse, vegetations are present, or the valves grow together at their margins and shrink; this is more frequent than valvular perforation or ulceration, which is rapidly fatal. If calcification develops there may be coincident stenosis of the aortic orifice. The endocarditic form usually follows a previous mitral lesion, occurs at an earlier age and is not attended by the arteriosclerotic changes in the aorta and coronary arteries which characterize the atheromatous form.

2. *The atheromatous form* accounts for 44 per cent. of leakage. Atheroma may be slight in the aorta and located mostly on the valves. It is a slow, degenerative process with marginal valvular retraction (see Arteriosclerosis); it is caused by excessive use of alcohol, by continued hard work or athletics. It is often, therefore, seen in middle-aged men. The valves may adhere to the aorta. Their edges are rounded and present no endocarditic masses. The atheromatous process may overlie or actually invade the coronary orifices; this is obviously dangerous. Coincident stenosis is uncommon.

3. *The syphilitic form* explains 29 per cent. In half the cases of pure aortic leakage, the aorta shows a *luetic mesa-ortitis* and about three-

quarters show the Wassermann test (pages 208 and 219). The following forms are infrequent:

4. *Relative insufficiency* occurs mostly in cases of aortitis, aneurysm and arteriosclerotic *dilatation of the root of the aorta;* the aorta relaxes one centimeter in circumference after the fortieth year. It may also occur in cases of *dilatation of the left ventricle,* resulting from hypertrophy, myocarditis or renal disease.

5. *Congenital Defects.*—These are rare; absence of a valve may be in part compensated for by the others, though anatomical anomalies predispose to sclerosing endocarditis.

6. *Trauma.*—Only 46 aortic cases are recorded in a total of 72.

Mechanism.—From aortic incompetence, a diastolic regurgitation of blood into the left ventricle takes place from gravity and contraction of the aorta. Blood currents are caused by the blood flowing (normally from the left auricle and pathologically from the aorta) into the left ventricle, thereby producing the *diastolic murmur.* The *left ventricle becomes dilated* to accommodate its added volume and *hypertrophied* to propel the normal plus the regurgitated blood; the heart may weigh 50 ounces ("ox-heart"—*cor bovinum*). The hypertrophied left ventricle suddenly forces a large amount of blood into the arteries, and thus causes the many *arterial phenomena* (Stewart believes that the amount of regurgitated blood is small).

Physical Signs.—1. *Inspection.*—(*a*) *Precordial prominence* is observed chiefly in plastic chests and in the endocarditic type and therefore mostly in women and children. Mensuration confirms the prominence (the right chest normally measures nearly an inch more than the left). (*b*) The heart *shock* is powerful and diffuse. (*c*) The *apex beat* is *dislocated downward* (6th, 7th, 8th interspace) and outward, beyond the nipple or anterior axillary line. It is *heaving,* pulsating and superficial. The lung is pushed back and slight rotatory systolic retraction of the chest wall is seen. These findings are referable to the dilated and hypertrophied left heart. If the leak is slight, there may be little or no dilatation or hypertrophy, clinically. The apex also lies low in arteriosclerotics, from relaxation of the aorta. (*d*) Pulsation in the first and second right intercostal spaces is usually dynamic, because the blood expelled with extra force causes dilatation of the aortic arch. (*e*) The *peripheral vessels pulsate with unusual violence;* pulsation in the jugular fossa or abdominal aorta may simulate aneurysm, and the temporals, carotids and radials attract attention before the patient undresses. The crossed leg may pulsate and the head may throb (Musset's nodding). These are all caused by ventricular hypertrophy, the extra volume of blood thrown into the vessels or an accommodative dilatation of the peripheral vessels. Pulsation may also be seen in the retina spontaneously or by pressure on the eye-ball, as occurs also in Basedow's disease. The liver may pulsate, though this is rare because a strong heart action with stasis is necessary. (The liver also pulsates in tricuspid incompetency from venous filling of the organ.) Pulsation also may occur in the spleen, tongue, palate and penis; the pupils may contract and dilate rhythmically. (*f*) The *capillary* (*Quincke's*) *pulse* occurs in over 80 per cent. of cases; it is also seen in health, fevers

or anemias. It is a symptom of overfilling of the arteries and capillary dilatation and is best observed when the hypertrophy exceeds the dilatation; it disappears when the heart weakens. It is elicited by exerting slight pressure on the tips of the finger nails, on the lips by a glass slide or by rubbing the forehead until it is red; there is a systolic reddening and a diastolic paling in these parts. It is sometimes found in the retina, palate or in complicating erysipelas. (*g*) The passing over of the blood from the arterioles into the veins may produce the *centripetal venous pulse*—most obviously in the forearm, and caused by relaxation of the vessels, as in fevers, nervous lesions and chlorosis; by delicate skin; and full heart action. (*h*) The *arteries are actually lengthened.* (*i*) Patients often assume the dorsal decubitus, for gravity increases the leakage; subjects with mitral insufficiency usually sit. The findings by inspection are practically pathognomonic.

2. *Palpation.*—(*a*) The hand corroborates the results of inspection—as the heaving, *throbbing apex* and the diastolic impulse. (*b*) *A diastolic thrill* over the base is infrequent, occurring mostly in cases of endocarditic origin. (*c*) In the jugulum, especially in arteriosclerotics, the widened aorta

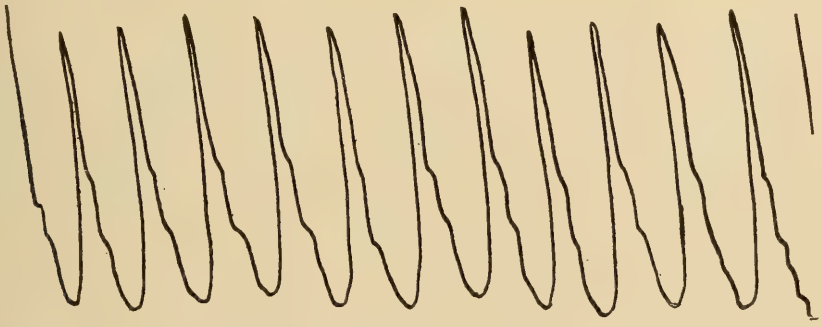


Fig. 21.—Sphygmogram of case of uncomplicated aortic leakage.

can be felt. (*d*) The *pulse* findings may seem paradoxical; the arteries are filled violently (the pulsus durus), though the tone of the vessels is less than normal. Oppolzer is reputed to have secured his professorship in Vienna by his diagnosis of aortic leakage from placing his hand on a patient's foot.

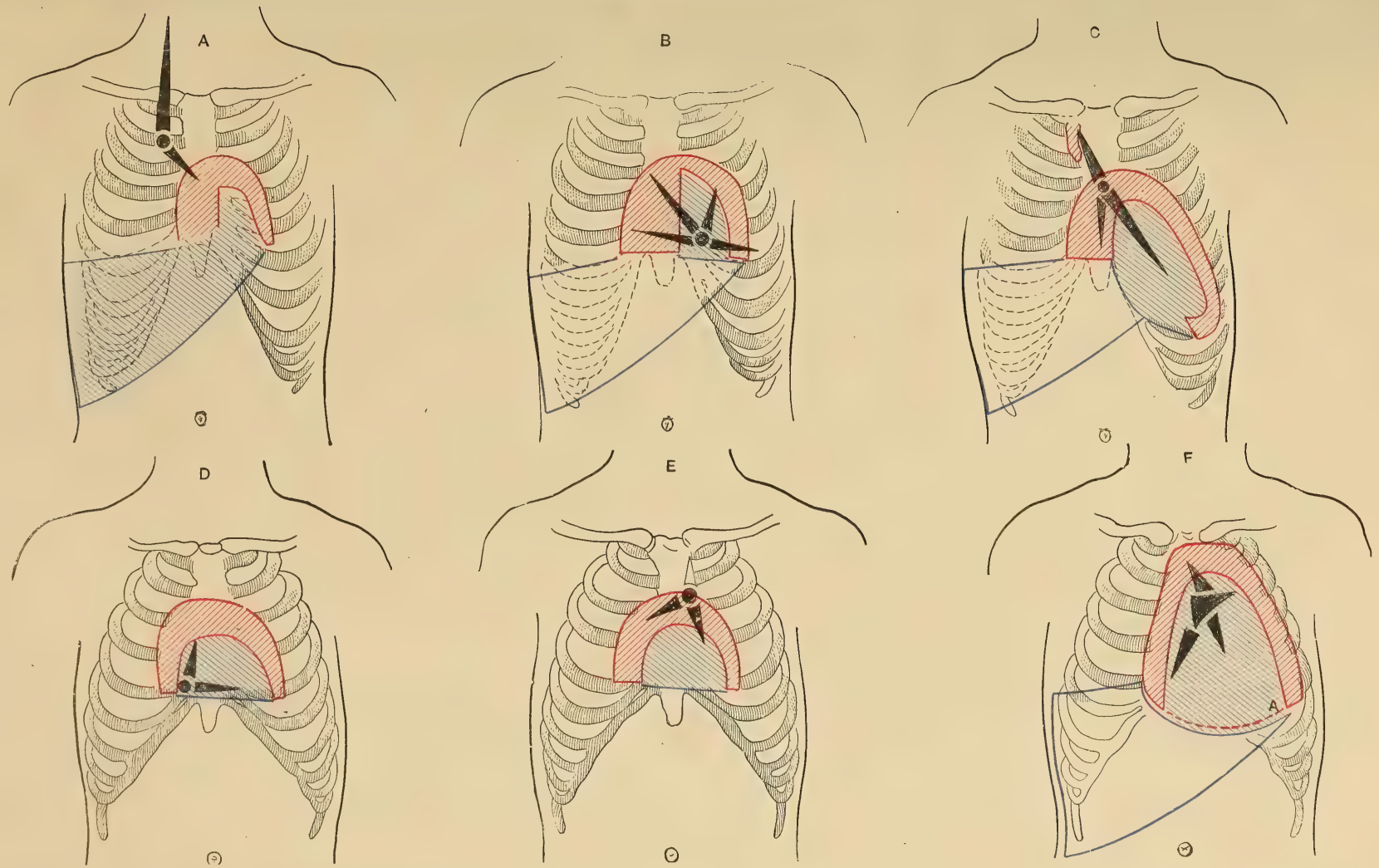
The radial artery is suddenly filled, the "pistol pulse" or *pulsus celer*, due to hypertrophy of the left ventricle. The heart action is quicker, at the expense of the diastole, shortening of which is favorable, since less time is allowed for the leakage. The condition of the heart muscle and pulse depends on the etiology of the case; hypertrophy is marked in the endocarditic form, but in the atheromatous type, coronary atheroma and fibrous myocarditis lessen the myocardial activity, render the pulse atypical and make the prognosis less favorable. The sphygmographic tracing shows the high up-stroke with an acute apex (the *pulsus altus*). The dicrotic wave is ill-marked and the pulse rapidly recedes (the "*collapsing pulse*"), due to the inability of the aortic valves to hold the receding blood. (Stewart's explanation is: a reflex dilatation of the peripheral vessels and heart stimulation by the increased pressure within the left ventricle.) The collapse of the pulse is sometimes increased by

lifting the arm. This pulse is strongly suggestive but also occurs in fevers or anemias. The typical findings are altered by atheroma; other valvular lesions (aortic stenosis or mitral regurgitation); or by heart weakness. When the pulse findings precede the murmur, it is claimed that the lesion is due to disease of the aorta. Thrills may be felt over the peripheral vessels. The *peripheral pulse is delayed* in half of the cases, because the regurgitation continues into the first part of the systole. The systolic blood-pressure is higher, and the diastolic is lower, than normal.

3. *Percussion.*—(*a*) The heart area is increased downward and *to the left;* the *x*-ray shows the heart rather horizontal, "recumbent" or of an "egg form." Dulness reaches the second or third rib to the left of the sternum (see Plate X, Fig. C). The dulness extends slightly beyond the apex and is due to dilatation of the left ventricle; the right heart may be crowded somewhat to the right, although dulness to the right of the sternum is infrequent. The papillary muscles are found flattened and thickened at autopsy, from the pressure of the regurgitant blood. (*b*) A small area of *dulness* in the first and *second right interspaces* denotes mechanical dilatation of the arch, from the cardiac hypertrophy.

4. *Auscultation.*—(*a*) *A diastolic murmur* is heard over or to the left of the sternum at the level of the second or third rib, and increased downward along the sternum, for the regurgitant column follows this direction (Plate X, Fig. C). It has a *blowing, deep* and less often sawing, musical or whistling sound (which latter suggests a relative aortic leakage). When slight, it may easily escape recognition; when well developed it is very characteristic, sounding like the word "*who.*" It may best be heard with the bare ear against the chest. It sometimes disappears before death when the heart is weak and the lesion great, but generally corresponds to the degree of leakage. It rarely reaches the neck, abdomen or axilla. Valvular rupture may produce a murmur which can be heard at a distance. (*b*) *A slight systolic murmur* over the aorta is common, but the too frequent diagnosis of aortic stenosis (*q. v.*) is not justified. It is soft in the atheromatous and often harsh in the endocarditic type. Its various explanations are: rough aorta or valves; physiological stenosis (the rigid valves lying between a wide aorta and a dilated ventricle); vibration of the aorta; or leakage continuing into the beginning of the systole. (*c*) *Weakness or absence of the second aortic tone* is explained by imperfect closure of the valves. As the second tone is normally heard over the carotid artery, it should be listened for there, because it is removed from the murmur and the second pulmonic tone. If it is present, one or more valves are closing, though probably imperfectly. By lifting the stethoscope a little from the skin over the aortic region, the second sound, previously obscured by the bruit, may be heard. (*d*) *The first mitral tone is often weak or lost,* because the papillary muscles are flattened and indurated by the regurgitant column, or a mitral systolic murmur may replace the first tone. (*e*) *An apical presystolic (Flint's) murmur* is due to aortic leakage continued in the systole, or to pressure on the mitral valves, which hinders the flow of blood from the left auricle into the left ventricle (Flint, 1862). Thayer found it in 60 per cent. of 74

PLATE X



Showing the Size and Shape of Heart in the various Valvular Lesions and in Effusive Pericarditis, the Location of the Murmurs and the Direction of their Propagation.

The black dots show the location of the murmurs and the black wedges show the direction of propagation, growing thinner as the murmurs grow fainter. Over the heart, the red area designates the relative and the blue area the absolute cardiac dulness. *A*, aortic stenosis; *B*, mitral lesions; *C*, aortic regurgitation (note dulness over aorta); *D*, tricuspid lesions; *E*, pulmonary lesions (dark wedges designating pulmonary stenosis and insufficiency, and the light wedge, pulmonary stenosis); *F*, effusive pericarditis (pyriform dulness; wedges showing where friction is most often heard; *A*, apex, well within the area of dulness).

cases, and in less than one-quarter of these was there coincident mitral stenosis. (*f*) *Arterial tones and murmurs* result from vibration of the vessel wall; the tones may become murmurs, when there is great difference in tension. The systolic vessel tone is increased by the larger volume of blood and increased ventricular action, and is most plain when the vessels are not sclerotic. The femoral systolic tone is convertible into a murmur on pressure. *Traube's double femoral sounds* consist of two spontaneous tones (produced without pressure), most satisfactorily explained by sudden change in tension. They are heard best when the heart is strong and the vessels soft. They may be confused with the double venous (Friedreich's) tones, heard over the vein in tricuspid leakage. *Duroziez's double murmur* consists of two murmurs, elicited by slight pressure and due to the progress and recession of blood in the artery. These phenomena also occur in other conditions.

Diagnosis.—Physical examination by inspection, palpation, percussion and auscultation, in the order named, prevents error in the interpretation of murmurs. The cardinal findings are: (*a*) dilatation and hypertrophy of the left ventricle; (*b*) the *pulsus celer* and *altus;* (*c*) the arterial phenomena; and (*d*) the diastolic murmur.

Comparing the types, *endocarditic aortic insufficiency* is generally rheumatic and occurs in younger subjects; precordial prominence and diastolic thrill are more common; the heart muscle hypertrophies and the pulsating vessels are more conspicuous; the pulse is more typically "pistol-like" and collapsing, as the vessels are not sclerosed; the capillary pulse and vessel tones are more frequent; on auscultation the diastolic murmur is rather harsher and nearer the valves, Flint's murmur is more common and if a systolic aortic bruit is heard, it is often harsher and coincident aortic stenosis is more frequent.

In the *atheromatous form,* sexual excesses, manual work and alcoholism are more evident. *Syphilis* is declared by the immobile pupil, lightning pains of tabes or Wassermann reaction. As arteriosclerosis readily involves the heart, anginal pains, myocarditis or dyspnea may stand forth. The dilated aorta is more frequently palpable in the jugulum and allows the heart to sag lower in the chest. The diastolic murmur is more characteristically deep and blowing and the vascular phenomena are considerably modified by the rigid arteries.

Relative aortic regurgitation is difficult to differentiate from the valvular; the murmur is a short, presystolic or postdiastolic, almost pathognomonic "huff" after the second aortic tone. The pulse is frequently small, tardy and tense, but not quick. The murmur varies from time to time, sometimes short, then longer, sometimes blowing, and again coarse.

Diastolic murmurs are rarely *anemic* (*cardiopulmonary* or *functional*); such bruits exist, however, in the cava and other veins; they are uneven and stronger in the beginning of the diastole. *Aortic aneurysm* (*q. v.*) may cause confusion. In *mitral lesions,* the capillaries are poorly filled and the skin is cyanotic; in aortic regurgitation the arterioles are overfilled and the skin is pale, perhaps red. In mitral disease, congestion of the lungs, brown induration and dilatation of the right heart obtain;

these are absent in aortic leakage; a hemorrhagic nephritis may take the place of the analogous brown induration of the lungs of mitral lesions.

Relative healing by vicarious stretching of sound valves, by increasing stenosis and by calcification is possible but infrequent.

Aortic Stenosis.—Stenosis of the aortic orifice, a better term than aortic stenosis, is the rarest of left-heart lesions; its frequency as a solitary lesion is 2.5 per cent. It may occur congenitally from constriction of the conus arteriosus (Dittrich's genuine heart stenosis). It occurs, however, largely in the aged; the valves become interadherent or fibrously thickened from atheroma or chronic endocarditic calcification, or retraction at the valvular insertion obstructs the orifice. Acute endocarditis, with massive vegetations, may produce some stenosis. It is usually associated with aortic incompetency.

Mechanism.—The rigid valves narrow the orifice and at the obstruction a systolic murmur originates. The resistance to the onward flow of blood from the left ventricle leads to its hypertrophy. The pulse becomes small and slow because of obstruction.

Physical Signs.—1. *Inspection.*—Some precordial prominence may be seen in yielding chests. The apex beat is sometimes strong and dislocated to the left, but often no apex is seen on account of diminution of the heart's recoil.

Fig. 22.—Pulse tracing in stenosis of the aortic orifice.

2. *Palpation.*—(*a*) A *pronounced systolic thrill* is often felt over the second right interspace, sometimes over the heart and cervical vessels. (*b*) *The pulse* is infrequent (*pulsus rarus*), because the systole is prolonged and the coronary arteries are poorly filled; small (*p. parvus*) from obstruction to its flow; tense (*p. durus*) from hypertrophy of the left ventricle; and slow (*p. tardus*) with tardy up-and-down stroke and some retardation.

3. *Percussion.*—Concentric hypertrophy (with the left ventricle smaller than normal), and simple hypertrophy, which are the more common as the stenosis is purer, give no change on percussion. Slight dilatation to the left is sometimes present, especially with aortic regurgitation, nephritis or atheroma.

4. *Auscultation.*—(*a*) The *systolic murmur* over the aortic area is generally *long, sawing,* whistling or musical and is transmitted with the blood current into the arch, the descending aorta in the back, its vessels (Plate X, Fig. A), or over the entire heart or even at some distance from the chest. It is not proportionate to the intensity of the lesion and disappears with broken compensation. (*b*) *The second aortic sound is weak,* from poor filling of the aorta and weak closure of the altered valves, or absent in coincident aortic regurgitation; (*c*) when the second aortic sound is absent, the *second mitral tone,* or indeed all the heart tones, are *weak or absent.* (*d*) The systolic tone over the aorta is scarcely to be heard.

Diagnosis.—The lesion may be favorable—a healing of an aortic regurgitation. Aortic stenosis is a frequent surprise at autopsy, having run a *latent* course. Diagnosis should be reserved, without these *cardinal signs:* (*a*) *systolic murmur* with propagation into the large vessels or over the entire heart; (*b*) *a systolic frémissement* over the aortic area; (*c*) the small, tense, tardy, *slow pulse, in contrast to the strong apex beat.* As the lesion is uncommon, its frequent diagnosis implies *confusion* with (i) *accidental murmurs* which are variable. (ii) *Acute endocarditic vegetations,* give a soft murmur which becomes harsher if actual stenosis ensues. (iii) *Atheroma of the aorta,* or acute aortitis, where the murmur is more localized over the aorta, the second aortic tone is metallic and the pulse proportionate to the apex beat. Atheroma, the most frequent cause of diagnostic error, is very common, while experienced clinicians see few cases of pure aortic stenosis. (iv) *Aneurysm* (*q. v.*), which is often distinguishable by its pressure symptoms.

Mitral Insufficiency.—Mitral insufficiency (incompetency, regurgitation) is the most frequent valvular lesion and more often endocarditic than atheromatous. It is (*a*) *valvular,* from adhesions between the valves or chordæ and ventricular wall, from vegetations, from thickening with retraction, from valvular aneurysm or perforation, or from shrinkage or rupture of the papillary muscles or chordæ; (*b*) *muscular,* from relative insufficiency due to such ventricular relaxation that the valves do not cover the orifice; this form is common in hypertrophy with dilatation, resulting from hard work, nephritis, syphilis, alcoholism, myocarditis and adherent pericardium; (*c*) rupture of the mitral cusps is very uncommon. Barié collected 35 cases of valvular rupture, of which 45.7 per cent. were aortic, 45.7 per cent. mitral and 8.6 per cent. tricuspid.

Mechanism.—During the systole, a backward flow occurs through the incompetent valves into the left auricle, which, as it also receives blood from the lung veins, first dilates and later hypertrophies as far as its thin musculature admits. The left ventricle dilates because it receives in the diastole the usual inflow of blood plus the regurgitated blood and, responsive to the dilatation, it hypertrophies because its work is increased. In aortic lesions, the first and greatest strain is felt in the left ventricle, but mitral lesions, by congesting the lungs, throw most work on the right heart. The stasis in the left auricle is transmitted to the pulmonary veins, capillaries and arteries, thus leading to increased blood-pressure in the pulmonary circuit, because (*a*) of this backward pressure, and (*b*) of the onward pressure from the right ventricle, which dilates and then hypertrophies. The lungs are enlarged, their vessels dilate and may become atheromatous, possibly causing infarcts in the lungs and fibrosis (brown induration). When the final atonicity of the right ventricle, *i. e.*, broken compensation, occurs, relative tricuspid leakage from dilatation, cyanotic induration of the liver and kidneys, edema, etc., result.

Physical Signs.—1. *Inspection.*—(*a*) The apex may be normally located or *displaced to the left*—even into the axilla, when the heart lies lower than normal. A normally strong apex beat in mitral insufficiency is evidence of *hypertrophy.* (*b*) Systolic *pulsation in the second left inter-*

space indicates a wide pulmonary artery from stasis, and (*c*) *a diastolic shock* there is due to closure of its valves; (*d*) *precordial prominence* occurs in yielding chests from dilatation; (*e*) *pulsation,* either epigastric or to the right of the sternum, results from dilatation and hypertrophy of the right ventricle. (*f*) The *enlarged veins* pulsate during the diastole from stasis, and sometimes during the systole from tricuspid incompetency.

2. *Palpation.*—(*a*) The *apex is dislocated to the left;* (*b*) the *pulmonary artery pulsation* and (*c*) *the diastolic shock* of the forcibly closing *pulmonary valves* are very often palpable; by placing one finger over the valves and another over the apex the two shocks alternate; (*d*) *pulsation* is felt to the *right* of the sternum from right ventricle dilatation; (*e*) a *systolic apical thrill* is often palpable but not pathognomonic. (*f*) In pure cases the *pulse* is about *normal* in *tension,* or weakens with decompensation. Irregularity is not common.

3. *Percussion.*—(*a*) *Dilatation of the right ventricle* is commonly followed by *hypertrophy,* in compensated cases, as the right heart bears the stress of this lesion. Dilatation causes dulness and palpatory resistance to the right of the sternum (see Plate X, Fig. B). The *x*-rays show the typical round heart. Hypertrophy without dilatation is usually found in the early and well-compensated lesions. (*b*) The *left ventricle* usually *dilates* to respond to the increased amount of blood, and *hypertrophies* because of increased work or the raising of arterial pressure by carbon dioxide.

4. *Auscultation.*—(*a*) The *systolic murmur* is due to abnormal eddies in the left auricle or to the vibrations caused by them in the mitral valves; it most often gives a *blowing* sound, best heard *over the apex,* which is less covered by the lung, but at times is localized over the left auricle, where it originates and is heard especially in early lesions. It is *transmitted chiefly* to *the left toward* the *left interscapular* region, and more rarely to the aorta, carotids and abdomen (see Plate X, Fig. B); at times it is heard intermittently, in certain postures only, and it may disappear with weakening of the heart. In very rare cases it is entirely absent. (*b*) The *first mitral tone* is usually absent because the valves cannot vibrate; if present it is referable to conduction from the tricuspid valve or to contraction of the ventricle. When obscured by the murmur, it may sometimes be heard by lifting the stethoscope a little from the chest wall. In extreme leakage, the murmur replaces all tones. (*c*) The *second pulmonic sound* is accentuated, "hammer-like," and is a measure and result of the right ventricular hypertrophy (Skoda). It sometimes reaches the vessels of the neck. When the tricuspid valves become relatively insufficient, it usually weakens. The second tone is sometimes split. (*d*) A *systolic murmur* over the pulmonary area is propagated from the apex or is due to vibrations in the distended pulmonary artery.

Diagnosis.—The cardinal features are (*a*) the systolic murmur, (*b*) accentuated second pulmonic tone and (*c*) right-heart changes.

Excepting the etiological diagnosis and the clinical evolution, there are no positive criteria by which we can differentiate a *muscular* (*relative*) from a *valvular insufficiency,* though in the former the murmur is more

variable, lessens with digitalis and is not associated with a diastolic stenotic murmur, since pure valvular insufficiency is exceptional, and there is usually, anatomically at least, some mitral stenosis. An organic valvular defect is probable when the apex, dislocated to the left, is forcible.

From the *functional* (accidental, hemic or cardiopulmonary) *murmur,* the diagnosis is usually easy. The murmur is inconstant, usually systolic, often basal, often depends on posture, is circumscribed, is not typically transmitted, follows fever, anemia and emphysema, is often associated with the venous hum in the neck, but is never associated with typical mitral changes in the heart tones and right ventricle. The intensity, *timbre* or *frêmissement* of the murmur must not alone be depended upon.

In *chlorosis,* we may obtain a hemic murmur, apparently large heart (because the lungs are poorly expanded), and a displaced apex (high diaphragm); deep breathing decreases the heart's dulness.

Pericardial friction (*q. v.*) may simulate endocardial murmurs.

Fräntzel's rules aid in accuracy: (1) Never make a diagnosis just before death. (2) Remember the great infrequency of right-heart lesions. (3) Avoid complicated diagnoses. (4) Subordinate the murmurs to other physical findings.

Mitral Stenosis.—Anatomically pure stenosis of the mitral orifice, without associated mitral leakage, is rare. In one group, especially in young girls, the stenosis is clearly endocarditic and the valves are irregular, calcareous and deformed. In another group, in old subjects of both sexes, the sclerosing process is probably endocarditic; and in the last group, almost exclusively observed in women, the nature of the lesion is uncertain; some rare cases are associated with congenital defects. The mitral valves are grown together or the ring constricted—Corrigan's "button-hole contraction"; the orifice in extreme cases is funnel-shaped.

Mechanism.—The stenosis obstructs the diastolic discharge of blood from the left auricle, which consequently dilates and later hypertrophies. The enlarged auricle or pulmonary artery may compress the adjacent bronchus or recurrent laryngeal nerve. The stasis is propagated backward through the lungs to the right ventricle, whose dilatation and hypertrophy cause the large right heart and the increased pressure in the lesser circulation. The effects on the right heart and lungs are more severe than in mitral insufficiency. The right auricle also dilates when the later venous stasis follows. Thrombi in either auricle may dislodge and produce pulmonary or arterial infarction. The arterial system is poorly filled.

Physical Signs.—1. *Inspection.*—This reveals (*a*) *precordial distention,* (*b*) *epigastric pulsation* and (*c*) a *diffuse cardiac shock;* (*d*) the *apex beat* may in comparison be *small,* and (*e*) two impulses may be seen in the second left interspace; one is systolic, from systolic filling of the pulmonary artery, and the other is diastolic, from pulmonary valve closure under high tension. All these findings depend on the right-heart hypertrophy and dilatation.

2. *Palpation.*—Palpation confirms the above, especially (*a*) the *apex* beat, which is often *weak in comparison* with the diffuse heart impulse, (*b*) the snap of the *second pulmonic sound,* (*c*) the pulsating right heart, and also (*d*) a *presystolic* (more rarely diastolic) *thrill, frémissement cataire,* which is usually limited to the apex, ends with the *snappy first apex tone,* and is felt sometimes only in the left lateral posture or after exertion. Its mechanism is identical with that of the murmur (*q. v.*). (*e*) The *pulse* is often *irregular* in pure stenosis, because dilatation interferes with cardiac conductivity and is usually *small* and of *low tension,* because the aorta is poorly filled; its weakness contrasts conspicuously with the active heart impulse. It may seem hard. The pulse and sphygmogram are not pathognomonic.

3. *Percussion.*—(*a*) The *right ventricle* in the earlier stages may be only hypertrophied, which does not show on percussion. *Dilatation* without hypertrophy exists in few cases fortunately, for the prognosis is then poor. Most commonly hypertrophy and dilatation coexist, the latter giving increased dulness to the right, as in mitral insufficiency. (*b*) The *left ventricle* in many cases of dominant stenosis shows concentric atrophy, so that at autopsy it looks like a mere appendage to the huge right heart, as shown also in the *x*-ray examination. It is sometimes both hypertrophied and dilated, because of stasis with increase in the blood of carbon dioxide, which raises the blood-pressure; or because of other lesions, as mitral leakage. (*c*) The *auricles* are distended, much of the dulness under or to the right of the sternum being due to the dilated right auricle and that above and to the left, to the left ventricle and widened right conus arteriosus. Increased dulness, due apparently to dilatation of the various chambers is often caused by their dislocation by the wide right ventricle. The left auricle is best determined by radiography.

4. *Auscultation.*—(*a*) An *apical presystolic murmur* occurs at the end of the diastole with the systole of the hypertrophied auricle which forces the blood through the narrowed ring; corresponding with its energy, it is loudest at the incipiency and ending of the auricular systole (crescendo murmur). It so closely precedes the ventricular systole that it is almost invariably called systolic by students. It is usually *rolling,* sounding like r-r-r-, *after which* comes the loud first tone, sounding like p (r-r-r-p). It is poorly propagated as a rule, corresponding to the localized thrill, but may in exceptional cases be heard in the axilla or back. It is often absent even with great stenosis, when the auricle and ventricle contract together and when the heart weakens. It is rarely diastolic (diminuendo murmur). (*b*) *Marked accentuation* of the *second pulmonic sound* is the proof and measure of hypertrophy of the right ventricle from pulmonary congestion. Its absence is a poor prognostic; it usually weakens when relative tricuspid leakage lessens the tension in the lungs. (*c*) *Splitting or reduplication* of the *second pulmonic sound* is due to difference in tension in the two arterial trunks, the aortic valves closing first and more weakly; it is best heard at the base and is often propagated into the neck or axilla. (*d*) The *loud first mitral tone* is explained in various ways; viz., by difference in tension of the

valves during the systole and diastole or by a vigorous contraction of the left ventricle upon little blood. It is sometimes heard at a distance of several feet. (*e*) The *second mitral tone* is weak because of poor filling of the aorta or because the right heart pushes forward; it may be split at the apex (the "postman's knock"); the cause is disputed.

DIAGNOSIS.—The cardial signs are (*a*) the presystolic thrill and bruit; (*b*) the involvement of the right heart; (*c*) the accentuated second pulmonic sound; and (*d*) loud first apical tone.

The thrill and bruit are also found in aortic insufficiency, in which the vascular phenomena and the involvement of the left heart are sufficiently distinctive, though one out of every three cases of aortic leakage is complicated by mitral stenosis. The bruit is also heard in lesions leading to dilatation of the left ventricle, such as adherent pericardium, and in auricular contractions.

Pure mitral stenosis is often overlooked in asthenic women, because the lesion is so benign; characteristic are the positive findings—loud apical tone, murmur and a third tone—and the negative findings—absence of hypertrophy, dilatation and systolic bruit.

An absence of the murmur indicates a slight lesion; a presystolic murmur argues for a strong left auricle; a diastolic murmur indicates a severe stenosis; when tricuspid leakage intervenes, the murmur of mitral stenosis is either absent or diastolic, because both auricles are atonic. Absent murmur with irregularity is indicative of the most serious lesion. When the left auricle is strong, it may suppress the systolic murmur of a coincident mitral leakage; when the auricle weakens, the systolic murmur appears. Auricular fibrillation (page 339) indicates severe disturbance in cardiac function.

DIFFERENTIAL TABLE OF THE COMMON CHRONIC VALVULAR LESIONS.

	Aortic insufficiency.	Aortic stenosis.	Mitral insufficiency.	Mitral stenosis.
ETIOLOGY.	Arteriosclerosis chiefly.	Often arteriosclerosis.	Endocarditis chiefly.	Endocarditis.
PULSE.	Pulsus celer. Water-hammer, collapsing.	Pulse small, slow, tense.	No characteristic alteration—often normal.	Small and often irregular.
LEFT VENTRICLE.	Hypertrophied and dilated greatly; heaving apex.	Hypertrophy usually concentric.	Some hypertrophy and dilatation.	No essential or regular change; may be small.
RIGHT VENTRICLE.	No change.	No change.	Hypertrophy and dilatation marked.	Both marked, also in auricles.
MURMUR.	Diastolic; blowing over sternum near second rib; propagated toward apex.	Harsh systolic murmur over aortic area with systolic thrill; propagated into neck.	Systolic at apex, blowing, transmitted into left axilla and behind scapula.	Presystolic (oftener than diastolic), apical, accompanied by presystolic thrill, fairly localized.
FIRST MITRAL TONE.	Usually weak.	Strong usually.	Usually present, perhaps covered by murmur.	Loud and sharp, clearly palpable and following murmur.
SECOND AORTIC TONE.	Weak or absent.	Weak only.	Somewhat weak.	Somewhat weak.
SECOND PULMONARY TONE.	Not accentuated.	Not accentuated.	Loudly accentuated and often palpable.	Accentuated and often split.
PERIPHERAL VESSELS.	Violent pulsation; capillary pulse; arterial tones; skin red.	Absent.	Absent. Skin cyanotic.	Absent. Skin cyanotic.

In conjunction with this table, Plate X should be consulted.

Pulmonary Insufficiency.—The rare pulmonary insufficiency of which Barié has collected 58 cases, is often combined with pulmonary stenosis or other rare right-heart anomalies. It is (*a*) *congenital,* resulting from fetal endocarditis or from supernumerary or rudimentary valves; (*b*) *acquired,* from atheroma, rheumatism or septic ulceration; or very rarely (*c*) *relative* from extreme intrapulmonary blood-pressure or from embolism of the pulmonary trunk.

Mechanism.—The mechanism is analogous to that of aortic insufficiency except that the right heart suffers instead of the left and the arterial phenomena are manifest in the area of the pulmonary artery.

Physical Signs.—1. *Inspection.*—The dilated and hypertrophied right heart pulsates to the right of the sternum. A systolic, dynamic pulsation may be seen over the dilated pulmonary trunk.

2. *Palpation.*—This determines the diffuse right-heart impulse and a diastolic thrill over the pulmonic area; the apex and radial pulse are normal or weak.

3. *Percussion.*—The *right ventricle* is *dilated* because of the regurgitant column of blood and *hypertrophied* to maintain the onward flow under this burden (Plate X, Fig. E).

4. *Auscultation.*—A *diastolic murmur* is propagated down the sternum over the right ventricle, and may be confused with aortic leakage, aneurysm, functional venous murmurs, congenital anomalies of the vessels or patent foramen ovale. The *second pulmonic* and the second tricuspid tones are *weak* or *replaced* by the murmur. The bruit is but rarely heard in the neck and the normal second arterial tone is heard over the carotids. A systolic murmur is heard in 25 per cent. of cases over the pulmonary trunk, due to vibrations in its walls or to coincident stenosis of the pulmonary ring. A double tone has been heard over the lung, as well as variation in the respiration during the systole—a capillary pulse in the pulmonary vessels.

Pulmonary Stenosis.—The etiology will be considered under diagnosis and under congenital heart lesions.

Mechanism and Physical Signs.—1. *Inspection.*—There is precordial bulging, a diffuse heart shock from hypertrophy and dilatation of the right heart, and a weak or absent apex beat.

2. *Palpation.*—Palpation confirms the above and reveals a systolic thrill in the left second interspace. The pulse is weak.

3. *Percussion.*—Percussion shows an area of increased *dulness* over the *right heart* from dilatation.

4. *Auscultation.*—On auscultation, a loud and whistling or musical *systolic murmur,* caused by the stenosis, is often heard, and the first and second pulmonic tones, hence also the second tricuspid, are weak or absent from poor filling of the pulmonary artery and from valve alteration. (See Plate X, Fig. E.)

Diagnosis.—The more frequent form is (*a*) the *congenital,* due to malformation, myocarditis or endocarditis, and usually associated with open ductus Botalli or foramen ovale. The obstruction may be below the valves, from myocarditic scars narrowing the conus arteriosus, in which case the second pulmonic sound is accentuated; or it may be at

the valves or in the trunk of the artery. Congenital cyanosis and acquired pulmonary tuberculosis are common. (*b*) The *acquired* valvular form is rare. Atheroma or gummata may be causal. The thrill is far less frequently felt in *acccidental murmurs,* which are most common over this area because the thin walls of the artery vibrate easily. These "functional" murmurs are so common as to justify extreme caution in the diagnosis of right-heart lesions in the adult. Functional murmurs may occur in health, fevers, anemia or as the cardiopulmonary murmur. The *organic murmur* rarely reaches the jugular or cervical vessels. The left heart is frequently atrophic. When the acquired stenosis is beyond the valve in the pulmonary trunk or lungs, the murmur is best heard along the right border of the sternum or in the back, and the second pulmonic sound is loudly accentuated, from which points a diagnosis occasionally may be made. In atresia of the orifice 80 per cent. of the subjects die in the first year.

Tricuspid Insufficiency.—ETIOLOGY.—(*a*) The *congenital* form occurs in 0.8 per cent. of valvular cases. The venosity of the blood in the right heart tends to prevent endocarditis, except in fetal life when the blood is arterial. Myocarditis or ulcerative endocarditis may exceptionally occur. (*b*) Of *acquired* forms, the *relative insufficiency* is very common, resulting from (i) valvular lesions of the left heart, especially mitral stenosis; (ii) obstructive lung lesions, as induration or emphysema; (iii) and in severe anemias, in which, however, cyanosis and dyspnea are absent.

MECHANISM.—The high pressure in the lesser circulation leads to dilatation and hypertrophy of the right ventricle. Extreme dilatation stretches the tricuspid orifice so that the valves cannot cover it, and relative leakage occurs. The leak into the right auricle produces its dilatation and hypertrophy, a systolic murmur and a systolic pulse in the veins of the neck and liver.

PHYSICAL SIGNS.—1. *Inspection.*—(*a*) *A systolic pulse in the jugular veins* is due to systolic entrance of blood into the veins from the right ventricle through the insufficient valves; it is also a sign that the auricle and ventricle contract together or that the auricle is paralyzed. It is presystolic-systolic and is known as positive to differentiate it from the negative or diastolic venous pulse which is observed in many other conditions, and either physiologically, as on deep inspiration (damming back of the blood by the auricular systole) or pathologically, as in stasis. It is seen best on the right side because of the more direct vertical connection with the right innominate vein. It may cease when the heart grows weak. Digitalis may cause it to reappear in cases of relative and sometimes of organic leakage. It is best seen in the dorsal decubitus. If the carotid be compressed as low down as possible, or the pulsating jugular vein be compressed at the middle; the upper part will then pulsate if the carotids beat against it (transmitted pulsation) and the lower part will beat if the pulsation is in the vein itself. It is sometimes seen in the inferior vena cava or peripheral veins. (*b*) A *liver pulse,* systolic in time, is venous regurgitant in origin. (*c*) *The right heart pulsates widely,* while the apex beat is weak.

2. *Palpation.*—(*a*) This establishes the systolic phase of the venous pulse, and (*b*) the *systolic venous pulsation of the liver,* which expands in the bimanual examination and is easier to find than the jugular pulsation. It must not be confused with an arterial liver pulse, which occurs

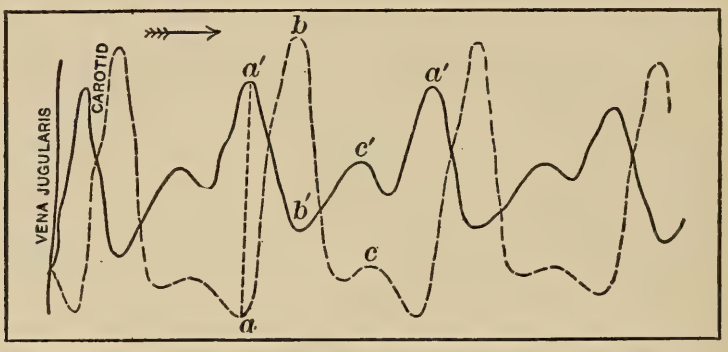


Fig. 23.—Negative (diastolic-presystolic) venous pulse. (After Riegel and Sahli.)

in aortic leakage and exophthalmic goitre, but is less expansile than throbbing. (*c*) A *systolic thrill* over the tricuspid area; (*d*) tones or thrills over the peripheral veins; and (*e*) a weak apex and radial pulse are also found.

Fig. 24.—Positive (presystolic-systolic) venous pulse. (After Riegel.)

3. *Percussion.*—This shows increased dulness to the right, from distention of the right auricle and ventricle. Changes in the left heart may indicate an older valvular lesion which causes the tricuspid leakage. (See Plate X, Fig. D.)

Fig. 25.—Illustrating the influence of respiration on the positive (presystolic-systolic) venous pulse of the jugular vein. (After Kovacs.)

4. *Auscultation.*—(*a*) A faint *systolic murmur* is heard over the tricuspid region; the causal mitral murmur is higher pitched, more superficial and louder and is heard in the back. The murmur is absent when the right heart is so weak that it fails to produce the necessary vibration or eddy. (*b*) The *second pulmonic sound is weak* because the pulmonary

circuit is poorly filled, but varies with the changing activity of the right ventricle. (*c*) Over the *cervical veins* may be heard a systolic tone referable to closure of valves in the veins; the tone may be double, or presystolic-systolic; or venous murmurs may replace the tones.

Diagnosis.—The relative insufficiency is diagnosticated by (*a*) the etiology, (*b*) the large right heart, (*c*) the faint systolic murmur, (*d*) the positive venous pulse in the neck, disappearing under digitalis and rest and (*e*) the weak second pulmonic tone. Rest or digitalis very often brings out the causal mitral lesion. The "safety valve" action of the tricuspid valves is compensatory; guarding the door between the venous and arterial circuits, their insufficiency indicates serious cardiac failure.

Tricuspid Stenosis.—Rarer than tricuspid insufficiency, it occurs alone in only 10 per cent. of the cases; it is more often associated with insufficiency, or with aortic or mitral stenosis; the latter is coincident in half the cases. In half the cases there is a history of rheumatism or chorea; three-quarters occur in women. Obstruction of this orifice induces hypertrophy and dilatation of the right auricle, with dulness to the right of the sternum. In the single lesion the other heart chambers and the large vessels are small. The pulse and second pulmonic sound are weak; a diastolic or presystolic murmur and thrill to the right of the sternum are detected if the heart is sufficiently strong, but they are often absent. The lesion is most serious, because it taxes the weak auricle and greatly engorges the venous system. In the jugular vein there is a presystolic wave. The diagnosis was made in but 10 of the 187 cases on record. (See Plate X, Fig. D.)

Combined Valvular Lesions.—These occur, clinically, in 33 per cent. of valvular cases, but anatomically, a pure lesion is rare, and *double lesions are found oftener than physical signs would indicate.* Single lesions occur most often on the arterial valves. One lesion may follow another; for instance, stenosis may gradually result from insufficiency, sometimes with good results; or combined lesions may develop simultaneously.

(*a*) *Combined mitral* lesions are most frequent, though one lesion usually predominates; (*b*) *combined mitral and aortic lesions, e. g.*, aortic vegetations reaching the nearest mitral cusp, are next in frequency; (*c*) *double aortic lesions* are less common; (*d*) *combined aortic, mitral and tricuspid lesions, e. g.*, stenosis of each orifice with no signs other than slowing of the circulation, occur in 16 per cent., and (*e*) *combined mitral and tricuspid lesions* (especially relative tricuspid insufficiency) occur in 9 per cent.

Diagnosis.—The diagnosis depends upon the character of the pulse, the quality and direction of transmission of the different murmurs, and the hypertrophy and dilatation of the right or left heart. Skoda's rule was that a murmur is usually most intense at its point of origin, except in cases of aortic insufficiency; and Oppolzer held that when listening to the mitral and pulmonary valves, murmurs transmitted to them disappear as examination is made more to the left, while murmurs due to lesions on these valves remain; on listening further to the right, the

true tricuspid and aortic murmurs remain and those which are transmitted disappear. Avoidance of too complicated diagnoses leads to the most practical results.

(*a*) *Aortic insufficiency* and *mitral insufficiency* lead to dilatation and hypertrophy of both ventricles; the combination is frequent in children; the aortic findings as to pulse are frequently modified; the mitral insufficiency may be relative (muscular) or organic (valvular), which is determined only by continued observation. (*b*) *Aortic stenosis and mitral insufficiency* are next in frequency and are the most common combination in adults; less blood reaches the arteries than in simple aortic stenosis; the right heart suffers disproportionately, whence the severity of this complication. The systolic murmurs differ in location, transmission and quality. (*c*) *Aortic insufficiency with mitral stenosis* is a relatively frequent and favorable combination, because the left ventricle is usually less dilated; both may produce presystolic apical murmurs and therefore the state of the right heart and the second pulmonic tone are all-important diagnostically. (*d*) In combined *aortic and mitral stenosis* the greater the aortic stenosis the more is the hypertrophy of the left ventricle; the more dominant the mitral stenosis the less is its hypertrophy and the greater is the dilatation and hypertrophy of the right ventricle.

General Symptoms of Valvular Disease and Decompensation.—The heart possesses not only a certain *reserve power* but in favorable instances its musculature *hypertrophies*. Every hypertrophy is more or less temporary in its compensation and ultimate cardiac failure is inevitable, either temporarily, "disturbed compensation," or permanently, "loss or rupture of compensation," the *asystole* of French writers. Failing cardiac competency is attended by (i) changes in the heart itself and altered movement of the blood; (ii) symptoms in organs remote from the heart, as the congested liver, etc.; and (iii) reflex symptoms, as hyperalgesia over the heart or liver. All lesions tend to reduce arterial tension, increase venous pressure, retard the capillary flow and promote embolism. The blood stagnating in the veins may cause capillary resistance and therefore raise arterial pressure, even though the circulation is embarrassed; in stasis, also, the cyanosis may raise arterial tension, from poor aëration of the blood. Mackenzie maintains that heart failure may develop without fall in arterial pressure, but here there is no loss of tonicity; when the heart becomes atonic (dilated), the blood-pressure falls to rise again when tonicity is restored. Dilatation of the compensatory type appears first and incites hypertrophy; as hypertrophy reaches its limit, the dilatation of the second type appears—the dilatation causing stasis. Circulatory disturbances may be spontaneous or induced by psychical excitement, physical exertion, intercurrent disease, recurrent endocarditis, malnutrition or excessive use of alcohol or tobacco.

1. **Constitutional Symptoms.**—Anemia is frequent (aortic insufficiency); Andral spoke of a "heart cachexia;" polymorphonuclear leukocytosis is present in half the mitral cases; the body temperature may be subnormal from slow circulation. Fever is due to recurrent endocarditis, embolism or intercurrent disease. In tricuspid stenosis the red cells

may be increased (polycythemia). The nails are sometimes clubbed. Skin hemorrhages are due to "blood dissolution" or less frequently to embolism.

2. **Cardiac Signs and Symptoms.**—Apart from actual changes in the chambers, such as dilatation and hypertrophy, the heart itself is not well nourished in "breaking compensation," for it receives less arterial blood and is engorged with venous blood. The importance of the integrity of the heart muscle and its functions is suggested in the introductory paragraph (page 319). Dilatation indicates failure of tonicity; irregularity is disturbance of rhythmicity; and disturbance in contractility has its obvious results. Cardiac insufficiency develops when the reserve force and functions of the heart are prematurely exhausted. The *heart muscle* is in some cases fatty, and in others is the seat of myocarditis about the arteries or cyanotic induration about the veins. These changes are less the cause than the effect of broken compensation. Here and there pressure-atrophy occurs, as in the papillary muscles (aortic regurgitation). The *rhythm* is frequently disturbed, especially in mitral lesions (stenosis). The sphygmogram sometimes shows two ventricular contractions occurring very close together, pulsus bigeminus, of which every second weaker beat is due to an extra-systole (*v.* page 339); when

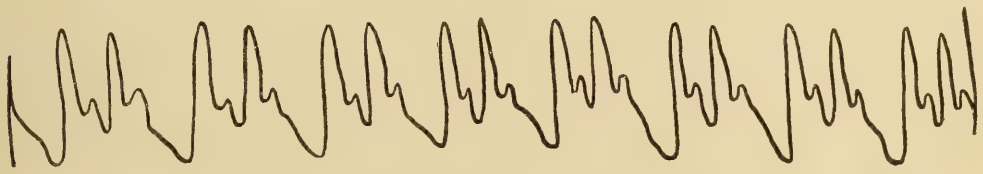


Fig. 26.—Pulsus bigeminus.

the pulse intermits to the finger, the tones of the abortive contraction may be heard over the heart with the stethoscope, the weaker wave failing to reach the periphery. The appearance of the pulsus alternans signifies serious disorder of contractility (*v.* page 340). In general, the pulse beats are fewer than the heart beats. The *gallop-rhythm* is sometimes heard (Bouillaud, 1847), as described on page 321. In great irregularity, as in auricular fibrillation of mitral stenosis (page 339), tones and murmurs are indistinguishable (*delirium cordis*). The pulse is slow (the *bradycardia* of aortic stenosis) or faster than normal, *tachycardia* (mitral lesions); advanced decompensation may be marked by heart-block (page 342).

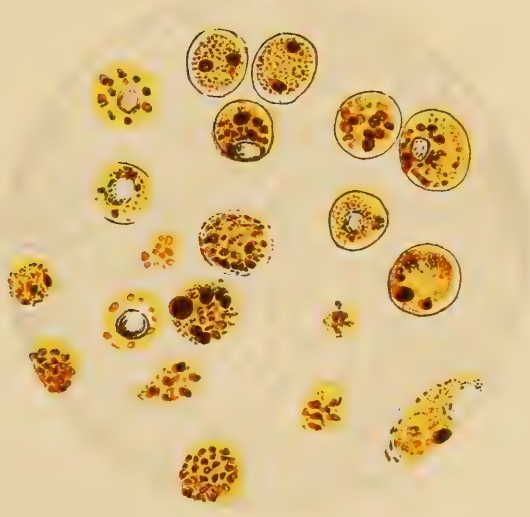
Among the complications are pericarditis (aortic lesions) and recurrent endocarditis. *Palpitation* is frequent, yet it may be absent with the greatest heart alteration. A sense of tension over the heart is not infrequent, and *pain* like angina pectoris occurs in aortic oftener than in mitral disease.

3. **Nervous Symptoms.**—These are not frequent. Syncope (aortic stenosis especially) occurs with arterial anemia of the brain, cerebral arteriosclerosis, and sometimes with extra-systoles. Epileptiform seizures are also due to brain anemia and occur in Adams-Stokes's disease, nodal bradycardia and vagus stand-still of the heart. Venous congestion is

evidenced by epistaxis, vertigo, tinnitus or *muscæ volitantes*. Brain hemorrhage is infrequent (mostly in aortic insufficiency). Chorea and embolism of the Sylvian arteries are sometimes observed. The psychoses are delirium, mania, melancholia and suicidal tendencies, and are usually of unfavorable import; stasis, inanition, carbon dioxide narcosis and renal inadequacy are their cause. *Retinitis* hemorrhagica and simple retinal hemorrhages result from stasis and anemia.

4. **Respiratory Symptoms.**—Lung stasis is sometimes confused with independent lung disease. *Cyanosis* occurs *more often in mitral* than in aortic disease and is most intense in lesions of the pulmonary orifice. *Recurrent laryngeal paralysis*, described by Ortner, is commonly explained by pressure of the dilated left auricle or pulmonary artery. *Bronchitis* and hemoptysis are most common in mitral affections. Bronchitis results less from congestion in the lesser circuit than from congestion in the cava superior, into which (and the azygos) the bronchial veins empty. Though usually bilateral, it may predominate on one side from local causes, as pleural adhesions. *Hemoptysis* results from lung stasis with vessel rupture, from embolism emanating from right-heart clots, thrombosis in the pulmonary and less frequently in the peripheral veins, or from sclerosis of the pulmonary artery. Mitral stenosis may simulate phthisis, by hemoptysis and dulness over and below the clavicle, with rales, due to pressure on the left bronchus by the distended left auricle or pulmonary artery. The *sputum* may resemble that of pneumonia, though it is darker, more venous and less viscid. It contains the "heart-disease cells." They are large granular delicate cells with oval nuclei, which often contain hemosiderin or more rarely hematoidin; they are probably alveolar epithelial cells and also occur in other lesions than brown induration (as emphysema, asthma, pneumonia and phthisis). (See Plate XI.) *Pulmonary edema* (*q. v.*) is (*a*) due to weakening of the left ventricle while the right heart beats more strongly, (*b*) changes in the bloodvessels from stasis or (*c*) far less frequently is inflammatory. Crepitant rales are frequently heard. In mitral disease the *lungs* frequently become *distended* and rigid from engorgement of the capillaries, and closely resemble emphysema if not identical with it (von Basch's *Lungenschwellung und Lungenstarrheit*). *Brown induration* or cyanotic induration of the lungs has for its anatomical basis, (*a*) capillary engorgement, often with fatty change in the intima, (*b*) increased interalveolar and perivascular connective tissue, (*c*) hemorrhage into the lung from vessel rupture, (*d*) consequent pigmentation of the lungs and sputum by the altered blood pigment, and (*e*) the "heart-disease cells." *Dyspnea*, the "cry of distress of the myocardium" (Sansom), is caused by the increased intrapulmonary blood-pressure, the bulging of the turgid capillaries into the alveoli, pressure of the heart, ascites, hydrothorax, edema, bronchitis or infarcts. The intimate mechanism of dyspnea is obscure: the disturbed circulation, poor exchange of the inspired air and lung air; decreased elasticity of the lung; a reflex from the coronaries or aorta which causes spasm of the pulmonary vessels; or sclerosis of the right coronary artery. It may come on without exertion when there is constipation or poor digestion; or it may occur especially at

PLATE XI



Heart Disease Cells, showing Alveolar Epithelial Cells, Loaded Down with Granules of Hematin. (Simon.)

night when the voluntary deep and diaphragmatic respiration of the waking hours is not in play, whence the respiratory centre becomes anemic. Dyspnea may be an air hunger or sense of suffocation; an inability to hold the breath; continued labored breathing; or appear as gasps, in sudden break of compensation. The dyspnea is not expiratory as in asthma but is mixed in type; the breathing is more rapid. The gallop-rhythm is rarely absent, and threatens relapse of the dyspnea; arteriosclerosis and nephritis are important elements in its production. Exertion not only increases the heart's work but the carbon dioxide and fatigue substances from the muscles irritate the respiratory centre, and the respiratory muscles ultimately become tired. *Cheyne-Stokes's* breathing (aortic stenosis) results from cerebral anemia, arteriosclerosis and uremia. *Hydrothorax* results from pressure on the azygos veins or pulmonary veins by the distended auricles.

5. **Gastro-intestinal Changes.**—Gastralgia, anorexia, hemorrhagic infiltration, vomiting or hematemesis, results from passive congestion and may be mistaken for independent gastric disease, such as cancer or ulcer. Delayed digestion and constipation are usual. The secretion of hydrochloric acid is normal except in marked weakness of the heart. Intestinal hemorrhage may result from capillary congestion, rarely from embolism of the mesenteric artery, and most infrequently from mesenteric venous thrombosis (*q. v.*).

6. **Changes in the Liver.**—The liver suffers stasis through the congested cava and hepatic vein; this leads to cyanotic distention of the central veins of the liver lobules (appearing dark) and to cloudy and fatty degeneration of their periphery (appearing lighter), the "nutmeg liver," particularly in mitral disease. Pigmentation occurs and connective tissue develops in the liver and its capsule (perihepatitis, "iced liver"). Small granulations develop on its surface and in some instances a liver shrinkage results from "cyanotic induration" (cardiac cirrhosis). The liver is symmetrically *large* before edema appears; when disturbed compensation is being restored, it is one of the last symptoms to disappear. Occasionally, from local changes, a tumor-like enlargement of the liver may simulate cancer. There is a *feeling of tension* and weight in the right hypochondrium and the organ is *tender* and *variable* in size; it may become smaller from rest and digitalis. Its edge is rounded and not hard except in the late stages. (See DIFFERENTIAL TABLE OF HEPATIC DISEASES.) The *icterus viridis* is a combination of cyanosis and mild icterus of the "urobilin" type; it is rarely sudden except in the infrequent embolism, which may cause a clinical picture resembling acute yellow atrophy. Decompensation is probably augmented by deficient hepatic and renal functions. *Ascites* is usually late and secondary to the liver congestion; its early appearance is indicative of independent liver disease or peritoneal affections.

7. **Changes in the Spleen.**—The spleen is at times swollen from embolism or liver disease, but is often hard and small.

8. **Changes in the Kidneys.**—The kidneys are enlarged from hyperemia and the capsule strips readily. The *stellulæ Verheynii* and glomeruli are prominent. Epithelial degeneration follows malnutrition

and cyanotic induration occurs in cases of long standing. Deformity from the stellate scars of healed embolism, and fresh infarcts are often encountered.

Renal symptoms are greater, the higher the venous and the lower the arterial blood-pressure. The urine is decreased—its daily amount being a gauge of the heart's force; it is of higher specific gravity (1.020) to 1.030), very acid, and highly colored from urates and increased uric acid; the urea is decreased and also the chlorides; albumin is frequent; and red disks are usually due to embolism or acute nephritis.[1] The kidney functional tests give fair results, compared with nephritis. Krehl found granular as well as hyaline casts, contrary to the usual experience. Chronic nephritis may coexist. (See DIFFERENTIAL TABLE OF DIFFUSE RENAL DISEASES.) Rest and digitalis increase the amount of urine and decrease or remove the albumin. In women profuse menstruation frequently results from stasis.

9. **Embolism.**—In about one-third of the cases embolism occurs as in acute endocarditis but its effects are wholly mechanical. Though most common in the kidney (77 per cent.) and spleen (54.7 per cent.), it also occurs in the left Sylvian artery (22.7 per cent.), extremities, retina, liver, aorta (with paraplegia) and mesenteric vessels (in 1 per cent.). In the lungs it results from right-heart clots or thrombosis in the large peripheral or pulmonary veins. The visceral features of embolism will be discussed under Diseases of the Brain, Lung, Kidney, etc.

10. **Edema.**—This results from venous stasis transmitted to the lymph vessels and interspaces, and from changes in the vessel walls from alteration of the blood (capillary dystrophy). It appears first in the connective tissue of the lower extremities (right leg): it disappears over night; and it extends upward to the vulva or scrotum, abdomen, chest and serous sacs (ascites, hydrothorax, hydropericardium and hydrocephalus). The loss of fluid to the system is unfavorable; the anasarca impedes the arterial circulation, threatens life from involvement of the glottis or lungs, increases the tendency to secondary infections and may lead to hypertrophy of the connective tissue about the genitalia—cardiac elephantiasis. It is most common in mitral disease. The prognosis is less favorable if the liver becomes very large before edema appears.

11. **Changes in the Joints.**—Swollen joints and tabes dorsalis (aortic insufficiency) are occasionally associated with valvular disease.

Prognosis of Valvular Disease.—Patients may live for years with compensated valvular affections; a case of aortic leakage lived thirty-eight years and one of mitral leakage lived for sixty-six years. Though prognostic circumspection is necessary, pessimism leads to even more errors. The chronic course is influenced by the following conditions:

1. **State of the Heart Muscle.**—The myocardium is the basis of hypertrophy and compensation. Myocarditis and coronary disease are most unfavorable, for digitalis then has less effect on the heart muscle. In

[1] Bittorf and Kohler demonstrated cells resembling the heart-failure cells of the sputum. In 60 per cent. of patients with chronic passive congestion, "heart-failure cells" are found.

cases where digitalis has immediate effect, the prognosis is favorable. Great dilatation or dilatation without hypertrophy is most unfavorable.

2. **Sex.**—The prognosis is generally better in women, because they are less exposed to infections, physical strain and coronary disease. Pregnancy as a rule is a rather unfavorable complication. Peter advises, "In girls (with valvular lesions), no marriage; in wives, no pregnancy; in mothers, no nursing."

3. **Age.**—In the first decade of life the prognosis is poor, even though the heart and coronary vessels are young, because hypertrophy and dilatation are extreme, exercise is difficult to regulate, the valvular lesion tends to recur and the endocarditis develops into carditis (pancarditis). After puberty the outlook is better.

4. **Valve Involved.**—Opinions vary widely. In aortic insufficiency, the outlook depends on the type, being fair in the endocarditic and ominous in the atheromatous or luetic type. Subjects of mitral insufficiency have the more favorable prospects. Broken compensation is more likely to be restored in mitral insufficiency than in mitral stenosis or in aortic lesions. Marked mitral stenosis is less favorable, although it is often found in women at an advanced age. Aortic stenosis is also found at an advanced age. Combined or multiple lesions, with some exceptions, are unfavorable. The writer observed a complete recovery from a double mitral lesion, after eight years. Pulmonic lesions predispose to early tuberculosis.

5. **Mode of Life.**—The social scale, hygiene, necessity of hard work, psychical excitement, alcoholism, exposure and the individual constitution are most important factors.

6. **Unfavorable Symptoms.**—Angina pectoris, embolism, persistent palpitation, dyspnea and irregular pulse are unfavorable symptoms. The "reserve heart power" of Martius is not present in all cases (failure of the coronary vessels to increase their caliber).

7. **Favorable Conditions.**—These are good general health, proper living and absence of rheumatic tendencies, endocarditic (not atheromatous) origin, sound ventricles and arteries, and freedom from stasis in the lungs, liver and kidneys.

8. **Sudden Death.**—In most cases, sudden death occurs in aortic affections from coronary disease, especially in subjects over thirty with combined aortic and mitral disease, or when valvular disease is associated with emphysema, myocarditis, contracted kidney or aneurysm; it may also result from cerebral or pulmonary embolism, thrombosis cordis, heart rupture, sudden ventricular dilatation and glottis edema.

9. **Intercurrent or Associated Disease.**—Tabes, syphilis and chorea have been mentioned. The acute fevers greatly tax the myocardium. Tuberculosis (*q. v.*) is found in 8 per cent. of autopsies.

Treatment of Valvular Disease.—(*A*) **In Compensated Lesions.**—In compensated lesions medicinal treatment is unnecessary. With some exceptions, the patient is informed of the nature of his trouble that his coöperation may be enlisted. The *mode of life* is important, *i. e.*, regular, orderly living, as free as possible from psychical or sexual excitement, worry or hurry, especially in aortic cases. Much depends on the *social*

status of the patient. The *diet* should be simple but not too schematic. *Tobacco* and *alcohol* should be interdicted as a rule; red wine, brandy or cognac are permissible in some adults and in the aged. Coffee may be given in the morning but should not be strong, for cardiac irregularity frequently results. A breakfast of milk may be taken before rising, and the noon meal should be the heartiest of the day. Nitrogenous food is best, with light carbohydrates, as toast; foods which ferment, such as cabbage, beans or brown bread, should be avoided and the amount of fluid ingested should be moderately restricted. Foods which load the stomach heavily are prone to elevate the diaphragm, and every meal somewhat taxes the heart. A light meal at night is the best precaution against gastric and cardiac oppression during the sleeping hours.

Exercise.—Exercise in cases not too far advanced is advisable, for it reflexly stimulates the heart muscle. Palpitation, recurrent endocarditis and intercurrent fever necessitate keeping the patient in bed. Moderate *massage* and deep breathing are beneficial. Tepid are more beneficial than cold or hot baths.

Climate.—High altitudes are avoided, although moderate elevations are tonic. The seashore often disagrees with rheumatic or catarrhal subjects. A warm climate should be chosen for the winter.

(*B*) **Broken Compensation.**—Broken compensation may be acute, gradual or terminal, as by coronary occlusion, and is evidenced at first by symptoms not necessarily cardiac, as fatigue, vague thoracic or abdominal pain or hyperesthesia, so-called neurasthenic symptoms, inertia, heaviness of the limbs, etc. The greatest therapeutic success follows the recognition of these extracardial evidences, not awaiting the severer signs of stasis, *e. g.*, dyspnea, irregular pulse and dilatation. The indications are as follows:

1. Absolute Rest in Bed.—Liebermeister's case of severe cardiac disease which recovered while in bed with a broken leg, is an example of compensation restored without digitalis. Rest is imperative in intercurrent fevers, bronchitis, recurrent endocarditis and cardiac insufficiency. Bed-sores can be prevented by water-cushions and cleanliness. Some patients are obliged to sit up because of dyspnea. The head may be propped up by back-rests, but they should have side supports so that falling to sleep will not waken the patient.

2. Diet.—Karell's treatment is a low calorie diet—a glass of milk every four hours; its effects are often striking in dyspnea and it rests the tissues, reduces the fluids ingested, is poor in salt and lessens alimentary intoxication.

3. Saline Purgation.—Epsom or Glauber salts ℥ ss–j in concentrated form should be given in the morning or compound jalap powder ℥ ss–j at night. These remedies deplete the portal circulation and relieve the heart. Constipation increases the size of the left heart.

4. Venesection.—When indicated by extreme venous engorgement evidenced by cyanosis, orthopnea, dilatation or tricuspid leakage, one pint may be drawn. The effects are often transient but are sometimes wonderful. Venesection raises low blood-pressure by relieving the heart, and lowers high blood-pressure due to excess of CO_2 in the blood.

In two cases apparently moribund, no blood could be obtained from the arms and the external jugular veins were opened; the patients lived more than a year afterward. Venesection is not to be left until the patient is *in extremis.*

5. The Oertel and Schott Methods.—(See page 323.)

6. Digitalis.—Digitalis was first recommended by Wm. Withering (1775) and its physiological action demonstrated by Traube. Digitalis attaches itself to the heart muscle, and becoming fixed in the tissue, it is slowly given off and eliminated. Operating on the myocardium, vagus, vasomotors and other muscular tissue, it aids the heart to elicit and coördinate all its latent power, but the drug cannot augment the absolute power of the heart.

Its *general indications* are: (*a*) *Dilatation;* digitalis *restores tonicity,* to the heart muscle upon which the drug exercises its main influence, *i. e.,* it overcomes dilatation. (*b*) *Irregularity,* or functional dissociation, is often corrected by digitalis; it stimulates the function "rhythmicity" by action on the heart muscle, the ganglia and the peripheral filaments of the vagus. In about 80 per cent. of patients helped by digitalis, there is continuous irregularity due to auricular fibrillation (Mackenzie); in this condition, the auricles contract rapidly and disorderly and convey impulses to the ventricles more rapidly than normal; digitalis regulates by decreasing conduction through the bundle of His, thereby protecting the ventricles from undue stimulation from the auricles (page 339). Digitalis does not help extra-systoles. (*c*) *Rapid, weak, heart action:* digitalis slows the heart by action on the vagus. It tends to abolish abortive cardiac contractions and synergizes the systole of the chambers (increased contractility), as is shown by the strong systolic spasm of the heart in cases of digitalis poisoning. The slower and stronger systole empties the heart chambers more completely, forces the venous and arterial blood into the main arteries, and squeezes the venous blood out of the heart muscle itself, thereby improving the intrinsic circulation of the heart. The slower, more complete diastole allows better filling of the heart and drainage of the engorged veins. Digitalis raises the arterial tension by increasing the working power of the heart and by its centric and local action on the vasomotors; often arterial pressure is not raised and then digitalis probably equalizes the distribution of the blood and lessens tension due to edema and poor aëration of the blood. The most marked vasoconstriction is in the splanchnic area.

It is seen then that digitalis increases tonicity, rhythmicity and contractility, while lessening excitability and conductivity. All in all, aside from details, *digitalis is indicated for cardiac insufficiency of whatsoever type;* which fact must be kept in view, when we consider its contra-indications.

In the individual lesions: (*a*) in *mitral stenosis* the longer diastole drains the left auricle and lung vessels and the stronger systole forces the retarded blood through the stenotic orifice; particularly in this lesion auricular fibrillation arises; (*b*) in *mitral insufficiency* the best results are obtained; the leaking cusps are better coapted and the stronger systole fills the aorta and pulmonary artery; (*c*) in *aortic*

stenosis, when dilatation occurs or the hypertrophy is inadequate, digitalis is valuable; (*d*) in *aortic regurgitation* it is theoretically contra-indicated, since it increases the diastole and hence the leakage; it is, however, as valuable in aortic decompensation as in mitral.

Digitalis is *contra-indicated* (*a*) in balanced compensation; (*b*) when the vessels are fragile, as in aneurysm, and advanced atheroma on account of the risk of brain hemorrhage; (*c*) in fatty degeneration of the heart, except when resulting from stasis and malnutrition; (*d*) in vascular contraction. In this last instance, it is combined with nitroglycerin, strophanthus or potassium iodide, to "unlock the arteries." The drug is not absolutely contra-indicated in the high tension of arteriosclerosis and nephritis, for paradoxical as this may seem it may relieve by lessening dyspnea and carbon dioxide narcosis and equalizing the circulation. (*e*) Of late, irregularity of the heart is considered a contra-indication, especially by Mackenzie, who stops digitalis as soon as irregularity develops. In partial heart-block digitalis may work harm; it may prove beneficial in some cases of complete heart-block. Mackenzie discontinues digitalis in severe cases of mitral stenosis, where the presystolic murmur disappears or when systolic waves appear in the jugulars, for then some heart-block has developed. Extra-systole is also a contra-indication. (*f*) Idiosyncrasy: individuals react differently to the drug, some exhibiting hypersensitiveness (*v. i.*, toxic symptoms). (*g*) As Janeway pertinently remarks, digitalis is indicated only in ventricular weakness and not in paroxysmal tachycardia, nephritic edema, inflammatory effusions or palpitation. (*h*) Digitalis may fail to operate unless rest, free purgation, venesection and mechanical removal of the anasarca have been first employed. (The Leipzig School uses digitalis on the first sign of decompensation.) Many of these contra-indications are relative only, since digitalis is indicated by cardiac insufficiency of any of the five types of Sahli (cardiac, pulmonary, high-pressure stasis, vasomotor or splanchnic insufficiency); it may be tested out, then, in decompensation due to acute endocarditis (where experimental evidence favors its use), chronic myocarditis, arteriosclerosis, hypertension, coronary disease, tachycardia, etc.

Toxic Manifestations.—Gastro-intestinal symptoms are generally from stasis; sometimes vomiting is due to rejection of the drug by a disordered stomach, or later, to toxic irritation of the stomach or the medulla, by the absorbed drug; there may be diarrhea, with green stools. Digitalis causes contraction of all unstriped muscular tissue (gastric, intestinal, vascular). It may cause delirium, headache, diplopia, blindness to green colors, blueness of the scleræ, exophthalmos, precordial oppression, palpitation, extreme weakness, pallor, renal suppression, violent cardiac overaction, or convulsions.

Cumulative action may attend the use of any efficient preparation, since the heart never becomes anesthetic to digitalis; digitalis preparations, without cumulative action, are inert. It is averted (*a*) by intermittent administration, although some patients tolerate digitalis continuously for months or years, as Thomeyer's patient who in eight and a half years, took over 15 ounces of pure digitalis; (*b*) by great care

in ambulatory cases; (*c*) by care in cardiac dropsy, on relief of which toxic quantities may be resorbed from the tissues; (*d*) by care in intercurrent febrile affections, for toxic effects often follow the fall of temperature; toxemia considerably neutralizes the slowing action of the vagus; (*e*) by withdrawal on the appearance of vomiting, syncope, reduced urine or irregular or very slow pulse, a sign of danger; here atropine may be advantageously combined with the digitalis.

Preparations and Administration.—Among the standardized official preparations, the powdered leaves stand first; the powder deteriorates little, even after eight years and is given in doses of gr. j–iss. The tincture (℥x) deteriorates but much less than the infusion (℥j–ij). For hypodermic use, the fluidextract (℥ij), well diluted, is least irritating. Beginning with small doses of digitalis to avoid sudden overtaxing of the heart, the dosage is increased; Withering, who discovered digitalis, pushed the drug until it acted on the pulse, kidneys, stomach or bowels, etc., its effects appearing in two or three days.

℞—Infusi digitalis . . . . . ℥iij
Spts. ætheris nitrosi . . . . . ℥j
Potas. bicarbonatis . . . . . ℥ss
Aquæ . . . . . ℥ij
M. et S.—One tablespoonful after meals.

The infusion is excellent for rectal use when the stomach is irritable. Regarding the active principles there is the greatest confusion. The *tincture* is thought to be the best cardiant because it contains digitalin and digitoxin. The writer has found digitalin almost inert. *Digitoxin* in the author's hands has often strengthened the weak and irregular heart when digitalis has failed (gr. $\frac{1}{400}$ t. i. d.); it easily disorders the stomach, and often cannot be given for more than six or eight doses. Fränkel finds the therapeutic and toxic doses very similar and fears its use. Digitoxin is often advantageously combined with champagne. Cloetta's *digalen* is a soluble non-irritant digitoxin, and may be given intravenously, 1 c.c. equalling 0.3 mg., or $\frac{1}{180}$ grain. The preparation acts promptly, though it is somewhat irritating when given hypodermically. Cumulative effects are least likely when the kidneys are intact. *Digipuratum* is an active purified product, from which is eliminated the digitonin—the element irritating the stomach and intestines; it is given in powder or tablet form (0.1 gm., grains iss) every twelve hours, or as a fluid in sterilized tubes for emergency intravenous use. Rapid absorption, lack of alimentary irritation, and free diuresis are claimed for it. Like digalen and digitalone it is expensive.

Combinations are suggested in the following prescriptions (*v. i.*):

7. Other Cardiants.—(*a*) *Strophanthus,* ℥v–x of the tincture every eight hours, may be given. Advantages: it is more rapid in action than digitalis, perpetuates the effects of digitalis and is indicated in the irregular heart of mitral disease; in children under twelve years; in cases where we are waiting for the digitalis to act; and in dilatation where the blood-pressure is high. Disadvantages: it is less certain and less energetic; it is inferior in edema and does not give tone to the vessels.

The tincture deteriorates rapidly in dilution or aqueous compounds. Of late *strophanthin*, gr. $\frac{1}{60}$ once daily, has been recommended for intravenous administration. It is said to be rapid in action and efficient. Its therapeutic dose and toxic dose are not far apart and it is inferior to digitalis. It must be used with caution in nephritis, high arterial pressure and bradycardia. Its toxic symptoms are those of digitalis poisoning, including irregularity, partial heart-block, vomiting, etc. It is eliminated by the bowel. It may not be used if digitalis has been recently employed. (*b*) *Strychnine* stimulates the vagus, vasomotors and the nervous, gastric and blood-making tissues (*v.* page 76). Given carelessly, it may irritate the heart, decrease the urine by spasm of the renal vessels, or even induce rupture of the vessels. (*c*) *Sparteinæ sulphas* is given in capsule because of its bitterness (gr. $\frac{1}{4}$–$\frac{1}{2}$ every eight hours); larger doses may be given, gr. j–iss; it seems also to quiet the nervous system but is inferior to digitalis. It does not contract the vessels.

8. Symptomatic Treatment.—(*a*) *Dropsy.*—Saline or hydragogue *catharsis* often relieves moderate edema. Digitalis is often combined with calomel, or blue mass and squills (Guy's pill, also known as Addison's or Niemeyer's).

℞—Digitalis . . . . . gr. x
Massæ hydrargyri . . . . . gr. vj
Scillæ . . . . . gr. x
M. et ft. capsulæ x.
S.—One after meals for one or two days.

Mercurials must be used with the greatest care. The author saw, in consultation, a case complicated by nephritis in which the tongue sloughed off after 5 grains of calomel had been given. Desperate stomatitis may result in alcoholic, myocarditic or nephritic subjects. Calomel acts by its conversion into bichloride, which often incites a bichloride nephritis. In isolated luetic aortic leakage, mercurials may prove specific. Excess of digitalis is held to suppress the urine by inducing renal spasm. It is better to use potassium salts, as potassium acetate gr. xxx, t. i. d., which are less abundant in the tissues than the sodium salts. Diuresis following digitalis is associated with increase of sodium chloride elimination; restriction of salt has few benefits. *Caffeine* with digitalis is beneficial in dropsy when the pulse is slow, the mind dull and the digestion sluggish; anginoid pains are often relieved by it; it may cause nervousness or insomnia (therefore given early in the day), irregular pulse (therefore combined with rum or brandy), or biliousness; gr. v should be given, but not for more than three days—

℞—Caffeinæ citratæ . . . . . ʒss
Sodii benzoatis . . . . . ʒss
Aquæ . . . . . ʒiij
M. et S.—One hypodermicful (ʒss) as indicated.

Caffeine stimulates the bulbar vasomotor centre, and also the peripheral vasomotor system, whereby some elevation of arterial pressure is accomplished; it also acts as a direct renal stimulant; and in shock

and collapse surpasses strychnine and camphor (Rhomberg). *Diuretin*, sodiosalicylate of theobromine (ʒj–iss daily) may act marvellously or not at all, operating on the renal cells directly, and possibly also on the heart and vessels. Theocin sodium acetate is easily soluble in water, and in doses of gr. iij may act when others of this type fail. *Canadian hemp* (fluidextr. apocyni ♏xv) is hard to obtain pure but sometimes justifies Benjamin Rush's term, "a vegetable trochar." *Sweats* and the use of pilocarpine are both dangerous and injurious. Mechanical drainage is recommended, by scarification, by multiple knife or pin-pricks, or by Southey's capillary tubes; but the best method is by a single, long, *deep incision* down to the bone, by which a quart to a gallon of fluid seeps away in a day (and up to 40 gm. of albumin). Ascites, from heart disease alone, rarely necessitates puncture; it is indicated chiefly in coincident liver cirrhosis or chronic peritonitis. Hydrothorax is far more important, and thoracocentesis should not be delayed when dyspnea is urgent.

(*b*) *Anemia.*—Digitalis with iron and arsenic, or the latter two drugs alone, often produce remarkable improvement.

℞—Digitalis . . . . . gr. xx
Ferri reducti . . . . . ʒss
Arseni trioxidi . . . . . gr. ss
M. et ft. pilulæ, xx.
S.—One pill after meals; take for a week only.

(*c*) *Insomnia.*—It is often difficult to relieve insomnia or unrestful sleep, broken by nightmare or sudden "heart starts." Spts. chloroformi, spts. camphoræ and spts. etheris compos. āā ʒss are often helpful. Sulphonal, gr. xx in hot milk at bed-time, may be tried but is somewhat dangerous in stasis, when used more than three nights in succession. Chloral hydrate is excellent in high tension. In morphine we place our chief reliance.

(*d*) *Dyspnea.*—The ice-bag, chloral, and spiritus glycerylis nitratis ♏j are of value for high tension. *Morphine* may be given in the early stages without hesitation and especially at night, for restlessness and dyspnea; it is excellent while waiting for digitalis to act. It is almost specific and lessens the irritability of the respiratory centre. Heroine hydrochloride (gr. $\frac{1}{12}$–$\frac{1}{8}$) operates similarly, but in some instances apparently it can be used longer than morphine. Opiates require care in weak heart or Cheyne-Stokes's breathing. Dyspnea is often renal or due to right-sided hydrothorax, chronic bronchitis or excessive dilatation, when the appropriate treatment is catharsis, paracentesis, potassium iodide or digitalis, respectively.

(*e*) *Respiratory Symptoms.*—Cough, cyanosis, pulmonary edema and hemoptysis are usually cardiac symptoms (pulmonary engorgement or brown induration) and are relieved by the cardiants.

℞—Ammonii carbonatis . . . . . ʒj
Tr. hyoscyami . . . . . ʒiv
Potassii iodidi . . . . . ʒj
Tr. digitalis . . . . . ʒj
Infusi calumbæ . . . . . q. s. ad. ʒvj
M. et S.—One teaspoonful every four hours.

Expectorants usually nauseate. Hemoptysis is often beneficial, being a species of lung venesection and is seldom urgent or fatal. Pulmonary edema necessitates phlebotomy, a 30-grain dose of lead acetate, gr. $\frac{1}{100}$ of nitroglycerin and gr. $\frac{1}{4}$ of morphine.

(*f*) *Palpitation and Pain.*—For palpitation and throbbing, the ice-bag is the best calmative; a precordial blister or a few minims of tr. belladonnæ and tr. aconiti are often helpful, with morphine, if necessary.

℞—Fluidextracti belladonnæ . . . . . gtt. xv
Tr. digitalis . . . . . ℥j
Aq. laurocerasi . . . . . q. s. ad. ℥j
M. et S.—One teaspoonful two or three times daily.

A light evening meal and laxatives are helpful. Iodide of potash and nitroglycerin relieve the pain, weakness, pallor or headache of aortic lesions.

(*g*) *Gastric Symptoms.*—These are ominous when the liver is enlarged and there is not much edema. They are due to portal stasis more often than to digitalis, which should then be given by rectum. Phenol, gr. $\frac{1}{4}$ every half-hour for five doses, and rectal nourishment are indicated.

CONGENITAL HEART DISEASE.

These infrequent cases are complicated and occur largely in the right heart, upon which more work devolves in fetal life. The causes are (*a*) *developmental errors,* by far the most frequent cause; (*b*) *fetal endocarditis,* nearly always sclerotic; (*c*) *fetal myocarditis* or (*d*) *adult endocarditis developing upon developmental anomalies.* They occur largely in males.

1. **Pulmonary Stenosis.**—This, by far the most frequent congenital lesion, occurs in various forms: (*a*) Stenosis or actual atresia of the orifice itself; the second pulmonic tone is weak or absent; (*b*) Constriction of the *conus arteriosus* which obstructs the flow of blood, and may make, as it were, a second ventricle; the second pulmonic sound is clearly heard. (*c*) The pulmonary artery beyond the valves is narrowed or atresic—a grave type. The second pulmonic sound is usually heard.

The physical signs are hypertrophy and dilatation of the right ventricle, a thrill over the pulmonic area, and a systolic murmur propagated into the neck when there is a defect in the interventricular septum (when a defect is lacking, the right ventricle is atrophic and the subject rarely lives over a year). Other congenital abnormalities are common: If the stenosis arises before the end of the second fetal month, the interventricular septum fails to close in 75 per cent. of cases and is pushed to the left, so that the aorta may partly or entirely originate in the right ventricle. If the closure develops after the second month, the foramen ovale remains patent (6 per cent.). Upon these openings acute endocarditis is prone to develop. The ductus Botalli usually remains patent, and conducts blood from the aorta to the pulmonary artery and lungs. Should the ductus Botalli and the pulmonary orifice be closed, collateral circulation is possible through the esophageal, pericardial and bronchial arteries. The right ventricle usually hypertrophies and the left atrophies.

2. **Defects of the Interauricular Septum.**—The foramen ovale is open to some extent in 44 per cent. of all autopsies and in 33 per cent. of congenital heart lesions; it usually closes in the first week of life, but fails to adhere when low pressure in the left auricle exists. Entire absence of the septum, the most extreme defect, is called the "*reptilian heart*," or *cor biloculare* when the interventricular septum is also absent or the *cor triloculare* when the ventricular septum is present. Its patency is almost a necessity when the valvular orifices are narrowed or closed or when the great vessels are transposed. Several cases have been found in subjects over seventy years of age. Symptoms are absent or are those of the more important associated trouble. Rare occurrences are presystolic murmur at the level of the third or fourth ribs; "crossed or paradoxical embolism," *e. g.*, in the brain from a clot in the leg, passing directly from the right to the left auricle; and positive venous pulse in mitral regurgitation.

3. **Defects of the Interventricular Septum** (*Maladie de Roger*, 1879). They are observed in 37 per cent. of congenital lesions and may be single, associated with pulmonary stenosis, or result from myocarditis. The deficit occurs oftenest in the upper "undefended" part of the septum. When wholly absent, the heart has but three chambers (*cor triloculare biatriatum*). Symptoms are absent, complicated or ambiguous. At times the signs are those of mitral leakage without pulmonary stasis. A loud, prolonged systolic murmur and thrill are found over the upper third of the precordium.

4. **Patency of the Ductus Botalli.**—The duct usually closes within the first month, because the pulmonary blood-pressure is lowered after birth. Patency occurs in 26 per cent. of congenital heart lesions and is favored by other congenital lesions or by atelectasis or pneumonia in the newborn. It leads to increased blood tension in the lung. The diagnosis has been made in 50 cases. The second pulmonic sound is accentuated, and the artery is frequently dilated, whence the dulness in the second left interspace, with thrill, pulsation and long vibratory systolic murmur which may reach the cervical vessels or the fourth dorsal vertebra behind. Sometimes the vessel compresses the recurrent laryngeal nerve. The *x*-rays show the large artery and the wide right heart ("round mitral heart"). It is distinguished from pulmonary stenosis by the greater cyanosis of the latter lesion, the less frequent propagation to the neck and the absence of the second pulmonic sound.

5. **Persistent Isthmus Aortæ.**—The fetal *narrowing*, described by Paris (1789), is located in 46 per cent. below the duct, 37 per cent. at the duct, and 17 per cent. over it, and is characterized (*a*) by hypertrophy of the left ventricle (50 per cent.) to overcome the stenosis, and sometimes by a systolic murmur in the interscapular region; (*b*) by largeness and fulness of the aorta above the isthmus and of the arteries of the head, neck and arms (25 per cent.); (*c*) by a small, retarded, abdominal and femoral pulse (below the isthmus); (*d*) by a pronounced collateral circulation between the branches of the aortic arch and those of the thoracic and abdominal aorta (internal mammary, inferior thyroid and transversalis colli arteries). Maud Abbott finds 198 cases on record.

The pathologist makes the diagnosis, as he cuts through the large vessels in the chest and abdomen, and the clinician suspects the lesion when he feels these thrilling large vessels or hears the murmur. Broken compensation may intervene, though latency is common. Aneurysm and mediastinal tumor are frequently confused with it. The aorta ruptures in 12 per cent. of cases. The lesion is twice as common in the newborn as in adults.

6. **Aortic Atresia or Stenosis.**—Moon (1912) collected 129 cases of congenital atresia. If the lesion is developmental, the interventricular and -auricular septa are open; if endocarditic or myocarditic, they are closed, provided the lesion develops after the third month of fetal life. The left ventricle hypertrophies in stenosis or atrophies in atresia, while the right ventricle hypertrophies in order to force the blood from the pulmonary artery through the ductus Botalli into the aorta.

7. **Tricuspid Stenosis or Atresia.**—In developmental or inflammatory atresia, both septa are open and the right heart is atrophic. Tricuspid insufficiency is most rare.

8. **Transposition of the Arteries and Veins.**—In this lesion the foramen ovale remains open; the aorta originates in the right heart.

9. **Valvular Anomalies.**—The semilunar valves, especially the pulmonary, may be increased in number from three to five; or they may be decreased to two, which occurs especially in the aortic valves. Accessory or abnormal valves are particularly prone to endocarditis. Mitral anomalies are rare.

10. **Anomalies in Location and Development.**—These are absence of the heart (*acardia*), double heart, or ectopia cordis, where the heart lies in the abdomen, or chest (with fissured sternum).

Dextrocardia (dexiocardia), described by Servius (1643), occurs with *situs viscerum inversus;* the mitral valve has three cusps, the tricuspid has but two; the pulmonary veins empty into the right auricle; the pulmonary artery arises from the left, and the aorta from the right, ventricle; the aorta runs to the right of the spine and esophagus and to the left of the cava; the innominate artery and vena azygos are on the left and hemiazygos is on the right; the right lung has two lobes, the left three; the bronchial fremitus is greater on the left side, the left bronchus being larger; the spleen, liver, stomach and colon are transposed; the right kidney and testicle are higher than the left. The heart is rarely transposed alone. Pollock and Jewell (1909) collected 323 cases.

Symptoms of Congenital Heart Disease.—Intense *cyanosis* develops chiefly in transposition of the great vessels, pulmonary stenosis with interventricular defects and in pulmonary and tricuspid atresia. *Morbus cæruleus* is general or limited to the nose, fingers, etc.; the purple skin is due to mixing of the arterial and venous currents, to sluggish circulation or to impaired function of the red cells. Sometimes the cyanosis is paroxysmal. A most striking increase of the red cells up to 10 or 12 millions (polycythemia rubra) is seen in some cases, probably as a compensatory effort. The hemoglobin may register 230. Dyspnea, cough, convulsions and marked clubbing of the fingers and toes are frequent.

The temperature is often subnormal. Mental and physical development is incomplete and exophthalmos, swelling of the face, lips and nose and of the optic disk are frequent. Edema and stasis are not frequent. Coincident maldevelopment occurs in 10 per cent. of cases in the stomach, colon, kidney, etc.; imperforate anus, hypospadias, cryptorchismus, polydactylia, hare-lip, spina bifida and anencephalus also occur. The murmurs are usually systolic and rough, although very complicated lesions may exist with normal heart tones; in a few instances the fetal murmur was heard before birth. The dulness concerns the right ventricle chiefly.

Prognosis.—The lesion is incurable and compensation is incomplete. The patient in 25 to 40 per cent. of cases succumbs to cardiac failure or to pulmonary tuberculosis in the second decade (81 per cent.), hence Rokitansky's rule, that cyanosis protects the lungs from tuberculosis, is not absolute.

Treatment.—The treatment is symptomatic. The body should be kept warm; baths should be employed to guard against colds; mental and bodily strain should be avoided; and in general the therapy of adult valvular disease should be followed, except that the heart stimulants must be used with considerable caution.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the pericardium. Known anatomically to Morgagni, it was first described clinically by Senac (1749).

Etiology.—The so-called primary cases are tuberculous, rheumatic or septic. Cold, exposure and trauma only reduce the physiological resistance to bacterial invasion. Pericarditis is mycotic, the organisms of suppuration, pneumonia and tuberculosis being most frequently found; perhaps toxins alone may excite inflammation.

Pericarditis is *secondary* (*a*) to *infectious diseases*, of which rheumatism (40 per cent.) is foremost, particularly when many joints are involved in rapid succession, usually developing within half a week to two weeks. It may be the only manifestation of rheumatism. It occurs with recurrent rheumatism, endocarditis and sometimes chorea. It is commonly serofibrinous.

It is common in pneumonia, septicopyemia, puerperal fever, ulcerative endocarditis, osteomyelitis, scarlatina—more rarely in other exanthemata—and in pulmonary or multiple serous tuberculosis. In children scarlatina and rheumatism are most important.

(*b*) It is secondary, *by contiguity*, to disease of adjacent organs; to pleurisy, pneumonia and aneurysm; to disease of the ribs, myocardium and endocardium, sternum, bronchial glands, spine, esophagus, stomach, spleen, liver, peritoneum; and to cervical cellulitis.

(*c*) It is secondary to *certain diseases of the pericardium*, as tuberculosis, carcinoma and other processes. In 30 per cent. of Matter's "cryptogenetic" cases, tuberculosis was found at autopsy. Scaglios (1904) could find but 8 cases of primary pericardial tuberculosis.

(*d*) It is *secondary to cachexiæ or dyscrasiæ, e. g.*, nephritis (especially contracted kidney), gout, diabetes, carcinoma, alcoholism, blood diseases, etc.; and often as a terminal infection; this chiefly adult type easily escapes clinical recognition. Pericarditis is most frequent between the fifteenth and thirtieth years.

Fibrinous (Plastic) Pericarditis.—A strict division into dry (plastic) and exudative (effusive) pericarditis is impossible, because these forms are usually combined. Dry (plastic, fibrinous) pericarditis is *circumscribed* over the base and large vessels, or is *diffuse*, and consists pathologically of vascular injection, punctate ecchymoses, loss of the smooth, glistening appearance of the serous membrane, and exudation of plastic lymph in small particles or irregular lamellæ, which may appear smeared as with butter, spongy, shaggy or hairy (*cor villosum*). Some fluid is found in the meshes of fibrin. Stripping off of the fibrin may reveal tubercles. The subjacent myocardium often suffers infiltration and degeneration.

Symptoms.—Symptoms are lacking in most cases; pain is inconstant and when present is by no means characteristic.

Physical Findings.—Inspection and percussion are negative. The writer has seen a strong apex beat where the autopsy revealed a fibrin deposit an inch in thickness. Palpation and auscultation may disclose a friction fremitus, fully described under:

Pericarditis with Effusion.—This form has been called the second stage, the fibrinous form being the first stage. The fluid may be *serous* with much or little cellular or fibrinous admixture (serofibrinous); the *hemorrhagic* type is observed in the aged, in scurvy, cancer, tuberculosis, purpura, hemorrhagic exanthemata or Bright's disease, the exudation being tinged with fresh or altered blood; the *purulent* type is usually fibrinopurulent rather than purely purulent, and is often associated with pyemia or contiguous suppuration; the effusion may be *putrid*, when caused by pyemia, carcinoma of the esophagus or stomach, or lung cavities. Sixty-seven per cent. of pericarditides are serofibrinous, 19 per cent. hemorrhagic and 14 per cent. purulent. The pathological sequences of pericarditis are *fatty and cloudy degeneration, inflammatory infiltration*, or actual exulceration of *the heart muscle;* rupture of a purulent exudation externally into the tissues of the chest, or possibly into neighboring organs or cavities; inflammatory extension outside of the pericardium (*pericarditis externa*), to the mediastinum or pleura; formation of polypi, which may, in rare cases, become foreign bodies; complete resorption of the exudate and *restitutio ad integrum*, which is a rather uncommon issue; organization of connective tissue, either as local adhesions, mostly near the fixed base of the heart, because the heart's movements may detach apical adhesions, or as general obliteration of the sac (*concretio pericardii*); and, finally, desiccation of the exudate, leaving a cheesy mass which may calcify. Endocarditis is less a sequence than an associate or cause of pericarditis.

Symptoms.—No symptom is diagnostic. Total latency of symptoms obtains in 55 per cent. of cases. Daily examination of the heart in those diseases likely to cause pericarditis may save many diagnostic errors. Irregular *fever* may exist from the disease or the causal affection. It is often absent, especially in terminal pericarditis, and the temperature may be subnormal, even in purulent effusions. High fever is infrequent.

Dyspnea or thoracic oppression occurs in 90 per cent., due largely to heart compression by the exudate or extension of inflammation to the myocardium, and less frequently due to vagus irritation, phrenic neuritis, venous, pulmonary and arterial compression. It is remarkable that patients sometimes walk into the hospital with enormous pericardial effusions. The respirations are increased.

Pain.—Painlessness in pericarditis is the rule and is often absent in secondary pericarditis and is more common in small effusions; severe pain oftener indicates pleurisy than pericarditis. Like Andral, the writer has observed anginal pain radiating to the shoulder, arm and neck. Epigastric pain may precede pain elsewhere (Guéneau de Mussy) and the writer observed initial pain over the appendix. Barlow's patient put on a belt to relieve his pain. Precordial tenderness is at times present, and palpitation is common.

Delirium, restlessness, mania, convulsions, psychoses or even coma may be due to pericarditis or to complicating uremia or endocarditis. The symptoms may suggest gastritis, while pericarditis is found on examination. Vomiting, singultus, dysphagia, recurrent laryngeal paralysis and tracheal cough are pressure symptoms.

Physical Findings.—On these rests the diagnosis:

1. Inspection.—Precordial prominence, *voussure*, first noted by Corvisart and Louis, may develop when the chest is plastic, as in women and children, and when the intercostal muscles are inflamed or paretic. The left chest often moves less, since that lung is usually compressed. The apex beat is somewhat lower when exudation depresses the diaphragm; it is more to the left and the *dulness transcends the apex to the left* (see Percussion). The greater the effusion, the weaker the apex becomes, and *disappearance of the previously distinct apex beat is all-important.* There may be only an indefinite, diffuse cardiac impulse. On bending forward, the apex beat or cardiac impulse usually reappears. The interspaces may bulge. Collateral edema of the chest wall and undulation due to the heart itself are most rare. Graves and Stokes described tumor-like extrusion of the compressed lung above the clavicle. The upper edge of the first rib can be felt at its sternal attachment, being separated from the clavicle (Ewart's "first rib sign"). An epigastric tumor-like bulging of the luxated liver was noted by Auenbrügger and Corvisart. The veins of the thoracic wall are large. The decubitus is usually half-erect, dorsal or left-sided, to spare the sound lung; the attitude is fixed and there is abdominal inactivity during respiration. During convalescence the *apex may retract* during the systole, from adhesions.

2. Palpation.—The *friction-rub* is often felt. Palpation localizes the apex. *Disappearance of an apex beat previously observed, and the presence*

of a strong radial pulse, are very important; mere absence of the apex is observed in many conditions. *Tenderness* beside the ensiform, or between the attachments of the sternomastoid, is due to inflammation of the phrenic nerve. Fluctuation is never felt.

3. Percussion.—Dulness was first described by Auenbrügger. In good-sized effusions, nearly all the dulness is absolute. Three to five ounces of fluid are usually necessary to produce distinct physical signs. There are two complementary recesses in the pericardium: one over the basal vessels, where the fluid may accumulate, in which case it is early revealed by a triangular dulness, whose apex is directed downward (Skoda, Oppolzer), which may compress the large vessels; and the other in the fifth right intercostal space (Rotch, Ebstein), where the fluid exudate replaces the relative liver dulness; Epstein's angle, the right-angled junction of the right border of the heart and the liver, becomes more obtuse. Extensive *exudates produce a triangular, pyriform or trapezoidal dulness,* broad nearest the diaphragm and with the blunt apex near the manubrium (see Plate X, Fig. F.). Though the outline is roughly triangular, the right border is more vertical than the oblique *left border,* which *is beyond the apex beat,* if the latter remains visible (Skoda). The dulness may even reach the interclavicular notch, the right nipple, the left axilla, or Traube's semilunar space, and compress the lungs and depress the diaphragm. The apex beat shifts abnormally when the patient lies on his side, because the distended pericardial sac permits greater cardiac movement. Skoda taught that the heart falls back in the fluid, but the heart lies above the fluid and close to the chest wall. The dulness varies with fluctuations in the effusion and with resorption. The pericardial dulness is least typical when the effusion is covered by emphysematous lungs, or when an adherent lung cannot be pushed back by the effusion. In the erect posture, the dulness may be one-third to one-half as broad again as when in the dorsal position; it also is not so high; these signs may also occur in enlarged heart and valvular disease. Change of the dulness, especially when the patient lies on the right side is uncommon physiologically, whereas the shifting in the left decubitus is of minor value.

Dulness may persist from cardiac dilatation, massive adhesions or pulmonary retraction. The effusion may be enormous (10 quarts, Kyber; or 8 pounds, Corvisart), and may lead to a diagnosis of pleurisy, or pleurisy plus pericarditis. As the normal pericardial sac holds only 650 to 800 c.c., the pericardium must be greatly relaxed by the inflammation, to allow such large effusions.

4. Auscultation.—Auscultation reveals the chief and most reliable diagnostic sign, the *pericardial friction* due to attrition of the inflamed pericardial surfaces, first fully described by Collin, Laennec's assistant, in 1824. (*a*) It is usually a "*to-and-fro*" rubbing not exactly synchronous with the systole or diastole, which character is most clear when the heart is slow. It is less often single, and then is late in the systole; it may be triple, *i. e.*, presystolic, systolic and diastolic. It depends less on the intensity of the inflammation, than upon the accidental quality and location of the effused lymph. It is often absent in very soft fibrin-

ous deposits, hemorrhagic or purulent inflammation. In contradistinction to pleurisy, where the effusion of fluid abolishes the primary friction, it usually persists, even with great exudation (Stokes), especially at the base, since here the heart may touch the parietal pericardium. (*b*) In *quality,* it may be harsh, soft, like a gentle interrupted scratching of the ear with the finger, creaking, crunching, musical or even metallic (resonance from the stomach or intestines). (*c*) In *location,* it is most often heard over the base or the tricuspid region, where it is especially significant. It may also be heard over the apex, or possibly most clearly at the angle of the left scapula. (*d*) It is very *superficial* and close to the ear, not deep as in endocardial murmurs. Superficiality of the friction sound is absent when partial adhesions exist anteriorly, and the pericarditis is most intense behind the heart. (*e*) Its *propagation* is not wide (*il nait et meurt sur place,* Jaccoud), though it has been heard at a distance of nine feet. The murmur usually ceases abruptly when the stethoscope has been removed a short distance. It does not follow the same lines of propagation as in valvular heart disease. Propagation along the sternum is common. In children or sometimes in adults, the author has heard it over the whole chest. Coincident hydrothorax (Graves) and cardiac hypertrophy—almost exclusively in chronic nephritis—by bringing the heart closer to the chest wall, diffuse the friction more widely (Stokes). (*f*) *Inspiration* usually increases the murmur, due to the closer apposition of the pericardial leaves, caused by the wedge of expanding lung (Traube). (*g*) *Moderate pressure* usually *increases the friction,* especially in plastic chests. Change of posture, *e. g.,* leaning forward, often makes the murmur clearer (Corrigan, Stokes). (*h*) *The murmur* is *variable,* now present, now absent, now systolic, or again systolic and diastolic, varying in acoustic properties, or changing with change of posture; it is often short-lived, lasting sometimes but two to six hours. Disappearance of the rub is explained by regression, adhesions or weak heart. As a rule, the *heart tones* gradually become more or less *weakened* and distant. The *x*-rays outline the pericardial effusion; Maragliano comments upon the absence of pulsation in the gastric bubble.

Secondary Physical Signs.—Cardiac incompetence is due to the *mechanical hindrance,* by the fluid, of the diastole, especially of the auricles; then the face is pale; it is also due to *myocardial degeneration;* then the face is cyanotic. The signs of cardiac incompetence are hepatic and renal stasis, ascites, dyspnea, cerebral anemia (syncope), edema of the lungs, etc. The *pulse* at first undergoes irritative acceleration, but lasting frequency indicates myocardial change; rarely is it normal or slow (to 36), as a result of compression; dicrotism is frequent; there may be the pulsus paradoxus, weakening or intermitting with each inspiration (Traube). A very strong or water-hammer pulse may suggest underlying aortic disease. Irregularity is occasional, as is the pulsus differens and difference in the pupils. *Lung compression* is frequent. Bamberger observed a small area of compression, the size of a dollar, at the angle of the left scapula, where bronchial breathing, dulness (or tympany) and increased fremitus are noticed; the compression clears up when the patient assumes the knee-chest posture. Pleural puncture excludes the

possibility of fluid, and the absence of crepitant rales excludes pneumonia. Bamberger's sign also occurs in hemopericardium. There may be tympany (or dulness) under the left clavicle and to the left (and sometimes to the right) of the heart in fair-sized effusions. Ewart describes tubular breathing near the right mamma. Tracheal compression and thrombosis of the innominate veins may result from compression. The second pulmonic sound may be early and sharply accentuated. The second tone may be split. Metallic heart tones may be due to an adjacent cavity, pneumothorax or the distended stomach and intestines. A systolic murmur over the aorta has been explained by pressure or inflammatory relaxation interfering with its normal vibration.

Diagnosis.—Differentiation rests upon the *friction, dulness* and *apex beat.* Puncture alone reveals the character of the exudate. Fibrin formation and serous exudation usually coexist.

1. THE PERICARDIAL RUB is practically *pathognomonic* of pericarditis (von Dusch). (It occurs very rarely in cholera, pericardial tubercles, cancer, soldier's spots, hypertrophied heart, and the heart beating against an inflamed diaphragm or peritoneum.)

(*a*) In distinguishing it from *endocardial murmurs,* its acoustic quality, superficiality, lack of rhythmic precision, variability, slight propagation, palpatory differences in the rub, its basal location and increase by pressure or inspiration are most decisive. Valsalva's experiment—*i. e.,* a long inspiration followed by expiration with closed glottis and tense abdominal muscles—intensifies pericardial friction and decreases endocardial murmurs. Frequently pericarditis is a complication or coördinate phenomenon of endocarditis which may become manifest only after the pericarditis subsides.

(*b*) In *pleuropericardial* (extrapericardial) *friction* originating in an inflamed pleura, friction depends not only on respiration, but also on the heart's action. The distinction lies (i) in the close connection of the pericardial rub with the cardiac activity, persisting during expiration, and (ii) in the location of the pleuropericardial friction along the lingual lobe, in its intimate relation to respiration and in its cessation in extreme inspiration or expiration (inspiration also augments true pericardial friction). Valsalva's test is also useful (*v. s.*). Pericardial friction toward the base or sternum is less often confused with pleural friction. Differentiation is difficult when the basal vessels beat against a tuberculous cavity or pneumonia.

(*c*) In *precordial emphysema* the crackling sounds of interstitial emphysema (air) in the anterior mediastinum resemble rales, but closely follow the heart's action and are often metallic. The condition of the heart tones is decisive.

(*d*) *Pericardial splashing* (see PNEUMOPERICARDIUM).

(*e*) *Crepitant rales* are easily differentiated.

2. *The dulness of pericardial exudation* is typically pyriform or triangular. The markedly increased *relative cardiac dulness* should be considered rather than the *absolute* dulness (which is often little altered in pleural adhesions or pulmonary emphysema).

(*a*) *Hydropericardium* and effusive pericarditis may be confused, because the friction may disappear, and fever may be lacking (chronic pericarditis). Hydropericardium is distinguished by its etiological factors (*q. v.*), hydrothorax which almost always develops first, by lower dulness, by resorption under digitalis and purges; and by absence of friction; paracentesis reveals the usual differences between exudates and transudates in their opacity, specific gravity and percentage of albumin (see PLEURISY).

(*b*) *Hemopericardium,* resulting from trauma, rupture of a coronary artery or ventricular or aneurysmal rupture, is usually sudden, is attended by syncope and blood is removed by puncture.

(*c*) *Pneumopericardium* (*q. v.*).

(*d*) From *cardiac dilatation* the differentiation may be most difficult, expecially when there is dilatation plus hydropericardium, in which case drastics, digitalis, friction, the history and evolution only, may decide. In dilatation the dulness is rarely triangular but rather is increased laterally, the shock-like impulse of the heart is more marked and often contrasts sharply with the weak radial pulse, compression of the lung is much less common, and the heart tones are more clear and "snappy". The *x*-rays are often decisive. The very distinctive relation between the apex and the outer dulness to the left is rarely fallible (*v. s.*). If the pulse is strong and the apex weak, pericarditis is present; but compression or weakness of the heart in pericarditis necessitates a weak, rapid pulse. Leaning forward may clearly demonstrate the apex within the dulness of the exudate. When the patient lies on his right side the apex in pericardial effusion becomes visible, which does not obtain in extreme dilatation; in children dulness oftener signifies dilatation than pericardial effusion.

(*e*) When there is *retraction of the left lung* away from the heart, the diaphragm is high, the apex and outer cardiac dulness coincide, the spleen is high, the pulmonary artery is exposed, and the proper signs of pulmonary retraction are elicited.

(*f*) In *mediastinal tumor* the dulness is irregular; there is no variation of dulness on postural change; and signs of increased conduction and bronchial breathing are noted.

(*g*) From *aneurysm* the differentiation is usually possible, as well as from *marginal infiltration of the lung;* callous pleura, encapsulated pleurisy, and anterior mediastinitis (usually with friction, but with more vertically elongated dulness).

Diagnosis of the Character of the Exudate.—The fluid is serofibrinous in rheumatism; ichorous in perforation from hollow viscera; hemorrhagic in scurvy, carcinoma, tuberculosis, hemorrhagic exanthemata and alcoholism; and purulent in sepsis and empyema. Paracentesis is the only certain test. Tuberculous pericarditis, even in clearly tuberculous cases, can be diagnosticated positively only by detection of tubercle bacilli in the aspirated exudate; a chronic pericarditis is probably tuberculous. In children 30 per cent. of pericarditides are suppurative and 17 per cent. tuberculous.

Course.—Exudation may follow the friction at once or only after many days. The exudate may be rapidly absorbed in a few days or endure for months. The average duration is one or two weeks.

Prognosis.—The prognosis depends largely upon the causal disease and the character and quantity of the fluid; it is usually good in rheumatic and serofibrinous cases. Septic, purulent, putrid or hemorrhagic types are serious. The pneumococcic form is fatal in 62 per cent. Death may be rapid in hemorrhagic cases; it usually is due to cardiac exhaustion, especially in chronic cases, and results from myocarditis rather than from simple pressure. The prognosis is more favorable in men than in women, young children or aged subjects. Benign cases may result seriously from *concretio cordis*.

Treatment.—1. The *hygienic treatment* is managed as in any acute infection with cardiac involvement. *Absolute rest* in bed is indicated, even during convalescence, to spare the heart. The patient should never sit up to urinate or defecate, for fatal syncope may follow the effort.

2. *Laxatives* should be given (sod. phosphate ℥ss–ij), for constipation *per se* may increase the heart action, and difficult bowel movements, with the attendant straining, may precipitate syncope.

3. The *diet* should be light or fluid, avoiding tea and coffee, which excite the heart. Alcohol may be given in sepsis.

4. *Local Antiphlogistic Measures.*—An ample ice-bag upon the precordium quiets and regulates the heart-rate, relieves palpitation and pain and is often the best cardiant and sedative. No drug quiets the heart like opium. A Spanish-fly blister, as large as the hand, often not only relieves pain but stimulates resorption.

5. *Causal therapy,* as salicylates, in the rheumatic form.

6. *Heart stimulation* is indicated when the pulse is irregular, fast or small, due to early cardiac irritation or to later mechanical compression by the exudate. Strychnine, coffee and camphor operate more quickly than digitalis or may be used to follow up its action. Cardiac depressants are obviously injurious.

7. *In treatment of the fever,* hydrotherapy is preferable.

8. *Compression symptoms,* such as dyspnea and vomiting, are met by the judicious use of morphine hypodermically. When the face is pallid the heart is compressed by the exudate and tapping is indicated. Tardy resorption of large exudates or sudden or severe symptoms therefrom necessitate *tapping of the pericardial sac,* under general aseptic precautions. The puncture is made in the fifth left (or right) interspace about an inch from the sternum, where the sac is not covered by the pleura. Gentleness in passing the trocar enables us to feel the giving way of the parietal pericardium and to avoid cardiac trauma. Removal of a small quantity frequently provokes absorption by relaxation of the vessels in the tense pericardium. The fluid is withdrawn very gradually by the gravity or siphon method (see PLEURISY). Sudden or extreme cardiac embarrassment may necessitate free venesection.

9. *Delayed absorption* is treated by potassium iodide gr. x–xx, by diuretin 60 to 90 gr. *per diem,* and by small doses of calomel followed

by salines. Hot packs, alcohol sweats and pilocarpine should be entirely avoided lest collapse occur.

10. *Early pericardiotomy* is indicated in suppurative or putrid forms. About one-half of the cases recover.

Adhesive Pericarditis.—*Pericardial Concretion (Synechia); Cicatricial (Callous) Mediastinopericarditis.*—Pericarditic adhesions within the sac or more extensive bands to the chest wall, pleura or spine develop in 41 per cent. of cases of pericarditis and are found in 6 per cent. of autopsies. In one group of cases, complete obliteration of the sac may occur with absolute *clinical latency;* the heart may beat itself loose from fresh or lax adhesions, especially about the apex. In a second group, there are undistinctive evidences of *cardiac insufficiency.* In a third series, *distinctive physical signs* exist. Adhesions most frequently follow serofibrinous pericarditis. A clear history or the actual observation of a previous pericarditis is a strong point in the diagnosis.

Symptoms.—1. Cardiac insufficiency is caused more often by dilatation or atrophy than by pressure on the coronary arteries or myocardial fibrosis. While severe heart symptoms without valvular disease in advanced life are indicative of myocardial degeneration, in the young they suggest pericardial adhesions (Wilks). Failure of the right ventricle, without valvular, renal or pulmonary disease, or sudden decompensation in valvular lesions, directs attention to possible pericardial adhesions. Weiss and Pick described an isolated ascites, associated with enlarged liver; it is probably due to inflammatory extension along the vessels from the pericardium to the peritoneum. Sudden death is sometimes due to pericardial synechiæ.

2. Systolic Retraction of the Interspaces at the Apex.—This may occur even with slight basal adhesions which hinder the systolic descent of the heart, while diffuse adhesions other than basal often produce no symptoms. It is especially significant when the normal systolic protrusion gradually is replaced after pericarditis by systolic retraction (for retraction of the apex also occurs in other conditions, and is merely an expression of impaired locomotion of the heart and lack of space at the apex). It is best seen on deep inspiration and disappears when the heart grows weak.

3. Retraction of the *sternum, epigastrium, diaphragm* and *lower chest wall* may be observed when adhesions also exist outside of the pericardium as an indurative mediastinitis, which attaches the heart anteriorly to the chest wall and posteriorly to the spine, and necessitates retraction, until cardiac weakness develops (Skoda). Broadbent's sign is a systolic retraction of the eleventh and twelfth ribs posteriorly.

4. The diastolic recoil of the diaphragm and chest wall which are retracted in the systole, is recognized by palpation and by auscultation as a dull vibration.

5. Diastolic collapse of the cervical veins is of great diagnostic value, and is due to their aspiration by the heart; it may produce diastolic pallor of the face.

6. The Pulsus Paradoxus.—The pulsus paradoxus, a pulse intermitting with inspiration, strongly suggests cicatricial mediastinoperi-

carditis, particularly with the previously mentioned signs. In mediastinopericarditis, inspiratory stretching of the great vessels by well-attached mediastinal adhesions, shuts off the pulse. Sometimes observed to a moderate degree physiologically, it is pathognomonic when (i) it appears without forced inspiration, (ii) when the pulse is completely suspended by inspiration, (iii) when the heart's action is strong and regular and (iv) when it is combined *with inspiratory swelling of the cervical veins* (*v. i.*). Slowing of the pulse during inspiration is explained as irritation of the vagus by the mediastinal connective tissue.

7. Inspiratory swelling of the neck veins (Kussmaul), which normally collapse during inspiration, is due to mediastinal bands, which, on inspiration, interrupt the return venous flow. Cyanosis may occur with each inspiration.

8. Other signs are fixation of the heart by adhesions, which gives no change of dulness by change of posture; absence of respiratory excursion over the heart; decrease of Traube's semilunar space by pleural adhesions; decreased movement of the left half of the epigastrium (diaphragmatic adhesion to heart); cyanosis and dyspnea due to adhesions with the diaphragm impeding respiration; expiratory weakening

Fig. 27.—Pulsus paradoxus (Kussmaul); *E*, beginning of expiration, and *J*, of inspiration.

of the apex by pleural adhesions; weak right heart with no accentuation of the second pulmonic tone; loud, even musical, murmurs, especially in the aged; systolic emptying of the veins of the thorax due to systolic dilatation of the internal mammary veins; a rumbling presystolic murmur at the apex, especially in children, without signs of mitral stenosis; precordial creaking when the patient moves the arm; and other mediastinal symptoms, *e. g.*, left recurrent laryngeal paralysis, venous thrombosis in arm, etc.

Diagnosis.—In the diagnosis, two groups of symptoms are observed; first, the myocardial (dilatation, hypertrophy, relative tricuspid insufficiency); and second, the mediastinal (pulsus paradoxus, etc.). (See Pick's Pericarditic Pseudocirrhosis, under Diagnosis of Hepatic Cirrhosis.)

Prognosis.—The prognosis depends upon the condition of the myocardium. Cardiac concretion is the usual cause of heart failure in juvenile rheumatic cardiopathies.

Treatment.—The treatment is identical with that of valvular disease or myocardial insufficiency. Brauer advocates surgical solution of the adhesions (cardiolysis), conditional on the myocardium being strong; 28 operations are reported (1910).

PNEUMOPERICARDIUM.

Gas or air in the pericardium. But 38 cases were collected in 1904 by W. B. James, from whom the figures below were taken.

Etiology.—It results from (*a*) trauma (18 cases), such as perforating wounds, crushing of the ribs or sternum; (*b*) perforating processes from cavities or viscera containing air or gas (15 cases); pulmonary cavities; gangrene, pneumothorax, esophageal or gastric ulcer, liver abscess or caseated bronchial glands. The attendant effusion may be ichorous, hemorrhagic, purulent (pyopneumopericardium), or more rarely serous (hydropneumopericardium). (*c*) Exceptionally gas has been observed without solution of continuity, due to the Bacillus aërogenes encapsulatus.

Diagnosis.—On *inspection,* there is *precordial bulging* and *absence of the apex beat,* though it may reappear upon leaning forward. On *palpation,* the vocal fremitus about the heart and the apex beat are gone, friction and occasionally splashing, churning, gurgling sounds may be felt. On *percussion,* when the patient is sitting, a *tympanitic,* cracked-pot, or even metallic note is obtained above or over the entire heart, especially when percussion is made with the plessimeter and the hard part of the percussion hammer. The tympanitic note varies during the systole and diastole; it becomes higher in the erect posture and varies with the amount of exudation. The change in note on change of posture and the condition of the apex depend upon hydrostatic laws, as is the case in pneumothorax. The lung is compressed. On *auscultation,* the heart *tones* vary with the fluid, being loud if there is no great effusion, and *usually metallic* or musical, like the sound from a zither. The same is true of the quality of the *friction* (for pericarditis is invariably present). The tones, the metallic tinkling (in 63 per cent.) or splashing, the falling-drop sound, the mill-wheel churning (in 50 per cent.) and succussion may keep the patient awake and may be heard in an adjacent room, as in cases of Stokes, Laennec and Graves.

Differentiation is required from cavities, encapsulated pyopneumothorax near the heart and distention of the stomach imparting a metallic note to the heart tones. Consideration of the heart dulness and apex beat renders the diagnosis easy; in James's series all but 6 were recognized.

Prognosis.—Death occurs in 70 per cent. of the cases, usually within two weeks, from the inherently fatal causal disease and from cardiac insufficiency; traumatic cases are more favorable.

Treatment.—Puncture to void the gas, and operation for putrid or purulent processes are indicated. (See PERICARDITIS.)

HYDROPERICARDIUM (HYDROPS PERICARDII).

Hydropericardium is secondary to *circulatory disturbances,* as heart or pulmonary lesions, or, infrequently, local obstruction in the pericardial veins; or *cachectic conditions* producing transudation, as Bright's disease or carcinoma.

Symptoms.—They are much the same as in pericarditis, except that friction is absent. The heart region is sometimes prominent, the apex beat is lost, the heart tones are distant, the fluid changes with change of posture and the dulness is clearly trapezoidal since no adhesions exist. In most autopsies a small amount of fluid is found which transudes during the death agony or after death. In hydropericardium, the fluid is yellowish-green, and is sometimes slightly tinged with blood. Evidences of inflammation are absent; the fluid is alkaline, contains some albumin, sugar and urea, and is of low specific gravity. Chyle-like effusions (chylopericardium) are very rarely seen.

Diagnosis.—The signs of fluid are the same as in pericarditis, but there is no temperature, tenderness or friction. Dilatation of the heart causes the most frequent diagnostic confusion (*q. v.* for differentiation). The etiological diagnosis is important, as finding a cardiac or renal lesion.

Prognosis and Treatment.—The prognosis and treatment depend wholly on the cause. Cardiac stimulants, blisters over the heart, laxatives, etc., are indicated.

HEMOPERICARDIUM.

Blood tinges the exudate in certain forms of pericarditis. A large quantity of blood may escape into the sac in trauma of the heart or of the sac, rupture of the heart (*q. v.*) or of aneurysms (*q. v.*).

The *symptoms* and *physical signs* relate to (*a*) the *acute anemia* with pallor, syncope and rapid pulse, which usually ends, in a few hours or days, in death; (*b*) signs of fluid in the pericardial sac. The *prognosis* is favorable only in the traumatic cases; death may be postponed eleven days. The *treatment* is expectant; the heart should be stimulated and salt solution infused.

DISEASES OF THE ARTERIES.

ARTERIOSCLEROSIS.

Synonyms.—Atheroma, arteriocapillary fibrosis (Gull and Sutton), endarteritis deformans (Virchow), angiosclerosis (Thoma).

Definition.—A regressive change, evidenced first by malnutrition in the walls of the large or small vessels and later by compensatory thickening, either localized or diffuse. Morgagni recognized atheroma.

Etiology.—1. Age.—Arteriosclerosis is the essential element in senile involution, although some individuals attain their hundredth year with no arterial change. Cazalis said, "A man is as old as his arteries," and Peter spoke of "the vital rusting." Though usually found after the fiftieth year, hereditary influences, syphilis or the *acute infections* may develop hard arteries before the fortieth year, in adolescence or even in infancy. Chiari found it in 27 per cent. of those under twenty-five years, and Fremont Smith collected 144 cases in children.

2. Sex.—Eighty per cent. of cases occur in men, from causes enumerated below. In women it usually occurs a decade later. In factory women Jusserand found a larger percentage than in men.

3. Arterial Poisons.—*Chronic alcoholism* causes 25 per cent. of all cases; lead, coffee, tea and tobacco (affecting the coronary vessels and those of the legs) are causative factors. Angiosclerosis has been produced experimentally by adrenalin.

4. Overwork is second in importance only to alcohol. Physical exertion brings one-third of the blood to the muscles, thus lowering the tension in other vessels (*v. i.* Thoma's Theory).

5. Poisons Produced by Perverted Metabolism.—*Interstitial nephritis* bears a threefold relation to arteriosclerosis: (*a*) It results from diffuse arterial hardening; (*b*) it raises arterial pressure mechanically (Traube) by increased peripheral resistance in the arterioles, or by alteration in the quality of the blood (Bright); (*c*) both processes are coördinate to a common cause. *Gout, diabetes, high living, overeating* and *obesity* come under this head.

6. Specific Infections.—Syphilis causes but 12 per cent. of cases. Tuberculosis (in 16 per cent.), typhoid, rheumatism, influenza, scarlatina and other infections have a direct bearing on vascular sclerosis; Oppenheimer described arteriosclerosis in children nine and ten years old, due to acute infections (*v. s.*).

7. Other Causes.—Nervous factors, as *worry, high tension* and *ambition are important.* Excessive brain effort tends toward cerebral localization, precisely as digestive excesses may develop changes in the mesenteric vessels, as working with the arms, as in butchers, may develop changes in the brachials, and as excessive walking may produce arteriosclerosis in the femorals.

Pathogenesis and Pathology.—(*A*) Thoma considers angiosclerosis a compensatory process; to restore the caliber of widened vessels, the tunica media contracts, or when it cannot contract, the subendothelial connective tissue increases in thickness. Thus in the fetus there is no connective tissue in the tunica intima, but it appears when the fetal circulation changes at birth, new areas of circulation being opened up. All bloodvessels have a nervous supply ending in the Vater-Pacini bodies. They respond to persistent low pressure or dilatation, by increased proliferation of the connective tissue. (*B*) Others regard arteriosclerosis as due to *inflammation in the nutrient vessels,* the vasa vasorum, leading to growth of connective tissue, *i. e.,* mesarteritis and periarteritis (Huchard, Köster). Arteriosclerosis in vessels with no vasa vasorum must be simply degenerative.

Conceptions of its morbid anatomy differ widely, atheroma of the aorta and endarteritis or arteriocapillary fibrosis being sometimes described under separate headings, but the difference is rather anatomical than pathological. Large vessels, as the carotid, contain more elastic than muscular tissue, but the small vessels are largely muscular. Two main forms are distinguished, the *diffuse* and the *nodular.* Without distinction as to form, Rokitansky found that arteriosclerosis occurred in the following order of frequency: ascending aorta, arch, thoracic, abdom-

inal aorta, splenic, iliac, femoral, coronary (next after the aorta, according to Huchard and Sansom), cerebral, brachial and carotid arteries. It is rare in the gastric, mesenteric and hepatic vessels and least frequent in the pulmonary trunk. Bergmann found the two varieties as follows:

	Diffuse form.	Nodular variety.
Radial	89 per cent.	2 per cent.
Ulnar	88 "	8 "
Tibial	87 "	8 "
Popliteal	64 "	25 "
Splenic	64 "	17 "
Femoral	58 "	14 "
Axillary	53 "	16 "
External iliac	49 "	15 "
Brachial	49 "	9 "
Subclavian	45 "	43 "
External carotid	44 "	22 "
Cerebral	40 "	43 "
Internal carotid	28 "	60 "
Ascending aorta	19 "	56 "
Common carotid	10 "	69 "
Abdominal aorta	0 "	75 "

(Both forms may coexist in the same vessels.)

It will be noted that where the diffuse variety is most frequent the nodular form is least observed and that the converse is true. The *diffuse* form is most frequent in the extremities where arterial tension is most variable and more collateral circulation prevails; the *nodose form* is most common in the aorta, its primary branches and the circle of Willis, where arterial pressure varies greatly.

To the *naked eye* the intima may be normal and smooth but most often is uneven and thickened. Small, gelatinous or yellowish, fatty areas are seen which though at first circumscribed, often fuse into large plaques; they occur where arterial pressure is greatest, as in the aortic arch (see SYPHILIS and ANEURYSM), or where the vessel divides or gives off branches, as the subclavian or intercostal branches. The plaques of atheroma or nodular arteriosclerosis represent nature's effort to fill out areas of degeneration and dilatation and thus even the caliber of the vessel. *Microscopically* the intima may be increased three- or fourfold from swelling, increase of connective-tissue and round-cell infiltration. Granular detritus, fatty needles and cholesterin plates are seen in the *atheroma plaques,* a name indicating their pulpy, grumous appearance. An atheromatous area rupturing into the lumen produces the *atheromatous ulcer,* which may cause embolism, thrombosis or occlusion. Deposit of lime in these areas leads to *calcification* or produces rough areas in the aorta which break on manipulation or may cut the finger. The lumen of the smaller vessels is often decreased or obliterated (endarteritis obliterans); their middle and outer coats at times are thickened by hyaline degeneration and fibrous tissue; the muscular coat may waste and all elastic tissue may disappear; the essential changes are in the elastic tunic. In the larger vessels dilatation may develop where thickening of the intima fails to protect the vessel wall.

The general results of arteriosclerosis are (1) *vascular* inelasticity, deformity, tortuosity, obliteration, and high arterial tension, and (2) *visceral* alterations in the heart, brain, etc. (*v. i.*).

The rare sclerosis of the *pulmonary artery* occurs in mitral lesions and emphysema; pulmonary atheroma may occur alone, sometimes with aneurysmatic dilatation of its branches and with pulmonary regurgitation.

Thoma's angiosclerosis includes *phlebosclerosis,* which frequently accompanies arteriosclerosis and sometimes occurs independently, in the lower extremities, lung and portal tributaries in cases of liver cirrhosis or pylephlebitis.

Symptoms and Diagnosis.—In diffuse forms the process may be *latent.* The clinical picture is extremely variable.

1. The *peripheral arteries* may be involved, with or without changes in the aorta, heart, brain and kidneys. The small arteries appear *tortuous, pulsating and lengthened,* as in the temporals or brachials when the elbow is flexed and the arm is rotated outward. With the finger, which

Fig. 28.—Pulse tracing in arteriosclerosis.

no instrument can totally replace, we distinguish between *hardness* of the vessel walls and *increased arterial tension,* although varying degrees of both conditions usually occur together. Normal blood-pressure does not mean a normal movement of the blood since accommodative vascular contraction may follow poor vascular filling; a large artery does not necessarily mean a strong pulse nor does a small artery always indicate a weak pulse. Pressing on the radial with one finger should obliterate the pulse beyond that point if the arteries are not sclerotic, unless there is an anastomotic or recurrent pulse from the ulnar to the radial vessel; this source of error is easily eliminated by simultaneous pressure on the ulnar vessel. The wall of the artery is hard, often uneven or nodular, and the serial atheromatous plaques sometimes feel like a rosary. In

Fig. 29.—Pulse tracing in extreme arteriosclerosis (Sahli).

extreme instances the vessel feels calcareous—the "pipe-stem artery." The Roentgen rays clearly show the arterial change. The *pulse* is hard (*pulsus durus*), retarded (*pulsus tardus*) or slow (*pulsus rarus*); it rises slowly and is long maintained (*p. rotundus*), the vessel is firm between the pulse beats, the elastic wave is lessened or absent, the tracing has a rounded or flat top, and the two radials may differ (*pulsus differens*). In vessel obliteration or absolutely rigid walls the pulse cannot be felt. Slight pressure on the vessels often elicits a systolic murmur. The right radial is normally larger and more often shows changes than the left. Changes may occur in internal vessels without change in the radials (and conversely).

Increased arterial tension is a symptom only; barely one-half of the arteriosclerotics show hypertension. The normal tension ranges between

110 and 140 mm., any permanent register over 160 indicating hypertension (excitement may raise the reading 10 to 20 points, temporarily). The causes of high tension are: (1) Toxins injuring the vessel walls (high living, sedentary life, intestinal putrefaction or tobacco); (2) causes raising blood-pressure without vascular injury (the toxemia of pregnancy); (3) local diseases, as nephritis, diseases of the brain, abdominal vessels or splanchnic circuit; (4) profound neurasthenia; and (5) arteriosclerosis. A pressure of over 160 indicates nephritis in 70 and of over 200 in 100 per cent. of cases. In few cases, hypertension—due to vascular spasm—is apparently independent of arteriosclerosis and nephritis. In determining blood-pressure instrumentally, we regard the systolic pressure (the maximum pressure, corresponding to the intraventricular pressure); the diastolic pressure (the minimal pressure, corresponding to the peripheral resistance in the arterioles); and the pulse pressure (the difference between the systolic and diastolic and normally 50 per cent. of the latter). Auscultatory estimation (Korotkow) gives the most accurate results. If the arm band is inflated to a point above the maximal pressure and the bell of a stethoscope is placed over the artery at the bend of the elbow, nothing is heard. As the pressure is slowly allowed to fall a systolic, knocking sound is heard over the artery as soon as the maximal pressure is reached. This sound (or first phase) is due to the sudden distention of the walls of the empty artery by the first jets of blood that pass below the occluding arm band. As the pressure falls, this knocking sound grows louder, is then often replaced by a murmur (second phase), which again gives place to a clear, knocking sound (third phase) similar to the first. This last sound then suddenly becomes dull (fourth phase) and, after a further fall of a centimeter or so, ceases entirely (fifth phase). The first and fourth phases are most important, indicating the systolic and diastolic pressure, respectively, and the mercury column is watched for the record at these phases. If the third phase is loud, long and clear, the heart is strong; if weak, there is decompensation.

2. *Heart Changes.*—(*a*) *Simple hypertrophy* of the left ventricle may occur, with heaving apex, loud second aortic tone, tense pulse and thickening of the arteries. Some "consensual" right-heart hypertrophy is common because of anastomosis between the ventricular muscular fibers or because of pulmonary stasis. The heart is not hypertrophied in the majority of cases; it does not necessarily follow arteriosclerosis in the extremities or brain, but most often occurs when the splanchnic vessels or the aorta are sclerosed. The apex may sink downward and to the left from relaxation and lengthening of the aorta. (*b*) *Hypertrophy plus dilatation,* with heart insufficiency, dyspnea, scanty urine and hydrothorax are common; a systolic murmur often leads to a wrong diagnosis of mitral insufficiency. Interstitial nephritis (*v. s.*) is the commonest cause of the hypertension, hypertrophy and dilatation. Cardiac weakness may accompany a blood-pressure of 200 to 300 mm. In high arterial tension a double impulse may be felt and seen at the apex, which precedes cardiac weakness. Edema is not frequent. Gallop-rhythm points to coronary or renal disease. The heart muscle shows various changes

since arteriosclerosis is prone to starve the myocardium; they are atrophy, myocarditis, fatty heart, myofibrosis, myomalacia cordis, the *cor renale*, heart aneurysm, angina pectoris, heart-block, Adams-Stokes's syndrome, coronary embolism, cardiac asthma and arrhythmia; these lesions fall under the two forms of visceral sclerosis: (i) inflammatory, occurring about the vessels, and (ii) dystrophic sclerosis, often somewhat distant from the vessel involved.

3. In *the aorta,* dilatation may give *dulness in the second (and third) right intercostal spaces,* confirmed by the fluoroscope; in the jugulum or abdomen may be felt the aorta, plaques or even crackling; the subclavian or innominate arteries may be felt even through their entire length. The normal aorta propels the blood onward after the aortic valves close; if the aorta loses its tone the peripheral vessels receive inadequate blood, depending wholly on the ventricular systole, giving perhaps a pulsus celer. *A short systolic murmur* over the aorta often results from atheroma or dilatation, and the *second aortic tone is then accentuated* and metallic. Its vibration may sometimes be felt; when emphysema is present it is best heard in the jugulum. The accentuation may be caused by increased tension, widening or rigidity of the aorta. The murmur and sharp tone are often heard in the neck and back, and the writer has heard the accentuation only in the neck. Atheroma may, by downward extension, cause aortic stenosis or regurgitation.

4. *Renal findings* are next in frequency. The kidneys are often contracted (the senile arteriosclerotic and the genuine contracted kidney). The renal findings are probably most often primary (see INTERSTITIAL NEPHRITIS). The urine may increase as a result of increased blood-pressure or decrease from asystole.

5. *Brain symptoms* rank next in importance. Syncope, vertigo, headache, tinnitus, neurasthenic symptoms, transient aphasia or hemiplegia result from transient ischemia. Brain hemorrhage and miliary aneurysms are frequent sequences; encephalomalacia results from cerebral embolism or thrombosis. Brain atrophy, depressive conditions, the lacunar sclerosis of Grasset and Marie, epileptic seizures and the Adams-Stokes's triad may be attributed to cerebral atheroma. Vascular brain lesions occur fairly often in cases which show arteriosclerosis in the extremities and temporal vessels, though sclerosis of the aorta, coronary or splanchnic vessels occurs more often without peripheral angiosclerosis. Most instances of myelitis, so-called, are not inflammation but arterial ischemia with myelomalacia.

Charcot's *claudication intermittente* (Erb's dysbasia intermittens angiosclerotica; intermittent limping; angina cruris) is chiefly due (*a*) to atheroma in the abdominal aorta and its branches in the limbs; it is seen in abdominal aneurysm in the horse. It appears also to result (*b*) from vessel spasm or (*c*) rarely atheroma in the spinal vessels. Erb, reviewing 159 cases found that 94 per cent. were in men over forty years of age; 33 per cent. were Jews; syphilis was causal in 23 per cent.; half were enormous, and half, moderate smokers; most cases developed in the upper classes. Pain, paresthesiæ and sometimes local cyanosis occur in the legs, which "give way" after moderate exertion. The dorsalis

pedis (sometimes also the tibial and femoral vessels) usually cannot be felt. Analogous changes occur in the arms, brain, heart, intestine, kidney and eye. Brooks reported an arteriosclerotic with the picture of myasthenia gravis.

Some vasomotor neuroses—as erythromelalgia, Reynaud's disease, acroparesthesia—and some neuritides and neuralgias are occasionally associated with angiosclerosis; sclerotic vessels are prone to spasm.

6. *The gastro-intestinal* symptoms rank next in frequency. Cases are described of dyspepsia, round ulcer, arteriosclerotic gastritis, gastro-intestinal hemorrhages, intestinal perforation and ulceration. Some cases resemble gastric cancer. Severe abdominal pain (angina abdominis) is due to local anemia (intermittent claudication of the abdominal vessels, intermittent dysperistalsis); Pal thinks spasm occurs in the mesenteric and gastric vessels; it develops in two or three hours after eating, and is often accompanied by distention and dyspnea. Proctitis, piles and blood in the stools are not uncommon. Abdominal angina is often associated with angina pectoris and angina cruris.

7. The *retinal vessels* are not infrequently embolized, narrowed or aneurysmatic, which leads to softening, hemorrhage and exudation. As a rule, retinal is associated with cerebral atheroma.

8. In the *respiratory tract,* emphysema and bronchitis are very commonly coincident or consecutive. Hemoptysis is rarely referable to atheroma of the pulmonary arterioles.

9. *Peripheral vessels;* gangrene results from obliterating endarteritis; 44 out of 81 cases of gangrene of the legs in von Eiselsberg's Clinic were arteriosclerotic. *Thrombosis or embolism* may occur in the aorta, cerebral or other arteries.

10. The *general nutrition varies.* Some writers distinguish between *a plethoric* and *an anemic type.* French writers speak of a *cachexie arterielle,* in which the pallor is distinguished from the dusky anemia of cardiac disease, but the anemia is more apparent than real.

Early Diagnosis.—Early symptoms are local *disturbances in circulation* and may only appear after exertion in cold weather, excitement, or mild infections as "bronchitis," which results from simple stasis, and responds only to cardiac therapy. The patient tires more easily and the arterial tension may rise; slight edema of ankles or epistaxis is suggestive. Early *nervous symptoms* are headache, irritability, emotivity, somnolence, difficulty in concentration or in undertaking new work, vertigo, neuralgias, insomnia, or neurasthenia appearing for the first time after forty years of age. Arteriosclerotic "rheumatoid" pains may be early symptoms. A slight albuminuria always should attract attention. Ocular and vasomotor disturbances may characterize the insidious onset. The above symptoms are important since successful therapy depends on the early recognition of what Huchard terms the "presclerotic" symptoms.

Prognosis.—The process is frequently latent until it is far advanced. Its early stage may remain stationary for a long period, but there is constant danger of apoplexy, angina pectoris, myocardial paralysis or uremia. When a mitral murmur develops, there appears every sign of failing tonicity and contractility; when stasis predominates anginal at-

tacks cease. Many deaths attributed to acute infections are deaths from arteriosclerosis. A patient may live years with a tension of 180 to 200 mm., asthma or alternating pulse; he may work with great energy for years, until the end comes—often very suddenly. The writer observed a blood-pressure of 315, with which the patient worked a year. The prognosis is most favorable in angiosclerosis of the peripheral vessels. A slow pulse is less favorable than arrhythmia and moderate increase in rate; the extra-systole is not unfavorable, whereas fibrillation is always menacing.

Treatment.—I. *Prophylaxis* concerns the etiological factors and the patient's mode of life. Alcohol and tobacco should be forbidden, mercury and iodides given in syphilis, the diet regulated in diabetes, colchicum given in gout, etc. Overwork and excitement are to be avoided. In early cases the process may sometimes be arrested.

II. *Hygienic and dietetic measures* are along lines recommended in chronic nephritis, though in general less strict. In some instances, mental rest or moderation, rest in bed and a low diet restore health, lower blood-pressure and revive the heart. To start with, the Karell diet is excellent, later carbohydrates, fats, fruits and vegetables are given, but the quantity of the intake is closely watched. Soups, gravies or meat rich in purin bodies are prohibited; later some meat, especially boiled, may be allowed. The salt-intake may require regulation. If the patient is able to resume work, exercise is adapted to his strength and to the effects upon the blood-pressure, but long hours of sleep, and keeping the patient abed longer than the sleeping hours, are most effective. Daily, prolonged warm baths and moderate sweats are excellent. *Massage, a warm climate, passive exercise* and the *Schott method* of treatment may be employed (page 323). Skoda employed hot hand- and foot-baths to reduce peripheral resistance. Saline laxatives may reduce hypertension.

III. *Drugs.—Iodide of potash* is generally advised—15 to 30 grains daily for twenty days; then during a ten-day intermission, nitroglycerin is used (see pages 227 and 335). In a non-syphilitic series of personally observed cases the walls of the nodular, hard vessels distinctly improved with administration of iodides. Rhomberg thinks that iodides operate less by vasodilatation than by decreasing the viscosity of the blood, and others believe that they stimulate the thyroid gland and Krehl warns against the symptoms of hyperthyroidism which iodides may elicit, as loss of weight (even resembling cachexia), nervousness and fever. Iodides are particularly indicated in arteriosclerotics with bronchitis.

Hypertension is pathological, yet clearly conservative; with some clinicians 200 mm. is an indication for drugs, yet here or with even higher figures, it is best to consider how the heart functionates. *Vasodilators must not be used indiscriminately. Nitroglycerin* gr. $\frac{1}{60}$ is equivalent to ♏iij of spiritus glycerylis nitratis, gr. j sodium nitrite or gr. iss erythrol tetranitrite. Nitroglycerin acts for thirty minutes, nitrite of amyl for seven, sod. nitrite for sixty and erythrol for one hundred and twenty minutes. The amyl pearls should be protected from the light, the spirits should not be diluted, as it thus deteriorates rapidly and the erythrol should be chocolate-coated. Nitroglycerin, if it initially reduces blood-

pressure, is not apt to maintain the lowered pressure, whereas sod. nitrite is more likely to maintain it, with repeated doses; nitroglycerin operates very rapidly when simply placed under the tongue. Erythrol is prone to produce violent headache. Hewlett proved that amyl nitrite not only dilates the vessels but stimulates the heart. Vaquez employs spts. ætheris nitrosi, ℥ss–j, over a protracted period. Lauder Brunton employs pot. nitrate and bicarb., āā gr. x and sod. nitrite gr. j in a full glass of water. These remedies may produce a distinct fall of blood-pressure and yet they seem also to benefit in some other way, possibly by reducing pressure or spasm locally, *e. g.*, in the coronary vessels. In considering their action, one must remember that spontaneous reduction of pressure may occur.

Chloral hydrate, gr. v–x, often operates better than nitroglycerin, in pain, high tension, insomnia and nervousness (combined with bromides). Chloral operated very successfully in one case with pressure of 315 mm. in which nitroglycerin always induced delirium. Coffee may relieve anginal and abdominal pains. Venesection, iron and *digitalis* are indicated when the heart muscle weakens. Huchard advises the following pill:

℞—Sodii iodidi . . . . . ℥j
Sparteinæ sulphatis . . . . . gr. xv
Pulv. glycyrrhizæ . . . . . q. s.
M. et fac in pil. no. xl.
S.—One after meals.

Rest is more efficacious than are cardiants. Morphine is a valuable nervous sedative, tonic and possibly a vasodepressant for severe cases of cardiac asthma, but must be used with great care.

ANEURYSM.

Definition.—A dilatation, usually localized and sac-like, of an artery. Aneurysm was described by Galen, Vesalius who made the first diagnosis, Paré, Lancisi and Morgagni.

Etiology.—(*a*) *Syphilis* is the most important cause; it was estimated at 80 and 100 per cent. The Wassermann reaction proves that the relationship between syphilis and aneurysm is as well established as that between syphilis and locomotor ataxia. Lancisi, in 1728, first recognized "venereal aneurysm". (*b*) *Trauma* is an overestimated factor. Sudden effort with weakened aorta may induce partial rupture of the aorta with consecutive aneurysm. (*c*) *Sex:* aneurysm is from two to eight times as frequent in males as in females. (*d*) *Age:* the old statement is still valid that aneurysm occurs before and arteriosclerosis after the fiftieth year; 80 cases under twenty years of age were collected by Le Boutillier (1903). (*e*) *Embolic and mycotic aneurysms* are usually small and may be multiple in the small vessels. Lewis and Schrager collected 97 cases (1909). (*f*) The *traction aneurysm* at the ductus arteriosus Botalli is rare. (*g*) The *arrosion aneurysm*, in which the inner coats bulge out when the adventitia is eroded, occurs largely in tuberculous cavities. The "aneurysmal diathesis" is spoken of by older writers; Pelletier found 63 in one case. Multiple aneurysms of the arch are clearly syphilitic.

Pathology.—The varieties are: (*a*) *True aneurysm,* in which the sac consists of one or more coats, very rarely all three arterial coats. The sac is usually (i) fusiform and diffuse, or (ii) sacculated and circumscribed. (*b*) *False aneurysm,* in which all the coats rupture, or in which one coat ruptures through the others, like a hernia. These are circumscribed or diffuse, especially in the transverse arch and abdominal aorta. (*c*) *Dissecting aneurysm,* wherein the intima of the vessel ruptures and the blood channels down the vessel between the intima and media or between the layers of the latter; it is a partial rupture. The rupture back into the aorta is an attempt of nature toward recovery; the patient may live for years. (*d*) *Varicose* aneurysm, wherein the sac ruptures into, *e. g.,* the innominate vein or cava; this form is also called *arteriovenous,* or aneurysmal varix when the main change is in the vein.

Atheroma occurs very frequently in comparison with aneurysm; and the pathology of aneurysm is that of *syphilitic mesaortitis;* at the roof of the aorta, fleshy nodes develop in the media and adventitia; about the vasa vasorum, cellular infiltration occurs which later necroses; cicatrization, shrinking or dilatation results; that these lesions are luetic, is attested by the history, presence of spirochetes (Reuter, Benda) and by the positive Wassermann reaction in 66 to 85 per cent. (*v.* pages 208 and 219). In the sac, thrombi form from the roughened intima and slowed circulation and to a small extent guard against extension and rupture. Aneurysm occurs at those points in the aorta on which the blood stream impacts most strongly; 59 per cent. of thoracic aneurysms occur on the anterior surface of the ascending aorta, 29 per cent. on the convexity of the arch and 12 per cent. on the posterior surface of the descending segment. Aneurysm of the pulmonary artery occurs in but 0.4 per cent.

Symptoms.—Bramwell distinguishes three classes of cases: (*a*) Those which are entirely *latent,* and become manifest only by rupture or at the autopsy. (*b*) Those presenting *symptoms* of *intrathoracic pressure* but without physical signs. (*c*) Those *signalized by distinct physical signs, e. g.,* by pulsating expansile tumor.

The Subjective Evidences of Aneurysm.—A disproportionate value attaches to the symptoms, because of its frequent latency or the variability or disappearance of its signs when the aneurysm changes the direction of its growth.

1. *Pain.*—Pain is either intrinsic, resulting from aortitis, localized, and dull or aching in character; or it is extrinsic, resulting from pressure on nerve trunks, especially in deep-seated tumors, or from the weight of the aneurysm, as illustrated by Stokes's case in which pain was relieved by the use of crutches. *Pain* in the majority of cases is the *first, the most notable,* and *most enduring symptom.* It is often alleviated by iodides, which is suggestive of aneurysm; it is usually paroxysmal, lancinating or boring in character (when, for instance, the spine is eroded); it is augmented by movement, but may cease when the part compressed is entirely eroded. The pain is local or is reflected into the brachial plexus, arm, neck or may cause numbness and paresis of the arm. Intercostal neuralgia is always suggestive of aneurysm. Though more usual

anteriorly, pain is sometimes felt in the interscapular region. Anginal pain follows involvement of the cardiac plexus.

2. *Dyspnea.*—Dyspnea is frequently *out of all proportion to the findings.* It is due to compression by the aneurysm of the heart, vagus or phrenic nerve, trachea, bronchi, lungs or pulmonary artery; or to compression of the plexus cardiacus or pulmonicus, producing bronchial spasm, and laryngeal spasm or paralysis. Dyspnea may occur with change of posture; it is paroxysmal and may subside entirely.

3. *Cough.*—Cough is one of its least distinctive features. It results from pressure on the air passages, vagus irritation, bronchitis or laryngeal involvement. It is often unproductive. Like all other eccentric pressure symptoms, it varies with the size and direction of the aneurysm, and it is paroxysmal (Morgagni).

4. *Difficult swallowing* results from direct compression, from pressure on the recurrent laryngeal nerve, inducing esophageal spasm, or most often from adhesions between the aneurysm and esophagus. It occurs particularly in low aneurysms of the "recurrent type" (*v.* page 404). One of the earliest aneurysms reported by Stokes was the size of an egg, and produced pain, dysphagia and compression of the left bronchus, with rupture into the latter. Dysphagia may be intermittent or present only in one posture. Its infrequency is explained by the ready lateral luxation of the esophagus. The danger of sounding the esophagus is well known; lethal hemorrhages have occurred in the physician's consultation room.

5. *Compression of the vagus* results in asthma, laryngeal spasm, vomiting, stenocardiac attacks, hiccough or dysphagia.

The Physical Signs of Aneurysm.—1. *Inspection.*—Stokes described *two distinct areas of pulsation* in the chest, "*like two separate hearts,*" most significant when the cardiac impulse is the weaker. Inspection is most advantageously made with the eye just below the level of the shoulder and near the chest. The typical *expansile pulsation* is best detected in superficial thin-walled aneurysmata in the ascending arch, or those presenting in the jugulum; pulsation is less a question of size than location. Visible pulsation may be absent even in the largest varieties; deeply situated aneurysms give diffuse or no pulsation. Throbbing over the aorta is least important when there is also marked arterial pulsation (aortic regurgitation). The *pulsation* must be *expansile* to establish a diagnosis; the author saw one case with an expanding throbbing aorta and dulness over the arch which was due to massive callous connective tissue involving the pleura and pericardium. Another instance was observed in pernicious anemia; the hands placed over the left side of the chest, as well as near the spine were lifted widely apart, and the expansile pulsation was seen at a distance of forty feet; the autopsy revealed no aneurysm. Pulsation lent to a tumor, a collapsed lung, the liver or a pleural exudate is readily distinguished.

2. *Palpation.*—Pulsation felt slightly later than the heart shock, is more obvious the slower the heart rate becomes. The *expansile pulsation* is more easily detected by the fingers than by the eye. Palpation may reveal remittent tension of the sac, *tenderness* on deeper pressure, resistance caused by lamellated thrombi and, in rare cases, fluctuation of the

tumor during diastole. Pulsation in a deeply situated aneurysm is best elicited by placing one hand anteriorly and the other posteriorly on the chest. A *thrill* may be felt which is most commonly systolic. A *diastolic shock* sometimes occurs over the jugulum and aortic area (Lancisi), due to retraction of the hypertrophic aorta near the heart.

3. *Percussion.*—*Dulness* is significant only when it is present to one side of the sternum, but not when confined to the sternum. Dulness in the left interscapular region is less valuable, because of the thickness of the chest wall. The dulness may so merge with that of the spine, heart, liver or spleen that no certain statement can be made. Aneurysms are often obscured by an emphysema, or they may grow from a deep origin into the lung substance. Dulness *ceasing* or *shifting* to another place, almost certainly indicates aneurysm. It is inexplicable that aneurysms often press upon relatively unyielding structures, while the softer parts in their immediate vicinity, as the lungs, are less compressed.

4. *Auscultation* is frequently negative, even in large sacs—explained by thrombi in the sac and weak heart action. *Two clear tones are most frequently heard;* the second circumscribed tone is pathognomonic of aneurysm especially of the ascending arch, and originates in the aneurysm itself (not in the closure of the aortic cusps). The frequent *systolic murmur* is blowing, roaring or scraping; it is due to vibrations in the aortic wall, to entrance or exit of blood from the sac, to blood currents, thrombi, or more rarely to pressure of the sac upon the aorta itself.

The less frequent *diastolic murmur* is probably caused by (relative) aortic insufficiency, when the aneurysm lies in the first part of the aorta. It may be due to folds in the aorta or to dissection of the aneurysm into the sinus Valsalvæ. A continuous murmur is rare. Of 132 cases only 12 presented a systolic murmur, 25 gave systolic and diastolic murmurs, and in 6, diastolic murmurs alone were found (Sansom). Douglas Powell detected murmurs in about half of his cases of sacculated aneurysms.

The Heart.—Its position varies with the size and location of the aneurysm. (*a*) Large aneurysms of the ascending aorta not only depress it, but cause such axial rotation that the left lies lower than the right ventricle; this type may cause pressure atrophy of the right ventricle. (*b*) Aneurysm of the undersurface of the arch may dislocate the heart to the median line. (*c*) Aneurysm of the descending segment may flatten the heart against the chest wall, simulating mitral regurgitation. (*d*) Secondary aneurysmatic outgrowths from the chief aneurysm complicate the physical findings; and sometimes attain incredible proportions. (*e*) In uncomplicated cases myocardial alteration is entirely accidental. The most violent aneurysmal pulsation Stokes ever witnessed occurred with marked myocardial atrophy. Failure of the heart to hypertrophy is explained by coronary disease or compression of the pulmonary artery, thereby decreasing the flow of blood to the left heart and coronary arteries; compression of the pulmonary artery may cause relative tricuspid regurgitation or pulmonary insufficiency. *Hypertrophy* of the left ventricle bears an intimate relation to aortic leakage; it may in rare cases result from aneurysm involving the entire periphery of the aorta. Stokes's early observations recount every symptom of aneurysm.

The Vessels.—The *pulsus differens* is frequently physiological, and the right radial is normally larger than the left, whence if the right radial pulse is smaller, it means more than if the left were slightly smaller. When the radials differ, it is advisable to compare the brachials. When one radial is small, its fellow ulnar is often vicariously larger. A pulse may grow smaller and later return to its normal size, due to change in the size and the direction of the aneurysm. Asymmetrical blood-pressures may be due to other causes than aneurysm, as atheroma, coagula, embolism or reduction of the lumen of a vessel by the dragging of the aneurysm or compression of the aorta by a sac between it and the spine. Changes are observed most often in the carotid and subclavian vessels. The *pulsus paradoxus* and capillary pulsation may occur with large aneurysm. *Retardation* of the pulse occurs especially in fusiform aneurysms. An aneurysm sometimes may be localized by regarding the vessels altered. If pressure is exerted upon the *superior cava*, dropsy of the upper portion of the body, of one or both arms, swelling of the face and of the tongue, sometimes exophthalmos and a "brawny" or "collar-like" distention of the neck occur. Obstruction above the azygos vein causes lividity and edema which are confined to the head and arms; when it occurs below that vessel the chest is also congested. The innominate and azygos veins or the venæ cavæ may be obliterated. *Rupture into the superior cava* is attended by cyanosis, and a continuous bruit (also heard in vascular sarcomata or aneurysms rupturing into the pulmonary artery or right ventricle.) In Ewart's case (not suspected during life) phlebotomy was performed, and bright-colored blood escaped in jets.

The Respiratory Tract.—*Tracheal tugging*, described by Oliver and Cardarelli simultaneously, is elicited by lifting the larynx with the fingers; a tugging sensation in the trachea is then felt with each systole; this symptom occurs also in left ventricle hypertrophy and in tracheal adhesions.

Tracheal pulsation may be demonstrated by the laryngoscope in many aneurysms. The examination should be made by direct sunlight, and the observer should sit before the standing patient. It also occurs in tumors which compress the trachea, and physiologically. A short systolic murmur can sometimes be heard over the trachea when the patient breathes quietly with the mouth open.

Recurrent laryngeal paralysis was first described by Morgagni and Todd was the first to note laryngeal muscular atrophy. Unilateral paralysis with the cadaveric position of the vocal cords, in the absence of other causes, is indicative of intrathoracic aneurysm, neoplasm or mitral stenosis. The cough is hoarse, clanging or brazen. It is a paralytic cough, *i. e.*, an imperfect expiration which lacks forcible closure of the glottis observed in ordinary coughing; it was called the bovine cough by Wyllie, from the fact that cattle have no false cords whose closure is an important mechanism in coughing. The right recurrent laryngeal nerve, curving around the subclavian artery, does not enter the thorax, and is therefore seldom involved; whereas the left recurrent nerve passes into the chest and curves between the aorta and left bronchus; Dieulafoy described a "recurrent type of aneurysm," which is usually

small, syphilitic in origin, often multiple, involves the left recurrent nerve, and causes spasm of the larynx, due to recurrent laryngeal irritation contracting the posterior transverse arytenoid muscle and inducing dyspnea and inspiratory stridor. Irritation of other vagus branches induces spasmodic dysphagia, anginal pain, etc.

Lung Symptoms.—Stenosis of the right bronchus is less frequent than that of the left, which lies nearer the aortic arch. The lung moves less freely than normal; it suffers inspiratory retraction, and the breath sounds and fremitus are diminished; at times air may escape only during the diastole. A short note elicited over the left upper lobe may arouse suspicion of the "recurrent type."

Rokitansky's dictum that large aneurysms are almost never asso-

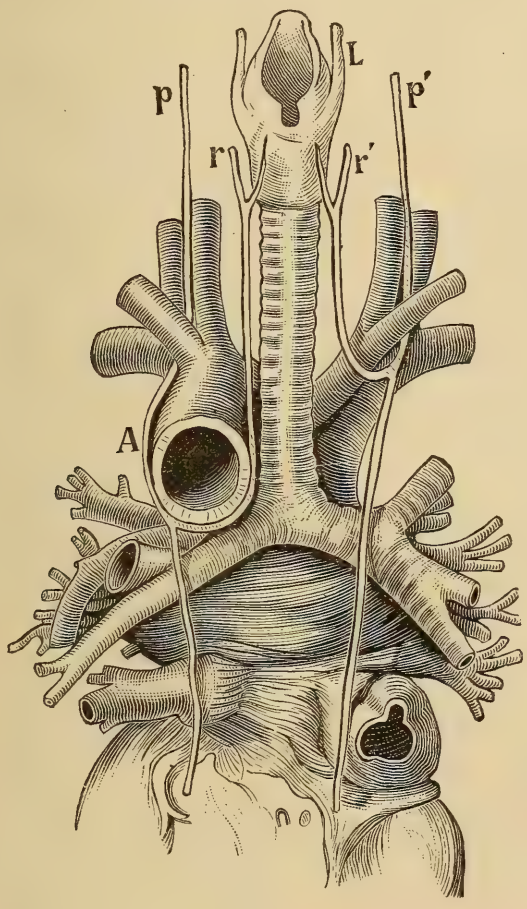


Fig. 30.—Dieulafoy's diagram of the region in which he describes his "recurrent type" of aneurysm; posterior view. *A*, aorta, in contact with the trachea and left bronchus; *L*, larynx (seen from behind); *p*, left and *p'*, right vagus; *r*, left recurrent laryngeal nerve, passing under the aorta, and *r'*, right recurrent, in relation to the subclavian artery.

Fig. 31.—Dieulafoy—I–VII, multiple syphilitic aneurysms.

ciated with *pulmonary tuberculosis*, was disproved by Stokes; tuberculosis results in 13 per cent. of cases of aneurysm from compression of the pulmonary artery or decreased lung excursion.

Bronchiectasis, bronchorrhea and suppuration of the lung are well called "aneurysmal phthisis" by Osler and Ross. *Pulmonary retraction* is observed from compression or adhesions. *Pleurisy with effusion* may complicate and entirely obscure an aneurysm. *Gangrene* of the lung or bronchial mucosa occurs from compression of the nutrient artery of the

lung or stagnation of mucus; it may produce subcutaneous emphysema. *Pulmonary hemorrhage* may occur from gross rupture into a large tube, from mere granulations in a compressed tube, or from an aneurysm slowly leaking into the parenchyma of the lung. The hemorrhage may be continuous, premonitory or fatal; it may be seen as flecks in the sputum, as rusty sputum, as prune-juice expectoration, as pure blood or more frequently as partly coagulated blood.

Sympathetic Symptoms.—The pupils are often involved; myosis results from sympathetic paralysis and high blood-pressure, and mydriasis from sympathetic stimulation and low blood-pressure. Irregular, immobile pupils, absence of the patellar reflexes and tabes compose the Babinski syndrome.

Diagnosis.—The clinical signs and symptoms are most variable (Stokes). The presence of an aneurysm should be suspected when *marked pain* or *paroxysmal dyspnea* occurs. *Variability* of physical signs is suggestive of aneurysm.

Early Diagnosis of Mesaortitis.—The sole therapeutic hope lies in the early recognition of aortitis. In the secondary stage of lues, its incipiency is marked substernal soreness, slight paroxysmal dyspnea and angina, pulsation of the cervical vessels, cardiac hypertrophy, substernal dulness and a positive Wassermann reaction in 85 per cent. (Longcope and Donath).

Diagnosis of Location.—1. Aneurysm of the *ascending portion*, called "aneurysm of physical signs," is most often observed in the first interspace to the right of the sternum, which is often eroded. Luxation of the heart to the left, radial slowing, compression of the superior cava and pulmonary artery with hypertrophy of the right ventricle, dyspnea and phthisis occur. Anginal or other cardiac symptoms are frequent, and the aneurysm, however small, is usually rapidly fatal from rupture into the pericardium, pleura, upper cava or heart chambers. Aneurysms just above the valves tend to grow downward because of the reflux of blood from the aorta, whereas those located higher grow upward because of impact of blood from the left ventricle. Sympathetic involvement is common.

2. *In the arch*, the "aneurysm of symptoms" is observed with left recurrent paralysis or spasm, stridor, pulsation in the jugulum, unusual prominence or luxation of the sternal ends of the clavicles, prominence of the subclavian arteries, dulness to the left of the sternum, myosis, slowing or lessening of the left radial pulse, compression of veins, particularly the left innominate, the trachea, thoracic duct, sympathetic ganglia, bronchi and left upper lobe, and disturbance in the brain circulation (hemiplegia) when the carotid artery is occluded. These aneurysms usually grow backward.

3. In the *descending segment*, the "aneurysm of latency" is most often encountered. If there are symptoms, pulsation in the left interscapular region, slowing of the femoral pulse, pressure on the azygos or hemiazygos, pressure on the spine with erosion and tenderness to touch, intercostal neuralgia (sometimes with recurrent attacks of herpes zoster), stenosis of the left bronchus or esophagus, pressure upon the heart,

forcing it against the sternum, and increased heart rate from plexus pressure, are present. The *x*-rays alone detect aneurysms lodged in the lung substance; pulsation is often observed, but not invariably. Letulle in three years detected 27 latent aneurysms by the *x*-rays alone.

The Wassermann test was present in 74 per cent. of 182 cases (Longcope), and in 92 per cent. in those under forty years of age (Harris). To illustrate the associations of aneurysm, Chiari found that 59 per cent. of luetics had mesa-ortitis syphilitica and Straub found 84 per cent. of paretics with mesa-ortitis.

Differentiation.—Differentiation from *solid tumors* is determined by their propagated non-expansile pulsation, their more rapid development, leukocytosis, adenopathy and cachexia. In aneurysm the lymph glands are rarely enlarged. Pain, recurrent paralysis and dysphagia are common to all tumors, aneurysmatic and neoplastic. Venous ectasia is more common in tumor. A few rare instances of coincidence of tumor and aneurysm are observed. The pulsation in carcinoma is rarely associated with a diastolic "back stroke," which is frequent in aneurysm. An unequal pulse suggests aneurysm or atheroma. The bruit in tumor is usually systolic, but murmurs are frequently absent in aneurysm. Tugging is present in both but more common in the latter. Aneurysm runs a much more protracted course than tumors. Exploratory puncture, the *x*-rays and diagnosis *ex juvantibus* with mercury and iodides are of differential importance.

Other causes for pulsation, such as a dynamic throbbing (aortic regurgitation), throbbing in acute aortitis, pulsation in an aorta dislocated from kyphosis, and pulsation in pneumonia, anemias, or in vascular tumor of the lung or pleura, are usually differentiated when all symptoms and signs are carefully considered. In *pulsating empyema* the dulness is usually low in the chest and largely left-sided; puncture reveals pus and stops the pulsation.

Prognosis.—Recovery is rare, though spontaneous cure is possible by compensatory thickening of the intima or by organization and calcification of thrombi in the sac; Adami collected 39 cures. The course is generally two years. The author watched for twelve years a blacksmith who worked, though he had an aneurysm of the ascending aorta, which was as large as his fist, and which protruded beyond the ribs. The size of the sac is of no prognostic value. Those of the small "recurrent type" are most dangerous, and large ones may be protected by connective tissue.

Mechanism of Death.—(*a*) *Rupture* occurs in 75 per cent. of cases: 20 per cent. into the left and 13 per cent. into the right pleura; into the left bronchus 16; into the pericardium 12; into the left lung 9; externally 6; into the esophagus 5; into the trachea 4 per cent.; (*b*) *suffocation;* (*c*) *marasmus;* (*d*) intercurrent disease; (*e*) embolism; and (*f*) brain lesions. Hemorrhage usually shortens the course, but in one case rupture into the bronchus occurred five years before death. Another patient suffered external rupture of the sac; he collected the blood in a basin and, though he fainted from the hemorrhage, he lived four months, only to die of typhus fever. Stokes described an external

rupture in which the presence of mind of the nurse, in stuffing some cloth into the sac, kept the patient alive for a considerable time.

Treatment.—1. The *Tufnell treatment* dates from Morgagni, Albertini and Valsalva, without the venesection recommended by Valsalva, *i. e.*, absolute mental and physical rest for several months, and food reduced to the minimum. The blood-pressure is greatly lessened. The treatment is of value only in cases of sacs with small necks.

2. *Iodides* were given prominence by Balfour, though used first by Bouillaud. They increase the secretions but do not inspissate the blood. It is questionable whether arterial tension and dilatation of the sac are lessened; it is certain that pain is alleviated. Twenty grains are given at a dose (*v.* page 399). Aside from relief of pain, they are useless in fusiform or large aneurysms with wide communications.

3. *Cold* applications may relieve pain when the sac is superficial. Supports may relieve the pain and protect against rupture. Calcium chloride does not promote coagulation.

4. *Gelatin injections* (1 per cent. solution) are recommended by Lancereaux to produce coagulation in the sac; the results are not convincing. There is danger of embolism and tetanus (*q. v.*).

5. Electrolysis, *introduction of fine wire*, compression and surgical intervention have given discouraging results. A few recoveries are reported but one cannot help comparing their small number with the probably equal number of spontaneous recoveries (Börger, in 1906, compiled 19 cases of dissecting aneurysms of the aorta which recovered). Fatal thrombosis or total embolism of the aorta may occur.

6. *Dyspnea* is treated as in cardiac disease. Venesection may afford temporary relief, and morphine is finally indicated. Tracheotomy may be necessary in laryngeal spasm (reflex recurrent irritation) but the actual compression by the sac is usually too low for operative interference. Digitalis is at all times contra-indicated.

Abdominal Aneurysm.—Constituting 5 per cent. of aneurysms of the aorta, over two-thirds occur in the upper abdomen and 40 per cent. at or above the celiac axis. The average size is that of an orange and the tumor usually develops from the anterior surface of the vessel and grows downward. Most are false aneurysms.

Symptoms and Signs.—Four per cent. of abdominal aneurysms are *latent* clinically; a good-sized sac may be hidden under the arch of the diaphragm. *Pain* is severe, boring and constant or paroxysmal; it may lessen after hemorrhage from rupture. Indefinite *gastric symptoms* are less frequent than *duodenal*, where obstruction and hemorrhage may occur. *Inspection* shows pulsation in 65, *expansile pulsation* in 15 and *tumor* in 57 per cent. *Palpation* may elicit the expansile pulsation and in some cases a *thrill*. *Percussion* may reveal *dulness* but its close relations give indefinite findings; the diaphragm bounds the aneurysm above; the kidney to the left; the liver, pancreas and kidney to the right; and the stomach, pancreas and colon in front. The tissues about the sac thicken and produce adhesions. On *auscultation*, a *systolic murmur* obtains in 50 per cent. or two tones are heard; a diastolic murmur is sometimes heard. Rupture, diffusion by dissection, great thickening

of the sac and thrombus formation may easily obscure all auscultatory signs.

Diagnosis.—In two-thirds of the cases an incorrect diagnosis is made.

1. Compression symptoms, gastric and duodenal, may suggest ulcer or cancer. The spine, eroded in 10 per cent. of cases, may simulate *lumbago*, myelitis, Pott's disease and spinal tumor. Pulsation should be searched for along the spine. The affection may suggest renal colic, kidney tumor, perinephritis or psoas abscess (fluctuating blood from rupture). Pressure on the splenic vein may provoke much splenic enlargement. The heart is rarely compressed. Retardation or absence of the femoral pulse and dyspnea may be of value in differentiation.

2. Abdominal Pulsation.—"Instead of being your first, it should be your last idea that abdominal pulsation is due to aneurysm" (Jenner). *Epigastric pulsation* may be confused with cardiac hypertrophy, liver pulsation, the *dynamic* pulsation of aortic regurgitation, the pulsation of acute hemorrhage, or especially neurasthenic pain and throbbing, with thrills, systolic and even diastolic murmurs—Rosenbach's "intermittent dilatation of the aorta." The pulsation must be *distinctly expansile* (even then occasional errors are made) and a definite sac must be felt.

3. Communicated throbbing, lent to a tumor of the stomach lying on the aorta, may also be attended by a systolic murmur but the differentiation is usually made with ease, except in soft neoplasms. Tumors are often felt to better advantage in the genupectoral attitude; they are more movable, more often produce dilated veins and ascites. Abdominal aneurysms may, in exceptional cases, be movable. Stokes held that tumors developed upward and aneurysms downward.

4. (*a*) Aneurysms of the *celiac axis* are difficult or impossible to distinguish. Over 20 cases are recorded. They are often traumatic or embolic (mycotic). (*b*) *Superior* (20 cases, Baccelli, 1904) *or inferior* mesenteric aneurysms occur most frequently in children, from sepsis. (*c*) Over 20 cases of *splenic* aneurysm are recorded. The spleen is often enlarged. (*d*) In the *hepatic artery* (40 cases, Rolland, 1908) it is induced by acute infections (73 per cent.), trauma, embolism or gallstones; 41 per cent. develop in the main trunk and 25 per cent. in its right branch; in but 1 case did it develop in the artery of the cystic duct. Pain (71 per cent.), hemorrhage (58 per cent.), icterus (63 per cent.), vomiting and large spleen have been noted; in 79 per cent. rupture occurred (in 10 cases into the peritoneum); they never pulsate; but once was there a systolic murmur; Kehr operated successfully. (*e*) Over 10 aneurysms of the coronary artery of the *stomach* are recorded. (*f*) *Renal* aneurysm is rare (26 cases, Skillern, 1906; in 3 operation was successful).

Prognosis.—The clinical course averages one to three years. *Rupture* occurs in 70 per cent.: 33 per cent. into the peritoneum; 16 per cent. into the pleura; 7 per cent. into the duodenum; and 7 per cent. into the retroperitoneal tissue. Death does not necessarily nor at once follow rupture.

Treatment.—Treatment is as in the thoracic type. From its accessibility, operative procedures are more common and somewhat more successful. Continued compression of the aorta has benefited cases of aneurysm located low in the abdomen.

ACUTE AORTITIS AND ARTERITIS.

These are rare lesions, occurring in the aorta and associated with syphilis (*v.* page 208), aneurysm (*v.* page 401), sepsis, endocarditis, or in the peripheral vessels after influenza or other infections.

Of periarteritis nodosa (Kussmaul and Maier, 1866), or "arteritis proliferans nodosa," only 26 cases have been published; it consists of localized proliferation and round-cell infiltration of the intima, with its rupture through the other coats, and leads to thrombosis or aneurysm. It is probably an infection. It occurs largely in men between twenty and thirty-five years of age. The nodes are seen in the vessels of the muscles, heart, kidney, intestines and skin; they produce hydrops, muscular pains, nephritis, convulsions, paralysis, vomiting, diarrhea, alimentary hemorrhage, epigastric pain, low temperature and a rapid pulse. The affection is fatal within six to twelve weeks and is usually mistaken for typhoid or meningitis. In one-quarter of the cases syphilis was an antecedent and one patient recovered under mercury.

RUPTURE OF THE AORTA.

Rupture results from trauma, endarteritis or narrow aorta. But 6 cases of rupture of a sound aorta are known and only 48 cases of spontaneous rupture. All coats may be ruptured, usually transversely. If the rupture is partial, the resulting aneurysm may dissect even to the popliteal arteries. It most often occurs in the first segment when hemopericardium results, or into the lung, mediastinum or retroperitoneal connective tissue. The symptoms are sudden pain, a sense of "something ruptured" and acute anemia; sudden death is usual, though patients with dissecting aneurysm may live eleven years or even recover.

Embolism of the aorta is due to impaction of atheromatous plaques, tumor tissue, echinococcus cysts, endocarditic plugs or ordinary thrombi. It is very uncommon. Sudden death results if embolism occurs in the first part of the vessel. If it occurs near the iliacs, collateral circulation is possible. There are pain and stiffness in the legs, sensory disturbances, paresis and contractures from anemia of the spinal cord. The femoral pulsation is absent. The bladder and rectum are disturbed. Hemorrhage from the stomach, bowels and kidneys has been observed. The clot may back up toward the heart or it may resorb. The prognosis is bad and the treatment is symptomatic. *Thrombosis* results from infections, weak heart, aneurysm, atheroma or compression, mostly in the transverse arch or abdominal aorta at its division. Its pain, paraplegia or anesthesia is sometimes confused with spinal cord disease. If gradual, collateral circulation may be established.

SECTION III.

DISEASES OF THE RESPIRATORY TRACT.

DISEASES OF THE NOSE.

ACUTE RHINITIS, CORYZA.

Definition.—An acute nasal infection.

Etiology. — (*a*) It is an *independent infection*, sometimes epidemic in the spring and fall; cold is only a predisposing factor. The most common organisms are the Micrococcus catarrhalis, Micrococcus paratetragenus, Bacillus septus, Bacillus Friedländer, and probably the Bacillus influenzæ and pneumococcus; 80 per cent. of cultures are negative. (*b*) It is a *concomitant* of various *infections*, notably of influenza, measles, pertussis, etc. (*c*) *Toxic factors*, as iodides, bromides, ipecac dust, dust, etc., may cause coryza. (*d*) *Local disease*, as chronic catarrh, polyps and ulceration are predisposing factors.

Symptoms.—The initial symptoms are depression, chilliness, fever of 101° to 102°, pains in the limbs and sometimes, in the young, delirium. The mucosa is dry, reddened, painful and swollen. There is repeated sneezing and anosmia. Herpes is common. There follows a clear, watery, nasal discharge (*coryza*) for one or two days, which subsides at night and recurs in the morning; within two days it becomes thickened and purulent. Extension may occur to the eyes, frontal sinus, antrum of Highmore, ear, by the Eustachian tube, or to the pharynx or larynx. In infants nasal occlusion prevents nursing and may cause dyspnea, because the tongue lies so near the palate. Acute rhinitis lasts one-half to one week and is seldom confused except with incipient measles.

Treatment.—At the very onset lavage with simple hot water is often sufficient; Dobell's solution (sod. borat. and sod. bicarb. āā ℥ij, phenol ℥ss to water ℥x), diluted freely to avoid irritation, is beneficial; adrenalin solution 1 to 1000 is excellent, but excessive application may induce edema of the throat and epiglottis. A cocaine solution (the plain alkaloid gr. j to albolene ℥j) relieves turgescence and frontal pain, but is a dangerous remedy to give into the patient's hands. Dover's powder gr. x, a saline aperient and a hot bath at the onset, usually give relief, especially when followed by:

℞—Extr. belladonnæ . . . . . gr. iiss
Fluidextr. aconiti . . . . . ♏x
Acetphenetidini . . . . . gr. xv
Camphoræ monobromatæ . . . . . gr. xv
M. et ft. capsulæ x.
S.—One capsule every hour for five or six doses.

℞—Phenolis . . . . . gr. iij
Mentholis . . . . . gr. iv
Olei theobromatis . . . . . q. s.
M. et. fac suppositoria no. x.
S.—One locally in nose every two hours.
Menthol in infants may cause cyanosis and asphyxia.

Drugs are futile unless the patient remains in bed. Salicylates mitigate the frontal pain. Cool baths may avert recurrence.

HAY FEVER.

Catarrhus æstivus or summer catarrh, was first described by Bostock (1819). Morill Wyman described the "June" or "rose cold."

Etiology.—The disease has long been attributed to the pollen of timothy, rag weed and golden rod. Dunbar demonstrated that pollen contains toxalbumins. It prevails especially in the United States and England, develops in persons between fifteen and thirty years of age, and twice as frequently in males as in females. It is more common in the educated classes, and is most prevalent in the warm months and in cities. Three special factors are noted: (*a*) *A predisposition* in the form of nervous instability. (*b*) A nasal *irritability* or turgescence, to dust, odors, etc., hyperesthesia of the inferior turbinates, polyps or septal irregularities; Austin Flint became asthmatic from sleeping on feather pillows. (*c*) The *exciting irritant*, as pollen, dust, sudden change of temperature and apparently in some cases, mental excitement. Local anaphylaxis is the most recent theory.

Symptoms.—The onset is that of the vulgar coryza, but with greater constitutional *depression*. *Injection* of the *eyes* with lachrymation and photophobia accompanies or precedes the coryza. The throat, larynx and bronchi are dry and burning. Fever, 100° to 101°, is fairly common. There is pain over the frontal sinuses, sometimes over the temporal and occipital regions. The nasal vessels are congested and the *nose* itself becomes *swollen* and injected, resembling closely the alcoholic facies. In many cases *cough* develops, frequently with *asthmatic* seizures like bronchial asthma (*q. v.*). Recurrences are usual and attacks of coryza may alternate with asthmatic paroxysms. With the first frost the affection stops, as if by magic. The outlook is good as to life, for death from asthma is rare; as to recovery, the outlook is much less favorable.

Treatment.—(*a*) Prophylactic cauterization of irritable, congested or hypertrophic areas, and correction of polypoid or other abnormalities cure some cases and alleviate others. (*b*) *Nasal* treatment; much relief may be given by irrigating the nose with 1 to 1000 adrenalin or other astringent solutions; suprarenal extract in doses of gr. ij to v has been used internally. (*c*) The *general physiological resistance* is increased by fresh air, cool rubs, tonics and nervines. (*d*) *Change of climate* is

advisable before the date on which, with certain regularity, hay fever appears; a sojourn at the seaside, in northern Michigan, Canada and in a high altitude, as the Adirondacks or White Mountains, greatly relieves or checks the symptoms. (*e*) *Asthma* (*v.* BRONCHIAL ASTHMA). (*f*) *Dunbar's serum,* patented as pollantin, is used in the nose every morning, as a powder. In 1240 cases Dunbar claims that 56 per cent. were greatly helped, and 31 per cent. benefited. Vaccines are being tested.

CHRONIC RHINITIS.

Minor cases of chronic nasal catarrh are often treated by the practitioner; some are due to syphilis, anemia or to other constitutional causes. Chronic catarrh may develop from repeated acute attacks, or may be chronic from the onset.

The Hypertrophic Form.—The nasal mucosa is red, swollen and covered with secretion which may desiccate and sometimes form rhinoliths; the vessels are wide and surrounded by round cells, and the submucosa is thickened. The nasal obstruction necessitates oral breathing, partially nasal speech, obtunded taste and smell, and produces an elevation of the hard palate and a peculiar facial expression—often associated with adenoids and follicular hyperplasia in the pharynx. The secretion is usually thick, yellow and purulent, rarely thin and abundant. The lachrymal duct may be obstructed, and Eustachian occlusion may induce deafness; laryngitis, bronchitis, bronchial asthma, polyps and sinus disease are not infrequent complications. Nasal disease may be the cause of facial spasm, limitation of the field of vision, vertigo, mental depression and dysmenorrhea. In its treatment possible constitutional factors, as anemia, syphilis or scrofula are to be considered. Douches of carbolic acid, insufflations of equal parts of alum and calomel, and the cautery or chromic acid are indicated. Douches should be given under low pressure, lest fluid reach the middle ear.

The atrophic form may begin as an atrophic process or result from hypertrophic disease. It usually begins early in life. The mucosa is pale, dry and metaplastic, the erectile tissue wastes, the nasal passages are unduly roomy and the secretion emits a most offensive odor (*ozena*). Its actual cause is not determined; it may originate in sinus troubles. Various microörganisms have been found, as the Bacillus fœtidus, mucosus and fluorescens liquefaciens. It is associated with a widening of the face in 33 per cent. of cases. Its sequences are those of the hypertrophic variety. The treatment is not satisfactory. Douches of a 1 per cent. permanganate of potash solution, iodoform insufflations, massage with iodine 1 part, potas. iodide 10 and glycerin 100 parts, and packing with gauze soaked in balsam of Peru are employed. Paraffin injections beneath the mucous membrane are recommended to restore the normal lumen of the nasal passages.

RHINITIS FIBRINOSA.

Rhinitis fibrinosa has been considered under nasal diphtheria, to which it is most often due. It may result from streptococcus and staphylo-

coccus infection; it occurs in measles, scarlatina, pneumonia, typhoid and also as an independent lesion. Irrigation with 1 per cent. carbolic acid or 1 to 1000 sublimate solution is efficacious.

EPISTAXIS.

Epistaxis is a symptom only. Its causes are: (*a*) *Local nasal* lesions, trauma, catarrh, picking of the nose, foreign bodies, ulceration (simple, tuberculous, syphilitic, carcinomatous) and telangiectases of the skin and nasal mucosa. (*b*) *Hyperemia*, either venous, as in cardiac and respiratory lesions, or arterial, as in cardiac hypertrophy, interstitial nephritis, atheroma, etc. Some cases are seemingly vicarious to suppressed menstruation. (*c*) *Acute infections*, principally typhoid. (*d*) *Hemorrhagic diseases*, as pernicious anemia, chlorosis, leukemia, pseudoleukemia, scurvy, hemophilia and cachexiæ. (*e*) Liver cirrhosis, icterus, and high altitudes are also causative.

The most frequent point of hemorrhage is the lower anterior part of the cartilaginous septum. The symptoms are local hemorrhage from the anterior nares, but occasionally trickling into the pharynx may simulate hemorrhage from the stomach when vomited, from the intestine when passed with the stool, or from the lung when coughed up. Occasionally acute anemia or death may occur, especially in blood diseases.

Treatment.—The patient should be kept quiet, in the semi-erect posture. After douching the nose with hot or ice-cold water to which adrenalin 1 to 1000 has been added, the nose should be tamponed by Bellocq's catheter, or in less serious cases the patient enjoined not to blow the nose, for this only dislodges the forming clot. Gelatin may be injected into the nose. Horse serum excels internal remedies, as ergotin. Erosions or ulcers may be cauterized later.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Acute catarrhal laryngitis is observed largely in males between twenty and forty, and in cold, windy, changeable weather. (*a*) *Cold* operates only by lessening the resistance, particularly in delicate subjects; the bacteriology of these "rheumatic cases" is unknown. (*b*) Acute laryngitis may complicate *acute* or chronic *affections* such as measles, pertussis, typhoid, hay fever and tuberculosis. (*c*) *Extension* may be caused *per contiguitatem* from the nose, pharynx or bronchi. (*d*) *Chemical*, thermal or mechanical agents, as dust, trauma, overuse of the voice, alcoholism, inhalation of tobacco smoke and internal use of iodides are etiological factors. (*e*) *Stasis* from thoracic lesions or enlarged thyroid, local ulcerations in the larynx, nephritis, liver disease and rhachitis are also causes.

Symptoms.—Sudden fever or chill is seldom observed. The local symptoms are *rawness* or tickling, dry, even spasmodic cough, and moderate pain on breathing; external tenderness or dysphagia usually indicates coincident pharyngitis. After a day a thin, transparent sputum appears, which later becomes purulent, yellow or in very rare cases, hemorrhagic. Some cases void no sputum (laryngitis sicca). The voice becomes *hoarse* or is wholly lost. The laryngoscope reveals redness, viscid mucus, swelling and occasionally epithelial erosions. The false cords are often swollen. The true cords lose their smooth, glistening appearance and become uneven, so that their even coaptation is hindered. The swollen mucosa may get between the cords, and their infiltrated muscles are often paretic. The acute symptoms last for a day or two and the catarrh remains about a week longer.

Diagnosis.—Nervous aphonia and cordal paralysis are distinguished by means of the mirror. (See VAGUS DISEASE.)

Treatment.—Alcohol and tobacco are forbidden. The general resistance is increased by cold rubbings, douches or baths; mufflers should be avoided and cold compresses should be applied over the larynx. The voice must be used carefully and the nose, uvula and pharynx should be examined for predisposing lesions.

At the onset sweating should be induced by hot drinks and a hot bath; Dover's powder gr. x or aconite, is indicated as in coryza; rest in bed is necessary. The voice must not be used. An ice-bag should be applied locally; codeine or morphine with bromides, and inhalations of moist air or steam should be given (*v.* ACUTE BRONCHITIS). Painting the larynx with a 3 per cent. silver nitrate solution may induce submucous extension and is not to be recommended.

℞—Heroin. hydrochloridi . . . . . gr. j
Terpini hydratis . . . . . gr. xl
Ammonii chloridi . . . . . ʒss
Extr. glycyrrhizæ . . . . . ʒj
M. et ft. capsulæ xxx.
S.—Two after meals.

In children acute laryngitis is attended by great swelling above and below the cords and by stenosis of the small, infantile larynx, resembling genuine croup (laryngeal diphtheria), from which it is distinguished as false croup, *faux croup* or *laryngite striduleuse.* After an attack of mild laryngitis, coryza or sore throat, the child becomes restless and anxious, usually during the night when the secretion accumulates. Inspiratory dyspnea develops, there is inspiratory retraction of the interspaces and epigastrium, the accessory muscles of respiration come into play, cyanosis appears and asphyxia seems imminent. The paroxysm subsides, spontaneously or after an emetic has been given, to recur on the next two or three nights. Trousseau remarked that there is rarely that complete aphonia observed in laryngeal diphtheria. The *diagnosis* is made from the history of coryza; the absence of diphtheritic patches in the pharynx and of diphtheria bacilli, found in diphtheria even when the throat seems normal; and the absence of severe constitutional reaction. *Laryngismus stridulus* (*v.* VAGUS NERVE) is not attended by

fever, laryngitis or aphonia. Laryngoscopic examination is notoriously difficult in easily frightened children. Treatment is the same as in adults; hot drinks, inhalation of steaming water and ipecac should be given to induce vomiting and evacuation of laryngeal secretion; the patient is awakened at intervals and given a hot drink to evacuate the mucus.

Other acute forms are (*a*) *laryngitis acuta nodosa,* observed in rheumatism and yielding to salicylates. (*b*) *Laryngitis fibrinosa,* which is usually diphtheritic, but may be streptococcic, in measles, scarlatina or other infections. (*c*) *Laryngitis submucosa acuta* invades the submucous tissues. This rare affection may develop as an independent lesion, may follow a similar process in the pharynx or may represent a septic localization. The invasion symptoms are severe, with fever, rigor and other septic phenomena. The laryngoscope shows injection and yellowish-white, bulging areas of submucous suppuration which, without operation, almost invariably cause death by edema of the larynx or by sepsis, for spontaneous rupture is exceptional. For treatment (*a*) an ice-bag should be placed over the larynx, morphine given for pain and whisky for the sepsis; (*b*) incision of the foci is indicated; and (*c*) if necessary, intubation or tracheotomy should be performed.

CHRONIC CATARRHAL LARYNGITIS.

Etiology.—The etiology is identical with that of the acute form, of which it frequently follows repeated attacks; it is often associated with, or is dependent on, pharyngitis or rhinitis.

Symptoms.—The symptoms are those of the acute form. *Hoarseness* alternates with fairly normal phonation. By the mirror there may be seen *injection,* darker than in the acute form, *swelling,* secretion and sometimes hemorrhages. There are forms with no secretion (*laryngitis sicca chronica*). In some forms granules appear, from proliferation of connective tissue (*laryngitis granulosa*). Erosions and rhagades may develop on the edges of the cords.

Treatment.—Prophylaxis is the same as in the acute form. Inhalations of 1 per cent. tannic acid or of 1 per cent. turpentine and sod. bicarbonate solution; insufflations of tannic acid, lead acetate and alum āā gr. j; and painting the larynx every third day with a 2 to 10 per cent. solution of silver nitrate with the aid of the laryngoscope, are valuable. Terpin hydrate gr. iij t. i. d. is often helpful. Sea air may be beneficial.

Other Chronic Forms.—*Chorditis tuberosa* occurs as small, grayish nodes on the cordal edges at the junction of their anterior and middle thirds, especially in singers and in alcoholics; they are composed of fibrous and epithelial hypertrophy. Scarification and lactic acid are indicated (*v.* TUBERCULOSIS).

Pachydermia laryngis is an increase of the submucous tissue and transformed epithelium into which connective tissue grows. *Warty excrescences* are common between the arytenoids, and the thick mucosa may resemble cancer. Topical application of salicylic or lactic acid, iodine in glycerin, electrolysis and the cautery are indicated.

Laryngitis hypertrophica inferior is a hyperplasia of the tissues below the cords, and is identical with rhinoscleroma, due to the downward extension of the rhinoscleroma bacillus from the nose. It may stenose the larynx. It is treated by scarification, dilatation, prolonged intubation, iodides, or, when extreme, by tracheotomy.

EDEMA OF THE LARYNX.

Edema laryngis is a better designation than edema of the glottis, as the vocal cords are seldom involved. It is most marked in the submucous tissue, about the epiglottis and aryepiglottidean folds; 70 per cent. of cases occur in males and 89 per cent. in those over fifteen years.

Etiology.—There are *three etiological forms:* (*a*) *Inflammatory edema* results from inflammatory diseases of the larynx, submucous suppuration or perichondritis; from typhoid, syphilitic, tuberculous or cancerous ulceration; from iodides, trauma, surgical measures, thermic, chemical and allied causes; extension from pharyngitis, parotitis or cervical cellulitis; and from infections. (*b*) *Congestive and hydremic forms* result from chronic heart and lung disease, tumors and nephritis, in which it may antedate edema elsewhere, or from cachectic conditions. (*c*) *Angioneurotic edema,* as in R. S. Morris's fatal case, is the last type.

Symptoms.—Symptoms appear with varying acuity, sometimes suddenly, as in Bright's disease, at other times gradually but progressively. *Hoarseness* or aphonia, and a *metallic cough* are common. The chief sign is *inspiratory dyspnea,* which characterizes nearly all forms of laryngeal stenosis. It is usually stridulous, and is attended by retraction of the interspaces, cyanosis, up-and-down excursion of the larynx and ultimate suffocation. The mirror shows a pallid swelling on incision of which a clear serum escapes. Edema of the epiglottis and aryepiglottidean folds may be felt with the finger if the tongue is drawn forward, or it may be seen without the mirror if the larynx is pushed up and the tongue depressed.

Treatment.—Treatment is urgent, irrespective of the cause, for the 75 per cent. mortality is due to delay until the patient's strength is exhausted. Ice over the larynx, purgatives and painting the larynx with a 10 per cent. silver solution are at best palliative. The edematous tissue should be fully incised, and in case this should fail, intubation or tracheotomy should be performed.

PERICHONDRITIS LARYNGIS.

Purulent perichondritis of the larynx nearly always results *secondarily* to various inflammations or ulcerations. Pus accumulates most frequently under the arytenoid or cricoid perichondrium. The cartilage may necrose secondarily. Perichondritis laryngis most often develops in males between twenty and forty years of age.

Symptoms.—Its symptoms are commonly obscured by the causal disease. The rare primary cases are the most acute. Well-localized pain and tenderness are peculiarly significant. *Dysphagia,* especially

from arytenoid involvement, is frequent, and even aphagia may result. *Phonation is impaired* by the fundamental disease, cordal paresis and muscular infiltration. The cervical glands sometimes tumefy. *Stenosis* and *dyspnea* are present. The *mirror* discloses focal accumulation of pus, swelling, redness, edema and perhaps fistulæ from intralaryngeal rupture. The probe may scrape on eroded cartilage. *Differentiation* from laryngitis and edema is impossible, unless definite pus pockets are seen. The *outcome* may be suddenly fatal from rapid edema; pus and cartilage fragments may rupture, thus giving relief or perhaps occluding the larynx; fistulæ may remain, and may burrow to the mediastinum; sepsis and aspiration pneumonia are not uncommon, and if recovery ensues, a distorted or stenosed larynx may remain.

Treatment.—Pain should be relieved by opiates, but early incision under cocaine, or tracheotomy or intubation is necessary.

ULCERATIONS AND NEOPLASMS OF THE LARYNX.

The tuberculous, leprous and syphilitic ulcers and glanders have been considered. Superficial erosions may follow catarrh; deep ulcers result from submucous suppuration, perichondritis, pressure by foreign bodies or laryngeal "bed-sores" analogous to intestinal ulceration in typhoid, etc.

Tumors include the benign singer's nodes (*v. s.*), fibromata, cysts, enchondromata, lipomata and the malignant tumors of which carcinoma is far more frequent than sarcoma. Primary *cancer* develops as an excrescence which assumes a cauliflower appearance; the basis of the later ulceration is nodular (*v.* page 209); hoarseness is an early symptom; incipient tumors are easily mistaken for laryngitis. Pain radiating to the ears or temples, cough and dysphagia are constant in the advanced stage. Early operation alone affords relief; inoperable cases must be narcotized.

Nervous aphonia, the *paralyses* and *spasms* of the larynx are considered under Affections of the Vagus Nerve.

DISEASES OF THE TRACHEA AND BRONCHI.

Tracheitis is frequent with laryngitis and bronchitis. Hemorrhage may result from ruptured varices. Tuberculosis, syphilis and tumors are very infrequent.

ACUTE BRONCHITIS.

Acute bronchitis in some localities constitutes 10 to 15 per cent. of all diseases. Three-fourths of all cases occur in cold, dusty or variable weather, between December and April. Its *forms* are (*a*) inflammation of the *larger air tubes*, bronchitis or tracheobronchitis, and (*b*) inflammation of the *smaller tubules*, known as bronchiolitis or capillary bronchitis, which in children is identical with bronchopneumonia (*q. v.*). Badham

(1819) coined the term bronchitis but it was first described by Laennec, who described every essential up to its bacteriology.

Etiology.—(*a*) It is a *symptom of various infections*, as *measles*, *pertussis*, *influenza* and *typhoid;* or frequently also in pneumonia, tuberculosis, sepsis, etc. In these maladies bronchitis is secondary and toxemic. (*b*) *Cold and bacteria.* It is disputed whether cold alone is an adequate factor without bacterial coöperation, but it at least plays a potent predisposing part. Even in cases of primary bronchitis the pneumococcus, pyogenic cocci, Friedländer's pneumobacillus and rarely fungi, yeasts, streptothrix or Vincent's organisms (*v. s.*) are detected. Pfeiffer's *Micrococcus catarrhalis* is found in 59 per cent. of cases (Ghon, Pfeiffer); it is larger than the staphylococcus and often occurs in pairs. (*c*) It may result from the *toxic effects* of potassium iodide or bromide, tobacco, mercury and alcohol which acts locally on the air passages. (*d*) *Mechanical irritants* are dust and particles of wool, stone, etc., which exist in many factories. (*e*) *Constitutional conditions*, as *nephritis*, rhachitis, obesity, diabetes and various cachexias, such as cancer or syphilis, are important etiologically. (*f*) *Venous stasis* in the pulmonary veins favors bronchitis; *mitral lesions* are far more important than other valvular diseases, arteriosclerosis, scoliosis, etc. (*g*) *Respiratory diseases*, as pneumonia, tuberculosis, abscess of the lung, asthma and bronchiectasis are associated with bronchitis. (*h*) *Age:* Acute bronchitis is peculiarly frequent in the *aged* and in *children* between six and thirty months old.

Symptoms.—1. Acute bronchitis of the larger tubes (*macrobronchitis or tracheobronchitis*) begins (*a*) with some *depression*, sometimes with fever and chilliness, or in children with marked toxemia. It often follows acute coryza or pharyngitis. (*b*) The *local symptoms* are *substernal rawness* or tickling; actual or intense pain or tenderness over the sternum is mostly tracheal, for the bronchi possess no pain nerves. On inspection of the trachea there is visible inflammation, diffuse or circumscribed injection, submucous ecchymosis, swelling and laxness of the mucosa, and dryness, which is followed by a transparent, glairy mucous secretion; the mucous glands are often distended by globules of mucin which slightly resemble young tubercles; these findings are also noted in the bronchi in the rare cases which come to autopsy, but injection and redness largely disappear after death. Microscopically we observe inflammatory paresis of the vessels, round-cell emigration and desquamation of the ciliated epithelium. (*c*) The *cough* is at first dry and unproductive and may remain so. Later a viscid, transparent sputum is raised, consisting of mucin and very few white cells (*sputum crudum*). A day or two later the coughing becomes less severe as the sputum grows more abundant and mucopurulent (*sputum coctum*); it contains polymorphonuclear and sometimes abundant eosinophilic leukocytes and epithelial cells showing myeloid degeneration. Localized inflammation at the tracheal bifurcation causes special irritation. (*d*) The *symptoms resulting from cough* are disturbed sleep; hoarseness from forcible closure of the glottis in coughing; pain in the side or at the diaphragmatic insertion, from unusual strain on the expiratory muscles; venous stasis which induces headache, vertigo, swollen cervical veins, cyanosis and epistaxis; mechanical

sequences, as vomiting, involuntary urination, especially in females, weak and old subjects; and in women, even abortion or uterine prolapse. (*e*) *Physical findings* are usually *bilateral;* unilateral signs suggest other lesions, especially tuberculosis. Auscultation reveals *rhonchi,* which result from swelling of the bronchial mucosa, slight stenosis by bronchial secretion and air passing through or dislodging the secretion; they at first are dry (*r. sicci*), and later moist (*r. humidi*). Over the large tubes these rales are sonorous, in the smaller tubules they are sibilant. They may be heard simultaneously over the large and small passages, or over the large and then by extension over the small tubules. These rales, large and small, may be felt with the hand (mucous fremitus) or may be heard without the stethoscope. The breathing may be "cog-wheel," lengthened or slowed, but in quality it is only exaggerated or puerile, never bronchial. Thoracic breathing develops in men when the tubes in the lower lobes are invaded. Percussion is wholly negative. Macrobronchitis lasts a few days, possibly longer than a week.

2. Microbronchitis or capillary bronchitis will be considered under bronchopneumonia, because of its great tendency toward pulmonary inflammation.

Treatment.—In the early stage a *full hot bath* may be given, followed by a *hot alcoholic drink.* A hot Turkish bath is dangerous from the exposure afterward. *Hot fomentations,* a hot water-bag or large poultice may be applied over the sternum, but cold packs over the neck and chest are even more efficacious. But few patients recover promptly unless they are kept in bed. With the initial symptoms, gr. x of *Dover's powder* should be given; it relieves pain, muscular soreness and coughing and modifies the inflammation; in children it should be given with caution because of the frequently unequal mixture of its constituents, and the peculiar susceptibility of the youthful nervous system to narcotics; in the aged it should likewise be administered with care, because the kidneys in old persons excrete slowly, and in cerebral atheroma may induce psychical disturbance and Cheyne-Stokes's breathing. Opiates are followed by some saline aperient. Other treatment is frequently superfluous, but if the cough persists or the initial symptoms are severe, we administer:

℞—Vini antimonii	ʒiv
Heroin hydrochloridi	gr. is
Vini ipecacuanhæ	ʒv
Syr. tolutani	q. s. ad. ʒij

M. et S.—One teaspoonful every three or four hours. The physician should watch lest gastric symptoms appear.

Brown mixture may be given (see Chronic Bronchitis).

℞—Potassii citratis	ʒiiss	ʒj
Codeinæ sulphatis	gr. iv	gr. j
Syrupi ipecacuanhæ	ʒiv	ʒiss
Succi limonis	ʒj	ʒss
Aquæ	q. s. ad. ʒij	ʒij

M. et S.—One teaspoonful every two hours. The first column is the dosage for adults, the second for children.

For high fever aconite and belladonna may be given, as in coryza, but tepid sponging is more beneficial. A kettle of water (with co. tr. benzoin ʒj–Oj) kept boiling in the room moistens the atmosphere, or a steam tent may be advisable, as in croup (see page 89). See formulæ under Chronic Bronchitis.

CHRONIC BRONCHITIS.

Etiology.—The etiology is that of the acute type. Chronic bronchitis, common in advanced life, may develop as a chronic process, or, more often, follows repeated acute attacks. It most often develops on change of weather or as the "winter cough."

Pathology.—The larger bronchi are most involved. Their color is a grayish-red or brown; the bloodvessels are thickened, and the bronchi become thicker from extravasation of leukocytes and proliferation of the peribronchial connective tissue; the mucous membrane sometimes hypertrophies and even protrudes in papillary excrescences, or is atrophied, and appears thin and pale like a serous membrane. Erosions are sometimes seen at the tracheal bifurcation. Stagnation of secretion is common. The inner surface of the bronchi is at times reticular, due to the prominence and hyperplasia of some elastic fibers and the wasting and sinking of other elastic or muscular fibers. Emphysema is frequently found at autopsy.

Symptoms.—Fever and pain are absent, except in the frequent acute exacerbations. The usual physical findings in *chronic macrobronchitis* are *large sonorous rales*, and sibilant rales when acute exacerbations cause invasion of the finer tubules. The cough varies with the intensity of the inflammation, the amount and character of the secretion, or changes in the weather. The sputum varies greatly, and upon its character is based the division into the following forms: (*a*) *Dry bronchitis*, the *bronchitis sicca* of Laennec, which is seen in the aged, associated with emphysema; the sputum is absent or very scant and viscid. (*b*) *Bronchorrhea*, in which the excessive bronchial secretion may be mucous, serous or purulent; in the serous form the secretion is thin and transparent, as in Laennec's case, in which two quarts were raised daily for twelve years; serous bronchorrhea may result from enlarged peribronchial glands; the purulent form may be confused with bronchiectasis, lung abscess or rupture of an empyema into the bronchi. (*c*) *Putrid bronchitis*, which complicates chronic bronchitis but occurs more often with bronchiectasis, foreign intrabronchial bodies and tuberculosis; in this form the sputum stinks, particularly when first voided. It consists of three layers: an upper foamy, a middle serous and a lower one of granular, grayish-green sediment, in which are the *mycotic plugs* of Dittrich (1850); these plugs contain various bacteria, leptothrix, strepto- and staphylococci, etc., and "acid-fast" bacilli which closely resemble tubercle bacilli, though narrower and more pointed at their ends. Fat and myelin droplets, fatty needles, leucin and tyrosin are also present. Fever is common; "drumstick" fingers may develop, and complications may follow, as lung induration, gangrene, pleurisy, hemoptysis, abscess, cerebrospinal meningitis or pseudorheumatism. (*d*) *Blood-stained sputum* occurs

particularly in the brown induration of the lungs, due to stasis (see page 368).

Chronic emphysema (*v. i.*) is an almost regular complication.

Diagnosis.—Determination of its relation to tuberculosis, chronic nephritis and other causal factors is most important.

Prognosis.—The prognosis depends on the etiological factors. Absolute recovery is most unlikely.

Treatment.—1. PROPHYLAXIS. Causal affections require treatment. Woolen underwear protects against sudden changes of temperature. In obstinate cases restriction of fluids may benefit.

2. CLIMATE.—A dry, warm, even climate is beneficial, as Florida, California, New Mexico, Egypt and the Riviera.

3. COUGH.—Codeine, morphine, creosote, cod-liver oil and other remedies are employed as in tuberculosis (*q. v.*). Ammon. carb. (*v.* PNEUMONIA) gr. iij can be given with syr. senegæ ℥j; syr. scillæ ℥j is incompatible with ammon. carb. but may be combined with ammon. chlor. gr. v–x.

℞—Ammonii chloridi . . . . . ℥iss
Misturæ glycyrrhizæ compositæ . . . . . ℥iv
M. et S.—One teaspoonful after meals.

The mistura glycyrrhizæ composita (Brown mixture; paregoric 12 parts, antimonial wine 6 and sweet spirits of nitre 3) may be given, ℥j-iv. If the heart is weak, it is contra-indicated because of the antimony. The stomach may become deranged by the ammonium, syrups, etc. Chloroform is valuable in paroxysmal, unproductive coughs, which opiates may not alleviate.

℞—Ammonii carbonatis . . . . . ℥j
Spiritus chloroformi . . . . . ℥iss
Syrupi senegæ . . . . . q. s. ad. ℥iij
M. et S.—One teaspoonful in hot water as indicated.

℞—Apomorphinæ hydrochloridi . . . . . gr. ss
Heroin hydrochloridi . . . . . gr. j
Spiritus chloroformi . . . . . ℥j
Aquæ . . . . . q. s. ad. ℥ij
M. et S.—One teaspoonful two or three times a day.

Syr. scillæ compositus (Hive Syrup) gtt. xx–xl, also contains senega, tartar emetic and antimony. Terpini hydras is excellent, gr. iij, t. i. d., given in capsules with an equal amount of benzoic acid. The *balsams* are even more efficacious, as *emulsum ol. terebinthinæ* ℥j in milk, though ol. santali ♏x–xx in emulsion is less likely to disturb the kidneys; *balsamum Peruvianum* ℥ss ranks with the best. The balsams and creosote are directly indicated in fetid bronchitis. Retention of secretion may be treated by rhythmical compression of the chest during expiration. Potas. iodide gr. v–x, t. i. d., is excellent in bronchitis, asthma and emphysema.

4. CARDIAC WEAKNESS.—Cardiac weakness is relieved more efficaciously by strychnine than by digitalis.

FIBRINOUS BRONCHITIS.

Etiology.—There are three types of fibrinous or croupous bronchitis: (*a*) The infrequent *primary* form, of which McPhedran collected 204 cases (1907); it occurs in males (66 per cent.), between ten and thirty years of age, and in weakly subjects; it results from the Bacillus diphtheriæ, pneumo-, strepto- and staphylococcus, aspergillus fungus, and protozoa, or from their toxins. (*b*) The *secondary* type develops by extension from laryngeal and pulmonary disease, or in infections, as diphtheria, pneumonia, etc.; tuberculosis causes 50 per cent. of this group. (*c*) *Mechanical, thermal* and chemical agents, valvular heart disease and exophthalmic goitre are causative factors in a few cases. Fibrinous bronchitis was first clearly described by Clark, in 1697, though Hippocrates and Galen mentioned the condition.

Symptoms.—The early symptoms of bronchitis, sometimes with fever, chills and hemoptysis (in 33 per cent.), are ambiguous until bronchial casts of fibrin or mucin are expectorated, after a severe coughing paroxysm. The casts may be eight inches long, and as thick as a finger; their upper, larger parts are circular or oval, solid or hollow, but the lower, small portions are branched, solid, spiral and often beaded with air and clubbed at their tips. Though usually fibrinous or croupous, many casts are mucous (bronchitis mucinosa). Their microscopic appearance is hyaline, their texture fibrillar, and their surface covered with blood disks, hematoidin or Charcot-Leyden crystals. Smaller casts may not be seen until the sputum is washed of the adherent blood, pus or mucus. Pending their evacuation, there are *signs of unilateral bronchial stenosis, e. g.*, dyspnea, cyanosis and inspiratory retraction, and absence of breath sounds in the corresponding lung. Slipping of the casts in the bronchus may cause peculiar flapping rales. Niemeyer observed the daily shedding of a complete cast of a bronchus and its divisions.

Diagnosis.—In some cases casts are not voided during life, and are found first at autopsy. Asthma and bronchitis fibrinosa have certain common symptoms, as paroxysmal occurrence, eosinophilia, crystals and spirals, but are usually differentiated with ease.

Course and Prognosis.—In *the acute form*, which lasts two weeks or less, there is a death-rate of 75 per cent. in the young and 50 per cent. in adults from asphyxia during evacuation of the casts. In *chronic forms* the process lasts for months, years or decades.

Treatment.—Treatment is unsatisfactory if not futile. Inhalation of steam and alkaline vapors and pilocarpin are given to loosen the casts, emetics to facilitate evacuation and *iodides* and mercurial inunctions to inhibit reformation.

BRONCHIAL DILATATION, BRONCHIECTASIS.

Etiology and Pathology.—Laennec first described bronchiectasis in 1819. It is found in 2 per cent. of autopsies and is *always secondary* (*a*) to *bronchial disease, stenosis* preëminently, and influenza, pertussis,

foreign bodies or pressure by aneurysm; (*b*) *to lung disease*, tuberculosis, interstitial or bronchopneumonia or atelectasis; coughing increases the expiratory pressure, even fortyfold; (*c*) to *pleural disease*, such as adhesions pulling on the bronchi. Nearly all cases are *acquired* and 77 per cent. occur in middle-aged males. The rare congenital cases may be divided into (i) the unilateral, universal or diffuse form, (ii) those due to congenital atelectasis and (iii) those resulting from bronchial (syphilitic) stenosis.

There are *two forms* of bronchiectasis: (*a*) The cylindrical or fusiform, *multiple* or *pater noster* form, which develops from the atrophy of the muscular and elastic fibers induced by protracted coughing and stagnation of secretion, *e. g.*, in catarrhal pneumonia, whooping-cough or emphysema. (*b*) The rarer *sacculated form*, in which the bronchus, reaching a diameter of even 3 inches, might be called a bronchial aneurysm. The connective tissue wastes, the ciliated are replaced by flat epithelia, and the mucosa is thin, red, sometimes villous. The sacs close at their necks, particularly when caused by aneurysm, syphilis or tuberculosis, and are filled with gelatinous or cheesy contents. Lung stones of calcium carbonate and phosphate may develop; they sometimes cause "bronchial colic," or symptoms like phthisis.

In multiple bronchiectasis the lung sometimes resembles a sponge or porous cheese. It occurs principally in the lower lobes, in bronchi of the third and fourth order, and is bilateral in 60 per cent. Adhesions are very common when the cavity reaches the pleura.

Symptoms.—Weil stated that most cavities escape detection and most diagnoses of cavities are incorrect. Sacculated give more symptoms and signs than cylindrical forms, which are almost always impossible to recognize.

1. The sputum and cough are paroxysmal. On change of posture or when secretion fills the sac and flows over onto the normal sensitive mucous membrane beyond it, cough develops and quantities of mucopus are voided in the "*mouthful*" *fashion* described by Wintrich. The largest amount is raised in the morning and frequently evokes vomiting. The daily quantity may amount to a quart. Its odor is acid, sweat-like, but less offensive than the fetor of putrid bronchitis or the foulness of gangrene. When collected it forms in three layers; the upper one is foamy, the middle serous and the lower purulent. A nummular sputum may be noted, as in tuberculosis, but if put in water it is more flocculent, more granular and airless; and microscopically it contains altered leukocytes and fatty needles, occasionally red cells, hematoidin crystals and very rarely, when the bronchial wall is ulcerated, elastic fibers.

2. Physical Examination.—(*a*) *Inspection* may reveal diminished respiratory movement, inspiratory retraction over the cavity or flattening of the chest wall. The patient generally leans toward the diseased side to lessen the trickling out of the secretion. In the rare bronchiectasis of an upper lobe the constant escape of the secretion results in exhausting coughing. The *x*-rays may locate the cavity. In rare cases the cavity may bulge through the interspaces. (*b*) *Palpation* and (*c*) *percussion:* Dulness prevails when the cavity is full; when it is empty there is

tympany. A higher note on opening the mouth, on inspiration or change of posture has the same value as in tuberculous cavities (*q. v.*). (*d*) On *auscultation, bronchial* or *metallic breathing* may be heard when the cavities are superficial but it is absent in dilatations which are small, deep or full of secretion. *Metamorphosing breathing* is a certain sign of a cavity. Skoda's superficial, "veiled-puff" sound is frequent. *Large* and *small rales,* heard persistently in the same locality, are suggestive of a deep bronchiectasis. The sound tissue over the cavity imparts a metallic consonance.

3. Complications.—Emaciation, putrid bronchitis, slight cyanosis and anemia are frequent. Hectic fever is often absent, but death may result with symptoms resembling phthisis. Hemoptysis from ulceration of varices occurs in 40 per cent. of cases; pneumothorax, pleurisy, empyema, lung induration or gangrene, emphysema, lobular pneumonia, amyloidosis and hypertrophy and dilatation of the right ventricle are fairly common. Marie's *ostéo-arthropathie hypertrophiante pneumonique,* rheumatoid affections, suppurative meningitis and brain abscess are occasional.

Diagnosis.—Diagnosis depends upon the character of the sputum, its paroxysmal voidance and the signs of cavity. *Differentiation* is required (*a*) from *tuberculous cavities;* this is made by elastic fibers and tubercle bacilli (definitelv determined by inoculation); tuberculous cavities are most often apical, but frequently bilateral; they void less sputum and suffer less rapid variation in their physical signs. Bronchiectatic cavities occur mostly in the lower lobes, void more sputum, less often induce hemorrhage and more frequently cause right-heart changes; (*b*) from *perforating empyema,* in which cholesterin and hematoidin crystals are far more frequent than in bronchiectasis; (*c*) from *abscess;* (*d*) from *gangrene* of the lung (*v. i.*); (*e*) from *putrid bronchitis,* in which there is no sudden volume of evacuated sputum or signs of cavity; (*f*) from *encapsulated pyopneumothorax,* which may be very difficult of differentiation; and (*g*) aneurysm or tumor causing bronchiectasis.

Prognosis.—Ultimate recovery is exceptional, except in acute cases, *e. g.,* postinfluenzal; patients have lived half a century. The complications require consideration (*v. s.*).

Treatment.—The therapeutic indications are (*a*) *evacuation of the secretion,* for which expectorants, raising the foot of the bed, and expiratory compression of the chest, as in bronchitis, are somewhat useful; sedatives should never be given lest gangrene develop; (*b*) modification of *putrescence* by balsamic remedies, as in fetid bronchitis; a dram of creosote is slowly vaporized over a flame, and the patient, in a closed room, inhales the fumes with the eyes closed and the anterior nares plugged; the procedure may be gradually lengthened from a few minutes to half an hour; the irritation evacuates the residual secretion; (*c*) *surgical drainage;* an accessible cavity may be incised and drained, though accurate diagnosis of its location is difficult, the mortality high and the affection multiple. In Garré's 57 cases, 63 per cent. recovered and 37 per cent. died; (*d*) *change of climate* (*v.* Tuberculosis).

TRACHEAL AND BRONCHIAL STENOSIS.

I. Tracheal Stenosis.—Etiology.—*Tracheal causes* are rhinoscleroma, syphilis (*q. v.*), tumors (cancer, polyp, fibroma, enchondroma, sarcoma or aberrant thyroid), foreign bodies and in rare cases perichondritis. Causes *outside the trachea* are more frequent, as retrosternal, cystic and circular goitre, or struma in which hemorrhage or inflammation has occurred. Aneurysm, adenopathies, mediastinal tumors, vertebral tumors or caries and thymus hyperplasia are other factors. Thiesen collated 135 cases of tracheal tumor, of which 89 were benignant and 46 malignant; 24 were fibromata and 10 intratracheal struma (penetrating the rings of the trachea, when sudden death may result, Paltauf).

Symptoms.—The symptoms develop *three stages:* (*a*) no symptoms or symptoms only on exertion; (*b*) constant dyspnea, and (*c*) suffocative attacks and final asphyxia. *Dyspnea* is mixed, *i. e.*, inspiratory and expiratory; respiration is slowed, there is inspiratory stridor and intercostal retraction, all the accessory muscles come into play and the head is held well forward. The pulse is tense. Tracheoscopy may reveal the location of the lesion; the patient should stand with his back to the strong sunlight, with the head bent forward, and the observer seated on a low seat before him. In 25 per cent. of tracheal stenoses, due to tumor or aneurysm, there is coincident paralysis of the vocal cords.

II. Bronchial Stenosis.—Etiology.—*Intrabronchial* causes are most common and consist of pus, mucus, blood, fibrin and foreign bodies; obstruction due to the latter is laryngeal in 33, tracheal in 26 and bronchial in 41 per cent. of the cases. *Extrabronchial* factors are next in frequency; aneurysm, mediastinal lymphosarcoma and perilymphadenitis leading to fibrosis occur most often; less common are pericarditis with effusion, dilatation of the left auricle, cancer of the esophagus, tumor of the lung and thyroid or thymic strumas. Twenty cases have been reported in which lymph glands ruptured into the trachea or bronchi, causing suffocation; only three were saved by tracheotomy. *Interbronchial causes* (*i. e.*, in the bronchial wall) are the least common, as syphilis, rhinoscleroma and tumors. Thirty-six cases of primary cancer are recorded. The right is stenosed nearly twice as often as the left bronchus.

Symptoms.—Occlusion of a bronchus lessens the amount of air entering the lung which it supplies, whence the *vocal fremitus* and *breath sounds* are *weakened* or suspended and the respiratory excursion and Litten's diaphragmatic sign are decreased. There is inspiratory retraction of the intercostal spaces on the affected side. *Inspiratory dyspnea* occurs, particularly in acute cases. The head is thrown forward. Absence of dulness excludes many causes of dyspnea, as pneumonia, etc.; after a time a somewhat tympanitic note may result from relaxation of the lung or acute emphysema. A local stenotic murmur over the bronchus is present in a few cases. Paroxysmal dyspnea may result from stagnating secretion. Coughing is usual and may resemble that of pertussis. The pulse is more tense. The *x*-rays and bronchoscopy may locate the obstruction. Death results from (*a*) the original disease, as aneurysm or tumor; (*b*) failure of the right heart; (*c*) asphyxia, with delirium, cyanosis or Cheyne-

Stokes's breathing, or (*d*) pulmonary edema, gangrene, tuberculosis, lobular pneumonia or hemorrhage.

Diagnosis.—(*a*) *Of the location:* In laryngeal obstruction there are local findings and free up-and-down laryngeal excursion; the subject holds the head well back, and the ingress of air to both lungs is equally impaired; tracheoscopy, bilateral lung involvement, absence of laryngeal excursion and the holding of the head forward are proof of tracheal obstruction; in bronchial stenosis but one lung suffers for air, the larynx is nearly immobile and the head is thrown forward. (*b*) *Of the nature* of stenosis, certainty is impossible without physical signs, as aneurysm, history of foreign bodies, etc.

Treatment.—Treatment is that of the cause, as syphilis, tuberculosis; thyroid extract in struma; or rest, mercury iodides and venesection in aneurysm. To dislodge foreign bodies the patient should be inverted, sharply struck on the back and shaken; the physician should have long forceps and tracheotomy instruments in readiness; the mortality is 52 per cent. without, and 23 per cent. with, operation.

Bronchial and lung stones (*v. s.*) result from tuberculosis of the bronchial glands, bronchiectasis, petrification of the bronchi, calcification and ossification of the lungs, and foreign bodies. Boerhaave described the case of the botanist, Vaillant, who expectorated 400 calculi. Symptoms are frequently absent or are those of the primary disease; purulent bronchitis, asthma ("bronchial colic"), hectic fever and hemoptysis mark some cases.

Stenosis may result from *bronchiolitis fibrinosa obliterans.*

BRONCHIAL ASTHMA.

Definition.—A paroxysmal dyspnea, due to nervous or reflex stenosis or spasm of the smaller bronchioles, and characterized by expiratory dyspnea, slowing of respiration, congestion of the bronchioles, a peculiar mucous exudation and acute emphysema. It is also known as asthma spasmodicum or nervosum, to distinguish it from other so-called asthmas, as the cardiac and renal types.

Etiology.—When due to no obvious cause, asthma is termed essential or *primary,* and when due to a clear cause it is called *symptomatic* asthma, reflex through the vagus: (*a*) *Nasopharyngeal disease* often initiates reflex asthma, *e. g.,* rhinitis, tonsillar hypertrophy, adenoids or pharyngitis granulosa; the nose may be so hypersensitive that the odor of flowers, dust, feathers or smoke precipitates an attack. (*b*) *Asthma* may be induced by *pressure on the vagus* trunk, by thyroid tumors or enlarged bronchial glands, following tuberculosis, measles, pertussis or rickets. (*c*) *Abdominal diseases* are not frequent causes, *e. g.,* asthma dyspepticum, uterinum, etc. (*d*) *Asthma toxicum* occurs in lead and mercurial poisoning, *uremia* and gout. (*e*) It occurs especially in weakly, anemic, nervous, scrofulous and rhachitic subjects, in the upper classes, and in males (66 per cent.) between the ages of twenty and forty (it has recently been claimed that 33 per cent. of the asthma cases develop under ten years).

The *mechanism* of the asthmatic paroxysm is explained as: (*a*) A spasm of the *bronchial muscles;* Trousseau spoke of asthma as "an epilepsy of the lungs." (*b*) Like the *exudative diatheses*, *e. g.*, urticaria, angioneurotic edema, intermittent hydrops of the joints, eczema, colica mucosa, etc. (*c*) An acute exudative bronchitis, causing bronchial spasm. (*d*) Meltzer suggests that it is an anaphylaxis. Its pathology is uncertain, as but half a dozen autopsies are recorded; the ciliated epithelium is desquamated, the bronchioles are congested and there is eosinophilic exudation.

Symptoms.—The attack *begins very suddenly,* often at night and with or without such causes as fright, cold, heat, change of residence, etc. Sometimes there are prodromes, as conjunctivitis, coryza or flatulence and sometimes the attack coincides with the menses. (*a*) *Dyspnea* is the first and the essential symptom; at first slight, it soon becomes urgent; the patient opens the windows, sits upright, grasps the arms of

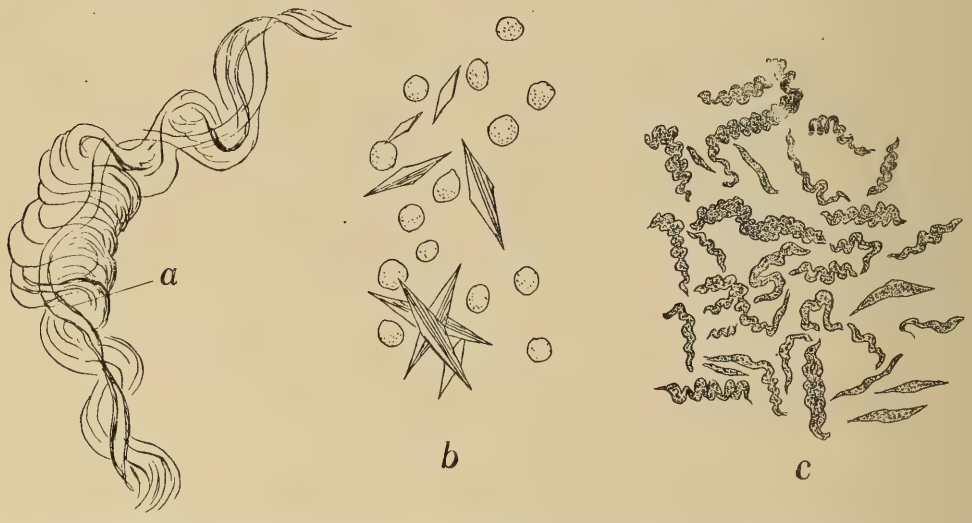
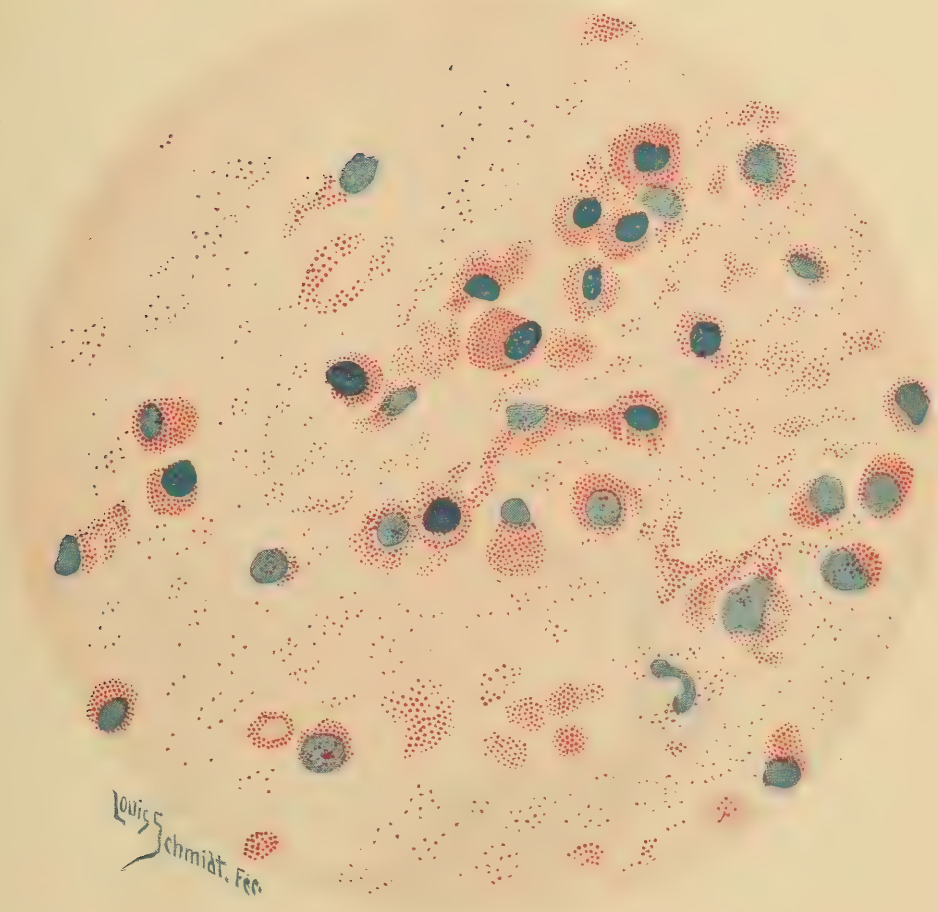


FIG. 32.—Spirals and crystals in bronchial asthma. *b*, Charcot-Leyden crystals; *a*, Leyden and Curschmann spirals magnified, and *c*, their natural size.

the chair to brace the accessory respiratory muscles and exhibits extreme distress, anxiousness and pallor, followed by cyanosis; the dyspnea is expiratory and the abdominal muscles are board-like during expiration; inspiration is somewhat, and *expiration is greatly, prolonged,* wheezing *and whistling;* respiration is generally slow. (*b*) There is extreme *cyanosis,* the cervical veins bulge out like cords, the skin is clammy, and the pulse indicates poor oxygenation by its tenseness, smallness and frequency, as also do the far rarer headache, delirium, twitchings, convulsions and coma. (*c*) The *cough is dry* and unproductive. (*d*) *Other physical signs* are as follows: On *palpation,* the vocal fremitus is found decreased by the bronchial spasm or secretion; rales are often felt. The normal *percussion* note is replaced by a *tympanitic* note, due to acute pulmonary emphysema; the voluminous lungs distend the thorax, cover the heart and depress the diaphragm, whose excursion is thus minimal. On *auscultation,* the vesicular murmur is usually faint or absent (Laennec) because of secretion in the bronchioles, and loud whistling, piping or *sibilant rales* are heard often without the stethoscope, largely during

PLATE XII



Sputum from a Case of Bronchial Asthma, showing large numbers of Eosinophilic Leukocytes and Free Granules. (Simon.)

It will be noted that the leukocytes are all mononuclear.
(Eye-piece 1, objective 1-8, Bausch & Lomb.)

expiration and due to bronchiole stenosis. (*e*) *Sputum* is first voided toward the end of the attack, as a species of crisis to the threatening, but very seldom fatal, asphyxia. A few drams of tenacious mucus, resembling egg albumen, and minute gray balls are seen. The latter are the "perles" of Laennec; when unrolled the "pearls" are seen to contain the *spirals,* best detected with the naked eye, and on a black background; they measure 2 or 3 to 10 x $\frac{1}{2}$ to 1 mm. and are *mucin casts of the bronchioles,* to which may cling leukocytes, epithelia, fat or myelin droplets; some show a central band of mucin; it is thought that they occur from the passage of mucin through the contracted bronchioles; they disappear within a day or two; spirals are also present in catarrhal or fibrinous bronchitis and pneumonia. The *Charcot-Leyden crystals* may be seen as yellow dots in the sputum; they are pointed and hexagonal (not octahedral), consist of an organic basis with phosphoric acid, and originate from the *eosinophile leukocytes* which are *increased* in the sputum and blood, constituting a quarter to more than half of the white cells; W. W. Herrick's case had 11 per cent. polynuclears, 15 per cent. lymphocytes and 72 per cent. eosinophiles. The crystals, spirals and the occasional fibrinous casts are a result, not a cause of the asthma. (Plate XII.)

The attacks last an hour or two, to be *repeated* at long or short intervals. In the hebdomadal form, attacks recur every Sunday or Monday, due to change in routine or to digestive excesses.

Diagnosis.—The diagnosis is made by its paroxysmal occurrence, the expiratory dyspnea and the transient emphysema.

Differentiation.—(*a*) All diseases causing *inspiratory dyspnea* can be at once excluded, such as edema or spasm of the glottis, paralysis of the post. crico-arytenoid muscles, tracheal or bronchial stenosis. In *glottis spasm* the spasm is inspiratory and short, the larynx moves up and down, and the epigastrium retracts during inspiration; there is no emphysema and no spirals are found. Spasm of the *diaphragm* is very rare (*v.* Phrenic Nerve); it lasts a much shorter time, the epigastrium bulges during inspiration, there is spasm of all the inspiratory muscles, hysterical stigmata are often observed and the lungs are normal. (*b*) *Expiratory dyspnea* may also occur in (i) *chronic bronchitis with emphysema,* in which the emphysema and rales are constant and eosinophilia is absent. (ii) It also occurs in *movable tumors* below the vocal cords which may turn upward during expiration, as shown by the laryngoscope. (*c*) *Asthma cardiale* is inspiratory and expiratory, and therefore readily differentiated; the "lungenstarr" or lung distention, due to engorged vessels, may simulate the emphysema of bronchial asthma (see page 368). Cardiac asthma may occur either with high or low arterial tension. Eosinophilia is absent.

Prognosis.—The prognosis is uncertain and depends on the etiological accessibility of the asthma. It is more favorable in young individuals than in those of advanced years. Cases which recurred for sixty-four years are recorded. Chronic bronchitis and emphysema are likely to develop. Death in an attack is very rare.

Treatment.—1. Of the Attack.—A hypodermic injection of *morphine* gr. $\frac{1}{4}$, with *atropine* gr. $\frac{1}{80}$ and spt. glycerylis nitratis ℿij, gives

the most prompt and reliable results. Chloral is useless, for the condition requires single doses of gr. xxx–xl, which are too large for safety. Adrenalin solution (1 to 1000) ♏x hypodermically, usually gives instant relief; it is not without danger, and the author saw edema of the lungs and doubling of the blood-pressure follow its use. It also may be sprayed into the larynx. Strong coffee, whisky, a cigar or cigarette, the application of cocaine to the nose, nitrite of amyl pearls, or a few whiffs of chloroform, sometimes give relief. The vexing question arises as to leaving morphine in the hands of the patient whose attacks often come and go without medical aid; as a rule it is dangerous. Inhalation powders, burned like incense, are very generally used. (Lobelia, powdered black tea and powdered stramonium leaves, each one ounce; soak well in two ounces of a saturated solution of potassium nitrate; dry.) The *Cigarettes d'Espic* contain the following:

Belladonna leaves	5½ parts
Hyoscyamus leaves	2¾ parts
Stramonium leaves	2¾ parts
Phellandrium aquaticum	1 part
Extract of opium	⅛ part
Cherry-laurel water	a sufficiency

All these drugs depress the peripheral filaments of the vagus.

2. Of the Tendency.—Etiological therapy helps over half the cases; cauterization of sensitive areas in the nose, even without gross lesions, is imperative. Strümpell effected 11 cures by sweating with strong lamps. The evening meal should be light, and carbohydrates and tea should be restricted; an emetic may abort an attack of the gastric type. Nervines (valerian, arsenic, bromides) may be used. The best remedy between attacks is potassium iodide, continued for months; it is combined to advantage with Fowler's solution and belladonna, not only to correct its action on the skin and secretory glands, but also for their antispasmodic effects:

℞—Liq. potassii arsenitis		ʒj
Tr. belladonnæ		ʒj
Potassii iodidi		ʒij
Fluidextr. grindeliæ (U. S. P.)		ʒvj
Aquæ	q. s. ad.	℥iv

M. et S.—One teaspoonful in water after meals.

A low level near the sea, in a semitropical climate, helps the bronchitis, emphysema and dilated right heart. General hygiene and the hardening by fresh air and cold rubs are important.

DISEASES OF THE LUNG.

EMPHYSEMA.

Definition.—A pulmonary disease, characterized pathologically by alveolar atrophy and distention, and clinically by a large chest of the inspiratory type, distended lungs, and hypertrophy and dilatation of

the right ventricle. Emphysema means a "blowing up" of the lungs. It was first described by Laennec (1826).

Etiology.—Substantive or alveolar emphysema occurs mostly in individuals over 30 or 40 years of age, in men more than in women, and in cold climates. The immediate cause is chronic bronchitis and bronchiolitis (Laennec), *catarrhe sec;* it may follow bronchial asthma, etc.; its infrequency (0.7 per cent. of autopsies) is not understood, whence a congenital hypoplasia of the elastic tissue of the lung is sometimes assumed. Coughing, blowing, as on wind instruments, straining during parturition or physical toil, artificial respiration in the newborn, and alcoholism are possibly causative.

Pathology.—On opening the chest at autopsy, the lungs are found distended (*volumen pulmonum auctum,* Traube); they overlap the heart, depress the diaphragm and narrow the mediastinum. Their surface is pale rose-colored and presents numerous small vesicles, ½ to 4 mm. in diameter, or some large bullous areas representing the fusion of several alveoli. The lungs crepitate slightly to the fingers, under which they feel, as Laennec put it, "like a pillow of down." The lungs collapse but little on section. In some places the pigment deposit is lessened—*albinism.* Emphysema is most marked on the surface of the lung, particularly over the upper lobes, anteriorly and near the spine. The normally thin edges of the lung are greatly rounded. *Histologically,* two findings are characteristic: (*a*) There is *atrophy* of the *interalveolar elastic tissue,* so that the *alveoli fuse,* corresponding to the vesicles seen on the surface; these changes account for nearly all the clinical findings in the lung. (*b*) The *capillaries* are *obliterated* by *thrombosis,* which explains the clinical hypertrophy, dilatation and ultimate failure of the right heart. The bronchi are frequently inflamed, thickened and shortened.

Pathogenesis.—The changes may be brought about (*a*) *mechanically* by forced inspiration or forced expiration, a theory favored by clinicians; or according to the pathologist, (*b*) by *nutritive alterations,* as vascular changes or atrophy of elastic or muscular tissue; calcification of the costal cartilages is probably more sequential than causal.

Symptoms.—Symptoms fall under two headings: (1) Expiratory insufficiency of the lungs, due to their distention (from the loss of elastic tissue); and (2) cardiac insufficiency, resulting from obliteration of the vessels in the lesser circuit.

1. Expiratory Pulmonary Insufficiency.—Normally expiration is wholly passive, resulting from the elasticity of the lungs and collapse of the chest. Expiration in emphysema is incomplete, as shown by the *cyrtometer* (showing the chest more circular); the *spirometer,* showing a decrease of 20 to 60 per cent. in the normal vital capacity (2000 to 4000 c.c.); and *mensuration* giving a reduction of the normal expansion of 2 to 3 inches. Upon *inspection,* dyspnea is the most conspicuous finding; it is expiratory and is due to the loss of lung elasticity and consequent impairment of lung circulation and oxygenation; it is enhanced by exertion, bronchial catarrh, accessions of true asthma and by cardiac insufficiency. The respiration-rate is increased to twenty-five or thirty and

the type is costal. The *facies* and *habitus* are most characteristic; the eyes are somewhat prominent, the nose is bluish and thickened, the head is thrown back and the chest forward to give play to the accessory muscles of respiration, and the skin is cyanotic. A network of dilated veins is often seen over the lower chest, resulting from venous obstruction. The jugular veins fill enormously on expiration and often pulsate during the diastole. The neck is short, and the hypertrophied sternomastoids, scaleni and trapezii protrude rigidly. The back and abdominal muscles are rigid, and thus supplement the inadequate action of the diaphragm. The shoulders are elevated, though stooped; the chest is of the permanent inspiratory type and is usually *barrel-shaped* (from the elastic atrophy), rarely of the paralytic type; the anteroposterior, oblique and sometimes the transverse dimensions of the chest are increased. The raised clavicles accentuate the supraclavicular spaces in which the apices sometimes protrude, on coughing, as hernial tumors. The angle of Louis is prominent and the interspaces are wider in the upper, and narrower in the lower, parts of the chest, and retract with each inspiration. The lower thorax shows a groove indicating the attachment of the expiratory muscles. The play of the diaphragm is limited, as shown by the *x*-rays and by the absence of Litten's sign. *Palpation* detects the rigidity and dilatation of the thorax, its poor excursion, the hard muscles, the calcified costal cartilages and decreased vocal fremitus. The edge of the liver may be found depressed and tender but the spleen is seldom palpable. *Percussion,* absolutely essential to a diagnosis, gives a note which is loud and deep—hyperresonant—or on the sides and back is actually "*box-like*" or tympanitic, also described as wooden and in extreme cases as dull (Skoda). The voluminous lungs narrow or wholly cover the cardiac dulness, depress the upper level of the splenic and hepatic dulness, invade Traube's space, and show little respiratory excursion. At the back the lungs may reach the twelfth dorsal or second lumbar vertebra. Upon *auscultation,* expiration is found greatly lengthened so that it is to inspiration as 2 or 4 to 1, the converse of normal. Vesicular breathing is absent, or the breathing is indeterminate, even when the stethoscope rises and falls. Rales result from bronchitis or stasis; a pearly mucin with few cells may be evacuated after coughing.

2. Cardiac Insufficiency.—The work of the right ventricle is increased because the pulmonary arterioles are in part occluded, and the imperfect expiration allows neither free aspiration of venous blood into the auricles nor adequate filling of the aorta. The *right ventricle* therefore *hypertrophies,* mostly about the conus, causing the accentuated second pulmonic tone. The diaphragm is low, whence the hypertrophied and more horizontal heart beats in the epigastrium. *Dilatation* soon follows and is accompanied, in advanced cases, by tricuspid leakage, nutmeg liver, congested kidneys, and other signs of cardiac insufficiency. The cardiac alteration, so marked at autopsy, is obscured during life, as the lungs cover the heart and muffle its tones. Functional heart murmurs, mostly systolic, result from myocardial degeneration or relaxation.

The *clinical course* often covers decades. Patients may acquire emphysema in childhood and live to 60 or 70 years of age.

Diagnosis.—Developed types are easily recognized by the barrel-chest, its hyperresonance, the low borders of the lungs, their lack of excursion, the obscuration of cardiac dulness, the expiratory dyspnea and cardiac stasis.

DIFFERENTIATION.—(*a*) *Acute distention of the lung* or acute emphysema, as after asthma or pertussis, cannot be distinguished by one examination. It is seen repeatedly in bronchial stenosis due to aneurysm. It regresses rapidly even after months, when its cause is removed. (*b*) *Vicarious or compensatory emphysema* often develops in one lung when contralateral pleurisy or cirrhosis of the lung exists; it may occur on the same side with the lesion; marked apical emphysema almost certainly indicates some deeper induration. (*c*) *Emphysema senile*, Jenner's "small-lunged" emphysema, is merely atrophy of the connective tissue; the lungs are not enlarged, but smaller, the diaphragm stands higher, the cardiac dulness is increased, the chest flattened, the ribs oblique, the respiratory muscles wasted, the right heart is not hypertrophied and respiratory excursion is retained. (*d*) *From interalveolar interstitial emphysema* the first differential point is the etiology; (i) trauma, external or internal (in tracheotomies), and frequently with pneumothorax. (ii) Foreign bodies. (iii) Ulceration of the air passages, gangrene, abscess or tuberculosis and less often gastric ulcer or cancer. Other etiological factors are (iv) forced entrance of air, as in resuscitation of the newborn; (v) severe coughing efforts, as in whooping-cough; (vi) severe pressure efforts, as in parturition; and (vii) spasm of the glottis. The second point is a series of symptoms often present, as creaking in the mediastinum, sometimes systolic; emphysema and crepitation of the cervical cellular tissue, bulging of the interspaces, creaking and tympany at the edge of the ribs; absence of the vesicular murmur; and obliteration of the heart dulness. It is usually fatal. (*e*) In *congenital hypertrophy* of the lung (*pulmo excessivus*) the lungs are very voluminous, but their excursion, auscultation and percussion-note are normal. (*f*) *Pneumothorax* (*q. v.*) is unilateral, has a more tympanitic note and is associated with succussion, tinkling rales and other pathognomonic signs.

Prognosis.—Recovery is unknown in the genuine substantive form. Transient forms often or usually regress completely. Life, even relative comfort, may be sustained for years or decades. Much depends on associated conditions, as frequent severe bronchitis, chronic nephritis, arteriosclerosis or myocarditis. Extreme forms impede the flow of chyle which, with dyspnea and fatigue, impair nutrition. The most grave complication is failure of the right heart upon which compensation devolves.

Subjects of emphysema show no predisposition to pulmonary inflammation. The doctrine that tuberculosis (Rokitansky) or valvular disease (Bouillaud) excludes emphysema is incorrect.

Treatment.—(*a*) *Prophylaxis* concerns chiefly the concomitant or causal bronchitis. Severe exercise is to be avoided. (*b*) *Coincident gastric, renal* or gouty disease must be treated. (*c*) *Climate* is as important a factor as in chronic bronchitis, the inseparable associate of emphysema; high altitudes are avoided, as they directly promote emphysema. (*d*)

Pneumotherapy is sometimes beneficial. It includes inhalation of thinned air, or, where there is marked catarrh, of compressed air. Two or three treatments of ten minutes should be given daily, and the time should soon be extended to half an hour. (*e*) *Chronic bronchitis* (*q. v.*), and (*f*) *asthma* (*q. v.*) are important considerations in treatment. (*g*) *Rhythmic compression* of the chest, three or four times daily, should be practised during 20 to 30 expirations; it increases the circulation in the lung. (*h*) *Cardiac insufficiency* is treated as in valvular or myocardial disease (*q. v.*) and strychnine, venesection and other measures may be used. (*i*) Freund's operation for calcification of the first chondrocostal joints, has been done in over 50 instances; in some cases, striking benefit is at once apparent but chondrotomy has narrow indications. The therapy is discussed briefly, as no measure can restore lost elastic tissue or patency to obliterated vessels.

BRONCHOPNEUMONIA.

This affection was first well described in 1840, by Rilliet and Barthez, who separated it from lobar pneumonia and recognized it as a secondary disease. It might be well classed among the infections, except that unlike fibrinous pneumonia, it is not due to a specific microörganism.

Definition.—Bronchopneumonia is defined with difficulty; (*a*) *etiologically* due to various microbes, it is generally secondary to the bronchiolitis of some specific infection, chiefly in children; (*b*) *pathologically* it develops about the bronchioles (bronchopneumonia) and in a few lobules (p. lobularis), rather than throughout a lobe; its exudate is rather catarrhal (p. catarrhalis) than fibrinous, and its distribution is multiple or disseminated (p. disseminata); (*c*) *clinically* it is identical with capillary bronchitis, which, in the first years of life, almost never spares the lung tissue; it is marked by fever, cough, dyspnea and cyanosis, often without signs of hepatization.

Etiology.—(*a*) Some cases, especially in children under two years, are *primary;* these are estimated by Holt and Connor at 35 per cent., by others at a much lower figure; (*b*) most cases are *secondary* to infections affecting the upper air passages; 32 per cent. follow measles; 24 per cent. pertussis; 16, diphtheria; 15, macrobronchitis; 7, ileocolitis; 3, scarlatina; 2, influenza; 0.5 per cent. each varicella and erysipelas. (*c*) *Predisposing causes* in the young are poor sanitation, malnutrition, syphilis, tuberculosis and rickets. Bronchopneumonia causes 9 per cent. of deaths in foundling asylums. (*d*) *Age.* Most cases are observed in children two or three years old. Seventy-five per cent. of cases of pneumonia in children under five years of age are lobular (Holt). Less frequently in weak, cachectic, nephritic and aged persons it may cause death. (*e*) In some cases microbes may act directly on the lungs, as in the primary cases in children, in the influenza of adults, and in aspiration or inhalation pneumonia. "Schluck pneumonie" is prone to develop in diseases in which the laryngeal sensibility is decreased, as in diphtheritic or bulbar palsies; in ether anesthesia in which the germ-laden saliva is drawn into the lungs; in comatose states, as apoplexy or uremia; in mental diseases; in the new-

born; in partial drowning; in operations on the throat; or when there is persistent vomiting. (*f*) It is probable that *infection* may occur by the *lymph or blood stream.*

Bacteriology.—Bronchopneumonia has no specific bacteriology. *In adults* one-third of the cases show more than one organism, the pneumococcus ranking first and the streptococcus second; and two-thirds show one germ, of which the pneumococcus constitutes 39, the streptococcus 31, Friedländer's bacillus 23, and the staphylococcus 7 per cent.; *in children* mixed infection is present in half the cases, the streptococcus predominating, while in the other half there is but one organism, the pneumococcus being the most common. Other germs found are the influenza, typhoid, tubercle and colon bacilli, Pfeiffer's Micrococcus catarrhalis, tetragenus and the meningococcus. The tubercle bacillus is often associated with the pneumococcus.

Pathology.—The foci of consolidation are usually diffused in the lower and sometimes in the upper lobes, posteriorly and usually bilaterally. They vary in size from a pin-head to a walnut or larger; most foci lie near the pleura, through which they may be seen and felt; the pleura is often granular or fibrinous. On section the consolidated nodes appear reddish-brown and later yellowish from fatty degeneration of the cells; they are firm, airless, whence they sink in water, and on section are generally smooth and glistening, though some pneumococcus infections show the granulation and viscid secretion of genuine fibrinous pneumonia. The bronchioles supplying the consolidated nodes are plugged with mucopus, so that air cannot be forced through them; the bronchioles are reddened, swollen or ecchymotic. Though atelectasis is frequent its importance has been overestimated. In fortunately made sections a dendritic or grape-like arrangement may be seen in which the bronchioles correspond to the stems and the alveoli to the leaves or grapes. *Microscopically* the bronchioles show dilated vessels and leukocyte emigration, the alveoli show epithelial desquamation, serous exudation, leukocyte outpouring and a scattering of red cells; the alveolar walls show interstitial inflammation. Though influenza pneumonia is strictly cellular (*catarrhal*), aspiration, pneumococcic and other forms not infrequently show a fibrin network, though decidedly less than in lobar types. Near the foci and in the upper lobes compensatory emphysema appears. *Extension occurs* from alveolus to alveolus or along the peribronchial tissues. In some forms numerous lobular foci fuse so that the greater part of a lobe consolidates; this pseudolobar form is often due to the pneumococcus, is seen in measles and diphtheria and clinically may closely simulate lobar pneumonia, though pathologically areas of collapsed, vascular or sound tissue lie between the bronchopneumonic patches.

Symptoms.—The clinical picture varies considerably. *In the primary form,* most often pneumococcic, the onset is brusque, with chill, vomiting or pain in the side. The fever rises suddenly, and often falls by crisis. Capillary bronchitis is absent, consolidation is often marked, the course short and the death-rate low. In adults this form is frequently unilateral. The *more common secondary form* begins insidiously, if it follows

a microbronchitis; it is often obscured by the disease it complicates. The fever gradually rises to 103° or 104°, and is irregular or remittent, rising with each new focus; fever in bronchitis seldom exceeds 102° and higher registrations suggest pneumonia; it may be low or absent in marantic subjects. There may be no physical findings, or they are frequently indeterminate. If recovery ensues, lysis is the usual solution.

Subjective and General Manifestations.—These are often *more obvious* than the objective pulmonary symptoms. The pulse rises to 120 or even 180 and in convalescence is more tardy in reaching normal than the fever. The respiration is often 60 to 80, the expiration is frequently catchy or grunting, and the respiration-pulse ratio, normally 1 to 4, becomes 1 to 3 or 2; dyspnea, restlessness and irritability develop early. Cyanosis arises from impaired flow of blood due to continued coughing and stagnant secretion. Pain is not as common as in lobar pneumonia. The cough becomes drier and more distressing; the sputum is usually swallowed in patients under seven years of age, but if obtained shows nothing characteristic. Meteorism is not uncommon in severe or moribund cases.

Lung Findings.—On *inspection,* irregular excursion is sometimes seen. The accessory muscles of the nose, neck, chest and abdomen are in full play. The interspaces, ribs, sternum and epigastrium in young plastic chests retract greatly with each inspiration. *Palpation* may show increased fremitus in a focus near the surface and measuring 2 x 5 cm. Stagnant secretion in the air tubes may temporarily suspend fremitus. *Percussion* is often negative for a few days or even throughout the course, as deep, isolated foci cannot produce dulness. In the thin, infantile thorax, special importance should be attached to light palpatory percussion. Dulness obtains over a focus having an area of 2 x 5 cm. It is most often found along the spine over the lower lobes. Dulness is most marked in the pseudolobar (confluent) forms. Atelectasis may cause dulness but not bronchial breathing. A hyperresonant note in front indicates emphysema; behind it shows relaxation. *Auscultation* reveals subcrepitant or crepitant rales, often heard over an area not exceeding the bell of the stethoscope. Bronchial breathing and bronchophony are fairly frequent, and occur especially when the child cries. In other cases the breathing may be rude or puerile.

Special Types.—Special types have been considered under measles, pertussis, etc. The following classification gives the clearest understanding of the physical findings: (*a*) *In capillary bronchitis* there is no sign of consolidation. Sibilant rales, coarse and fine crepitant rales are spread over one or both lungs. Sibilant rales are due to swelling of the mucosa of the larger and smaller tubes. A feeble respiratory murmur indicates areas of congestion. (*b*) In *disseminated bronchopneumonia,* characterized by small areas of consolidation, percussion usually gives negative results, or very slight dulness, and the fremitus is not altered, since the consolidated areas are deep in the lung, or too small to alter the percussion-note. Auscultation reveals crepitant rales and bronchovesicular breathing over the consolidated areas. Vocal and crying resonance is exaggerated. (*c*) In *acute generalized broncho-*

pneumonia, the percussion-note is dull over a large area, even an entire lobe, with increased vocal fremitus, bronchial breathing and fine moist rales, while the rest of the lung shows bronchitis.

Issues.—*Death* may occur from carbonic acid narcosis, irritability passing into apathy, the harassing cough becoming less marked and allowing stagnation of bronchial secretion ("suffocative catarrh"), eventuating in heart failure. *Complete resolution* is the rule in cases which recover. *Delayed resolution* with remissions and exacerbations may occur. *Complications* may develop; (*a*) pleurisy is the most frequent; (*b*) gangrene; (*c*) abscess, especially in aspiration and influenzal forms; (*d*) pulmonary induration; (*e*) bronchiectasis; and (*f*) tuberculosis, less often a sequel than awakened by the new infection.

Diagnosis.—Reliance on physical signs alone leads to many errors and a diagnosis is often determined only by the symptoms, sudden high fever, mild fever suddenly rising during bronchitis, dyspnea, rapid respiration, cyanosis, etc. In the very young or marantic and aged the symptoms may be very atypical. *Lobar pneumonia* (*q. v.*, variations in children) occurs mostly in sound children over three years of age, runs a cyclical course, ends by crisis, is unilateral and involves one or more lobes; while bronchopneumonia occurs more often in younger weakly children, often after bronchiolitis, runs a longer, more atypical, unfavorable and relapsing course, resolves by lysis, is bilateral and is less marked by definite pulmonary findings. *Tuberculosis* is often distinguished only by the course; it invades the upper lobes more and bacilli-laden sputum may be recovered by washing the stomach or swabbing the throat after the child coughs. *Pleurisy* (*q. v.*) is diagnosticated by the aspirating needle.

Course and Prognosis.—The *acute course* lasts a week, of which the measles pneumonia is the prototype; the *subacute,* lasts two to eight weeks, exemplified by pertussis pneumonia, and the *chronic,* covering months, as tuberculous bronchopneumonia.

The *prognosis* depends on the patient's social situation, age, the disease which the pneumonia complicates and its extent. The outlook is serious, especially in the secondary forms (measles and pertussis); in the very young it is almost as fatal as infantile diarrhea; aspiration forms are very grave, as are those in marantic and aged subjects. In private practice the death-rate ranges from 10 to 33 per cent., in hospitals 33 to 50 per cent., and in asylums even 50 to 100 per cent.

Treatment.—1. Prophylaxis—(*a*) In nasal or pharyngeal infections the nose and throat should be frequently cleaned with alkaline antiseptics. (*b*) In laryngeal or bronchial inflammation, congestion and atelectasis are avoided by frequent and regular change of posture. It is a great advantage to avert influenza, pertussis and measles, in the first three years of life. (*c*) Deep breathing is enforced by affusions of cold water on the neck and thorax. (*d*) Exposure should be avoided by firmly fastening the nightgown to the foot of the bed.

2. Therapy.—Actual treatment is symptomatic. (*a*) The temperature of the room is maintained at 67° to 70° with constant *moderated ventilation.* (*b*) The diet should be *fluid,* diluted or predigested; egg

albumen, milk and plenty of water are indicated. At the onset calomel gr. $\frac{1}{10}$ every hour for five doses, is followed by castor oil. (*c*) *Expectorants,* as ammonium carbonate, tartar emetic or ipecac, derange the digestion, which is of greatest importance. Aromatic spirits of ammonia, ♏v–xx every three or four hours, is the least deranging, and stimulates. (*d*) *Pain* rarely indicates opiates, which induce stagnation of secretion, notably in young, old or weak subjects; dyspnea, pain, restlessness, cough, cyanosis and rapid pulse may be relieved by paregoric, which is preferable to Dover's powder or morphine. *Fresh air is better than drugs.* (*e*) *Local applications,* as poultices or pneumonia jackets and blisters, are useless. (*f*) In the treatment of *fever,* antipyretics, aconite, antimony and other cardiac depressants must be avoided; cool applications are equally efficacious; they also lessen tympany and carbon dioxide narcosis, stimulate the heart and brain centres and deepen respiration, but they must be used with care in very young and marantic subjects; in such cases a warm sponge or bath may be given, with cold affusions to the neck (*v. s.*). (*g*) For *cardiac stimulants,* brandy in daily doses of ℥ss–iij, very well diluted in water or peptonized milk, aromatic spirits, digitalis, saline infusions or enemata, camphor or strychnine may be used. (See page 269 for dosage.) (*h*) Respiratory stimulants include tr. belladonna, oxygen, thoracic faradization and mechanical compression of the chest. (*i*) *Emetics* are unreliable; ipecac has no effect in carbon dioxide intoxication, whence it is given with coffee, camphor or cognac. Phlebotomy is inadvisable. (*j*) *Inhalations of steam* (*v.* DIPHTHERIA and CROUP). (*k*) *During convalescence* a sojourn in the country or in a warm climate is advisable.

INDURATIVE PNEUMONIA, LUNG CIRRHOSIS, FIBROID PHTHISIS.

Definition.—An overgrowth of the pulmonary connective tissue.

Etiology.—1. DISEASES OF THE LUNG.—These may induce *local,* lobular or lobar, unilateral or bilateral fibrous induration. Local induration frequently occurs about tumors, tubercles, gummata, abscess, gangrene, etc., and is of no importance, except as it demarks dangerous foci of disease. *Diffuse* induration is important. (*a*) *Fibrinous pneumonia* sometimes results in lobar induration (0.7 per cent.), notably in marantic subjects from delayed resolution; the lung becomes airless and dense from fibrous tissue within the alveoli whence it involves contiguous structures; on section its surface is smooth, and the connective tissue is strikingly translucent. (*b*) *Bronchopneumonia* in measles, pertussis or influenza, sometimes leads to induration, beginning from an alveolitis or peribronchitis. (*c*) *Foreign bodies* and *aspiration pneumonia;* (*d*) *chronic tuberculosis;* and (*e*) syphilis, gangrene, etc., are etiological factors.

2. BRONCHIAL AFFECTIONS.—(*a*) *Bronchiectasis,* noted by Corrigan, who first used the term lung cirrhosis; (*b*) *bronchial stenosis,* as by aneurysm or gumma; and (*c*) *pneumokoniosis* and putrid and chronic bronchitis may cause indurative pneumonia.

3. Pleural Affections.—In Charcot's "pleurogenous" interstitial pneumonia, strands of connective tissue invade and cirrhose the lung, converging toward the hilum.

Pathology.—The pathology of an affection with such numerous causes can scarcely be described. The early induration is reddish, and the older connective tissue is darker, slate-colored or gray. The new connective tissue may be formed in the bronchi, septa, alveoli, bloodvessels or pleuræ. The lung is airless and dense; is cut with difficulty and on section presents either a granulated or a smooth surface, the latter in cases following lobar or aspiration pneumonia and sometimes gangrene. The lung shrinkage produces changes in the mediastinum, heart, diaphragm and sound lung (*v. i.*).

Symptoms.—Symptoms in incipient or localized lesions may be *inseparable from the causal affection* (tuberculosis, bronchiectasis) or may follow closely upon it (pneumonia, pleurisy). There may be a low fever, cough, dyspnea on exertion, pain in the side, rapid pulse, cyanosis, anemia, malnutrition and night sweats.

Physical findings in the typical chronic case are as follows: (*a*) On *inspection* the interspaces are narrowed on the affected side, the nipple and scapula are nearer to the median line, the shoulder is lower, the spine is convex toward the sound side and the measurement is unilaterally decreased. In left-sided induration the lung retracts from the heart, increasing its pulsation and disclosing the pulmonary artery, over which may be seen and felt its systolic filling and the strong diastolic closure of the pulmonary valves; in right-sided induration the heart beat may appear to the right of the sternum or may be covered up by the compensatory emphysema of the left lung. (*b*) *Palpation* corroborates these findings, and, as a rule, shows increased vocal fremitus. The second pulmonic tone is much accentuated, corresponding to the routine hypertrophy of the right ventricle and the occasional atheroma of the pulmonary artery. (*c*) *Percussion* elicits dulness below, and sometimes a slightly hyperresonant note above, the indurated area; the apex of the upper lobe is on a lower level and the diaphragm stands higher (the liver rising or Traube's space increasing); there is no respiratory excursion. (*d*) *Auscultation* brings out the absence of breath sounds below, and bronchial breathing and subcrepitant, crepitant and bubbling rales above. Additional signs result from the right heart hypertrophy and dilatation, from coincident bronchiectasis or cavity formation on the same side, and compensatory emphysema on the opposite side. There is nearly always some sputum.

Diagnosis.—Diagnosis is more easily made in chronic than in the subacute postinfluenzal or postpneumonic induration which may suggest tuberculosis or interlobular suppuration. Slow-growing tumors of the lung may cause confusion. The *x*-rays are helpful.

Prognosis.—The prognosis varies with the etiology. Ulcers from arterial occlusion or infected bronchial secretion, explain the frequent lung hemorrhage (in 50 per cent.) and its occasional role as a cause of death. Amyloidosis sometimes results but death is usually caused by right heart failure.

Treatment.—Life may be prolonged for decades under proper hygiene. Exercises with the arms may check the early shrinking, but nothing can remove an established induration. Warm climates at the sea level or at slight elevation are beneficial. Some writers hold that turpentine checks induration.

PNEUMOKONIOSIS.

Zenker employed the term to include various indurative diseases of the lungs due to "dust" inhalation.

Etiology and Pathology.—1. *Anthracosis* is due to inhalation of carbon, coal dust, soot, charcoal or graphite, and is known as "coal miner's disease" or phthisis melanotica. (*a*) Moderate amounts of carbon inhaled into the bronchi are absorbed by the leukocytes, worked upward by the ciliated epithelial cells and expectorated. (*b*) Larger amounts penetrate the bronchial mucosa to the connective tissue, or enter the lymph stream whence they reach the small lymph nodes around the vessels and bronchi or mediastinal lymph glands. A moderate pigmentation occurs in all city inhabitants, while the lungs of countrymen are pink. (*c*) Very large amounts reach the alveoli, when the lungs are ink-black; *connective tissue proliferation* develops in *insular foci* (endoperialveolitis nodosa and endoperilymphangitis fibrosa); the foci occasionally measure 5 x 15 cm., on section are hard and exude an inky fluid. *Induration* involves the *mediastinal lymph glands,* and often extends beyond them (perilymphadenitis); by this latter process the glands may become adherent to, and rupture into, the pulmonary veins, whence pigment-metastases into the liver, spleen, kidneys and mesenteric glands are not infrequent. Bronchial perilymphadenitis may also lead to *diffuse induration,* mediastinopericarditis, stenosis of the trachea, bronchi, esophagus, cava, vena azygos and pulmonary artery, to esophageal diverticulum, fatal hemorrhage into the pericardium or bronchi when the aorta is also eroded, to aspiration pneumonia by intrabronchial rupture or to recurrent laryngeal paralysis by pressure. Lehmann holds that the great bulk of dust and similar bodies find their way into the system by absorption from the digestive tract.

2. *Siderosis pulmonum* (Zenker) is a similar change, due to inhalation of metallic dusts, as iron, brass or bronze; there are the same interstitial deposit, lung induration, bronchial gland cirrhosis and metastases. The irritation is greater, nodes are more frequent and induration is more intense than in anthracosis.

3. *Chalicosis pulmonum* (Peacock) is due to inhalation of alumina, quartz or sandstone, known as "stone-cutter's or millstone-maker's phthisis," "grinder's rot" or "potter's asthma." Calcium is less irritating than silica or quartz. Of all forms, the most nodules and induration occur in chalicosis.

4. Similar changes are observed from inhalation of wool, flax, cotton, grain, tobacco, glass, porcelain, pepper, cinnamon, bone, lead, mercury, phosphorus, zinc, arsenic and horn.

Symptoms.—After many years, the symptoms of *chronic bronchitis, asthma, emphysema* and finally of *lung cirrhosis* appear. The *sputum*

contains coal dust, soot, charcoal particles, as in Traube's celebrated case, and yellow oxide or dark phosphate of iron, quartz and silica. *Cavities* may form from bronchiectasis, softening of the nodose indurations or tuberculous complications; in some cases lung stones, set free by ulceration, may be raised. Tuberculosis occurs in 13 per cent. of anthracosis and in 44 per cent. of chalicosis. Scissor-grinders rarely live beyond forty years of age and blasters in mines usually succumb in four years.

Treatment.—Treatment is largely prophylactic and that of the concomitant bronchitis and emphysema.

ATELECTASIS.

Apneumatosis is a congenital or acquired "absence of air."

I. Congenital atelectasis was first described by Jörg (1834).

Etiology and Pathology.—This variety is due to deficient inspiratory efforts from weak muscles, insufficient stimulation of the bulbar centres and aspiration of mucus or meconium. The base, anterior margin and lingual lobe are most involved and the distribution is lobular. If the subject lives, the atelectatic portions do not become pigmented. These airless areas are sunken, bluish, lax, do not crepitate and sink when put in water. Bronchiectasis may result; the ductus Botalli and foramen ovale are usually patent, the right heart is dilated and thrombosis is frequent in the brain sinuses, kidney and right heart.

Symptoms.—The newborn with atelectasis breathe superficially and cry but little. Inspiration is marked by retraction of the epigastrium, subclavicular and intercostal spaces, for the lungs cannot follow the inspiratory distention of the thorax. If the undistended area measures 2 by 5 cm. it may be dull and increased fremitus with bronchial breathing may be detected, but more often the breathing is weak or entirely absent. Carbon dioxide narcosis develops, the pulse becomes small, the sensorium becomes dulled and, without active intervention, asphyxia causes death. If the patient lives, the chest is peculiarly deformed, its lower parts being sunken, so as to show the ribs.

Treatment.—Removal of meconium and mucus by swabbing or aspiration, artificial respiration, swinging, and warm baths with cold affusions to the neck and chest are indicated.

II. Acquired Atelectasis.—Acquired atelectasis was first described by Legendre, Bailly and Traube.

Etiology.—The lungs, already distended and ventilated, collapse and the air is absorbed. (*a*) The *marantic* form occurs in bed-ridden individuals reduced by typhoid, infantile diarrhea or cachectic conditions. (*b*) *Obstructive* collapse develops most frequently in bronchopneumonia, as the bronchioles in the young are narrow, or in any stenosis, bronchial, etc. (*c*) *Compressive* apneumatosis follows compression by aneurysm, dilated heart, tumors, exudates, pneumothorax, abdominal tumors, kyphoscoliosis, etc.

Pathology.—Pathologically the same general findings obtain as in the congenital type. The lung is reddish-brown or bluish. Hyperemia

may cause a flesh-like appearance (carnification), or serous transudation may cause a spleen-like aspect (splenization). There may be coincident inflammation or vicarious emphysema. Long-standing atelectasis results in bronchiectasis, fatty degeneration of the alveolar epithelium and hyperplasia of the interalveolar connective tissue, known as collapse induration.

Symptoms.—Symptoms are easily obscured by the causal pleural effusion or bronchopneumonia, with whose foci it may be confused. Inspiratory retraction, lessened vesicular breathing, a slightly tympanitic note from relaxation of the collapsed lung and crepitant rales are common. Increased vocal fremitus and bronchial breathing are uncommon. Dilatation of the right heart is usual. In recent cases the *physical signs disappear after a few hours of change of posture, or after deep breathing,* which, with absence of fever, pain and cough, effectually differentiates from infarct, pneumonia or effusive pleurisy. After sleep for some hours on the back, a few crepitant rales may be heard physiologically over the lower posterior parts for three or four inspirations.

Prognosis and Treatment.—The prognosis and treatment vary according to the cause. In all fevers and all respiratory inflammations the posture must be changed frequently; orders to this effect should be written, and the change should be recorded on the history sheet. Cold affusions, hydrotherapy and cardiants are indicated.

ABSCESS OF THE LUNG.

Suppuration may occur in a sound or diseased lung.

Etiology.—(*a*) *Lobar pneumonia* causes 62 per cent.; upper lobe localization, alcoholism, pneumonia complicating emphysema and lung induration and hemorrhagic types are the predisposing factors. (*b*) *Lobular pneumonia,* especially the influenzal type, may suppurate. (*c*) *Aspiration pneumonia* is causal in 18 per cent., particularly in insane or delirious subjects; foreign bodies, food, saliva or pus from lesions in the upper respiratory tract, vomiting, esophageal cancer rupturing into a bronchus, are its usual causes. (*d*) *Embolic suppuration* produces 17 per cent. (*e*) *Trauma* is another factor. (*f*) Abscess may complicate tuberculosis, bronchiectasis, echinococcus, etc. (*g*) *Rupture into the lung* of contiguous suppuration, such as empyema, suppurating peribronchial glands, subphrenic abscess, spinal caries and gastric ulcer, causes 3 per cent. of lung abscesses. Most cases occur in middle-aged males.

Bacteriology.—The staphylococcus is most frequently the causative microörganism; the streptococcus, pneumococcus, pneumobacillus, influenza bacillus, colon bacillus and leptothrix are also found.

Pathology.—The cavity varies from the size of a hazel-nut to an orange; it may occupy even an entire lobe or lung. The abscess is usually single, except in the embolic form. Its shape is irregular and it is sometimes multilocular. Its inner surface is irregular with floccular mural shreds, and is walled with pus-secreting granulations, which in some cases fill and obliterate the cavity. Fibrous tissue develops about the cavity, and

in chronic abscess becomes dense and scar-like. The cavity is usually near the lung's surface, whence the frequency of complicating pleurisy; 80 per cent. of lung abscesses are in a lower lobe (Tuffier).

Symptoms.—The symptoms vary with the causal lesion; in lobar pneumonia the crisis is delayed and resolution is incomplete; in influenza the respiratory symptoms and fever persist; in sepsis, multiple lung involvement perpetuates the septic process. (*a*) *The sputum* is purulent, offensive or rancid without putridity, alkaline and may measure a pint to a quart daily. It separates, on standing, into two layers, a granular sediment and a serous upper layer, sometimes with frothy surface. In metapneumonic cases the sputum is sometimes grass-green. The sediment reveals *shreds of lung tissue* (Traube and Leyden) and yellowish-green or gray flocculi; these are absolutely essential to diagnosis and consist of elastic fibers to which alveolar epithelium adheres. *Hematoidin* crystals or platelets, short sheaths of *fatty crystals*, *bacteria* (*v. s.*) and in chronic cases *cholesterin* and scar-like connective tissue are also found. The "mouthful expectoration" is voided periodically, as from cavities of other causation. (*b*) The *decubitus* varies with the location of the cavity; the patient assumes the erect posture if the cavity is in a lower lobe, to prevent leaking of the secretion into the bronchi. (*c*) *Signs of cavity* are found if the exulceration is large and superficial, and in 80 per cent. occur in the lower lobes. They are essentially those of tuberculous or bronchiectatic cavities (*q. v.*) and vary with the stagnation or evacuation of the pus; thus a tympanitic note, bronchial or amphoric breathing and increased vocal fremitus are found when the *vomica* is empty, and dulness, weakened breath sounds and decreased fremitus obtain when it is full. The *x*-rays also serve to localize, but when the findings differ the physical signs are more trustworthy. (*d*) *Hectic fever*, sweats, rigors and leukocytosis occur from resorption.

Diagnosis.—The etiology and cavity formation are often, and the sputum always, characteristic. (*a*) *Abscess breaking into the lung* from the spine or the subphrenic or pleural spaces is sudden, sometimes suffocative in onset, and the hematoidin crystals and shreds of pulmonary tissue are absent. (*b*) *Gangrene* (*v. i.*). (*c*) *Tuberculosis* usually occurs in an upper lobe; abscess in a lower one; elastic fibers and hematoidin crystals are more abundant in abscess, and tubercle bacilli are found in tuberculosis. (*d*) In *bronchiectasis* elastic fibers are seldom voided.

Prognosis.—Spontaneous recovery may follow granulation and cicatrization, especially in lobar pneumonia, where recovery is the rule. In influenzal or aspiration pneumonia or in sepsis the outlook is far less favorable; marasmus, hectic fever, amyloidosis, lung induration, empyema, pneumothorax and pericarditis are frequent issues.

Treatment.—(*a*) The *general strength* should be maintained by a full diet, tonics and alcohol. (*b*) *Decomposition*, if present, should be modified as in bronchiectasis or gangrene, by administration of creosote or turpentine internally. (*c*) *Surgical intervention* is indicated (i) when the abscess is large, shows no tendency toward spontaneous cure and the contents decompose; (ii) when rupture into the pleura occurs; and

(iii) when the abscess results from the breaking into the lung of a contiguous focus of suppuration. The mortality is 25 per cent. under surgical, and 54 per cent. under expectant, treatment.

GANGRENE OF THE LUNG.

Definition.—A necrosis and mortification of lung tissue. Gangrene, though more common than abscess, is found in 0.1 per cent. of clinical cases only and in 1.6 per cent. of autopsies.

Etiology.—Reduced physiological resistance, *e. g.*, from diabetes, is a necessary factor. *Lobar pneumonia* may terminate in gangrene when, as in abscess, the patient is weakly, diabetic, addicted to alcohol or suffering from putrid bronchitis; though its frequency is but 0.4 per cent., pneumonia causes 23 per cent. of lung gangrene. *Lobular pneumonia* is less frequently causal, though grippe pneumonia may terminate in gangrene. In Coupland and Hensel's combined cases, 14 per cent. were due to *pulmonary tuberculosis* and 10 per cent. to *tumor of the lung. Aspiration pneumonia* resulting in gangrene occurs particularly in insane, delirious, paralyzed or emaciated subjects; foreign bodies, necrotic diseases of the throat, larynx or bronchi, bronchiectasis, putrid bronchitis and bronchial stenosis promote gangrene. *Embolic gangrene,* a cause of 19 per cent. of lung gangrene, may result from suppurative gangrenous lesions in the appendix, uterus or extremities. *Rupture into the lung,* as of esophageal cancer or diverticulum, putrid pleurisy, subphrenic abscess or gastric ulcer may cause gangrene. Gangrene usually occurs within the *second to fourth decades* of life and *in males.*

Bacteriology.—Some maintain that certain germs produce the necrosis and other germs the gangrene, but probably one microörganism produces both changes. The staphylococcus is found, also the streptococcus, colon bacillus and "acid-fast" pseudotubercle bacillus; the B. pyocyaneus, Micrococcus tetragenus, a micrococcus which produces skatol and indol, and causes a putrid odor in the cultures, and a bacillus resembling that of malignant edema, are enumerated.

Pathology.—Genuine gangrene must not be confused with softening of the lung, due to regurgitation of the gastric juice into the lung during the death agony or after death. There is a rare non-putrid pneumomalacia, analogous to myomalacia cordis. Lung gangrene occurs in the right lung in 45 per cent., in the left in 37 and in both lungs in 18 per cent. of the cases. Laennec, who first described pulmonary gangrene, distinguished *two types,* the circumscribed and the diffuse. (*a*) *Circumscribed* gangrene occurs chiefly in the lower lobes near the surface, and oftener on the right than on the left side. The focus may attain the size of an orange. Laennec distinguished three stages: (i) The formation of a yellow, green or brown slough, which may be expectorated entire; (ii) liquefaction, or moist gangrene; and (iii) formation of a cavity which is irregular in shape, has ragged walls and contains a stinking liquid resembling the sputum of gangrene (*v. i.*). The cavity is limited by granulations and later by connective tissue, or gradually spreads, corroding more and more lung tissue, or extension may follow

aspiration of the septic matter into other lung areas. Recovery may follow localization, organization of granulations and contraction of connective tissue. Microscopically there is (i) an inner zone of offensive fluid or necrotic tissue, bacteria and emigrated leukocytes; (ii) outside of this is a zone of granulations, beyond which is (iii) congested or edematous lung. (*b*) The *rarer diffuse gangrene* has no demarkation, and a large area, an entire lobe, or even the whole lung may be discolored, stinking and macerated. The diffuse form occurs more often in the upper lobes, and on the right side. In both forms the sputum irritates and injects the bronchial mucosa, the bronchial glands are tumefied and degeneration or metastasis may occur in various viscera.

Symptoms.—1. Sputum.—The sputum is characteristic, essential to diagnosis, and absent only when the putrid focus has no bronchial connection. It is extremely offensive, fecal, stinking or garlicky. It is most offensive at the moment of evacuation, and on standing may gradually lessen. The sputum, *voided periodically* and sometimes in mouthfuls, is usually abundant (a pint to a quart), and on standing separates into three layers, an upper layer of foam, a middle of green or gray serum, and a lower of green or brownish granular sediment. In the sediment there are *flocculi* of lung tissue, best seen against a dark background, varying in size from small particles to pieces as large as the thumb and showing alveolar arrangement; elastic fibers are much less common than in abscess of the lung, for a trypsin-like ferment rapidly digests the fibers; *Dittrich's plugs* are found, consisting of detritus and bacteria, and emitting a most fetid odor (*v.* pages 421 and 422); *long fatty needles*, lung pigment, pus cells, red corpuscles, hematoidin crystals, bacteria, infusoria, leucin, tyrosin, phenol, indol, skatol, ammonia and sulphuretted hydrogen are also found.

2. Chest Findings.—Besides *bronchitis* caused by the offensive sputum, signs of infiltration or cavity formation are obtained if the focus is superficial and measures two and one-half inches. In the *common circumscribed form* the signs of cavity are identical with those in tuberculosis, bronchiectasis or abscess, *i. e.*, a tympanitic note, increased vocal fremitus, bronchial or amphoric breathing and metallic rales are obtained when the cavity is empty, or dulness, decreased fremitus and weak breathing when it is full. In the *rare diffuse form* there are signs of infiltration, as in pneumonia, which signs are also found in the initial stage of the circumscribed gangrene. The *x*-rays are valuable in localization of the gangrene. The patient lies so that the cavity is dependent, to avoid constant trickling of the secretion into the bronchi, *i. e.*, on his back when it is posterior or on his face when it is anterior or in an upper lobe.

3. General symptoms result from the causal disease or sepsis. Remittent fever, chills, sweats, emaciation, pallor, rapid pulse, vomiting, diarrhea, leukocytosis, albuminuria and in severe cases a profound typhoid state mark the degree of intoxication.

4. Complications may dominate the clinical picture. (*a*) Pleurisy, usually purulent or putrid, may be the chief symptom, and operation may evacuate offensive fluid with fragments of necrotic lung. (*b*) Hemop-

tysis is seldom fatal, though frequently profuse. (*c*) Pyopneumothorax, or (*d*) rupture into the esophagus, aorta, mediastinum, etc., may occur. (*e*) There may be metastatic deposits in the brain, liver, joints or muscles. (*f*) Clubbed fingers or amyloid degeneration occasionally result from septic absorption.

Course and Prognosis.—The course is usually fatal within a week in diffuse, and is more chronic in circumscribed, forms. The prognosis depends on the etiology, resistance of the patient, degree of toxemia, localization and surgical accessibility.

Diagnosis.—Exploratory puncture is dangerous, for it favors diffusion. (*a*) In *fætor ex ore* the breath is fetid but the sputum, if any, shows no findings characteristic of gangrene. (*b*) In *putrid bronchitis* the sputum shows Dittrich's plugs and fetor, but no fatty crystals or pulmonary tissue. (*c*) In *abscess* elastic fibers and pus are more abundant, the fatty crystals are oftener in sheaths than in long needles and hematoidin crystals are more common; bronchial plugs and fetor are absent. (*d*) In *bronchiectasis* elastic fibers are very rare and lung tissue has never been found. (*e*) In *tuberculosis* coincidence of the two affections and the pseudotubercle bacillus may alone cause confusion (*v.* TUBERCULOSIS).

Treatment.—1. SURGICAL.—Expectant treatment results in a mortality of 90 per cent. With operative treatment the death-rate is 45 per cent. (In 20 personal cases, 66 per cent. recovered under medical treatment.) The surgical indications are (*a*) the vital indication when acute gangrene assumes the *foudroyant* type, or (*b*) when, in chronic forms, there is no tendency toward spontaneous healing. Accurate localization by physical findings or by the *x*-rays was possible in 65 per cent. of Tuffier's series.

2. EXPECTANT TREATMENT is indicated in deep-seated gangrene, hopeless metastases or apparent demarkation. The patient is kept on his back to avoid leakage into sound bronchi. The diet should support the strength and include full alcoholic stimulation. Carbolic acid, balsam of Peru, creosote, or turpentine (when there is a complicating hemorrhage) may be given internally.

TUMORS OF THE LUNG.

I. Carcinoma.—Cancer of the lung may be primary (17 per cent.), metastatic (73 per cent.) and extension by contiguity (10 per cent.). It occurs in 0.17 per cent. of autopsies or in 1.8 per cent. of cancer autopsies (Pässler). Most primary cases develop between forty and sixty years of age. In primary forms Pässler found 73 per cent. in males; the secondary forms are more frequent in women, following cancer of the mamma or the digestive or genital organs.

Etiology and Pathology.—Cancer usually develops in the bronchi, in which previous ulceration, syphilis or adherent rupturing lymph glands are predisposing factors. It probably may develop from the bronchial mucous glands or even from the alveolar epithelium. Histologically the cylindrical type is by far the most frequent, though

medullary, scirrhous and other forms are encountered. Cancer usually begins at the hilum, and may extend toward the pleura, either by contiguity along the bronchi or by the lymph spaces. The upper lobes are more often involved than the lower and the right twice as frequently as the left. Primary disease is generally unilateral. Secondary cancer is generally bilateral; the lungs are strewn with miliary nodules resembling miliary tubercles, or present many larger nodules. Calcification and ossification may occur. Krieg collected 201 primary tracheal tumors (40 carcinomas and 21 sarcomas, most of the balance being benign).

Symptoms.—With a primary tumor in another part of the body, cyanosis, dyspnea, blood-stained sputum and signs of lung infiltration or of cavity, facilitate a diagnosis of lung metastasis. When there is a hidden primary focus or a primary lung tumor, diagnosis is for a long time difficult or impossible.

1. Early General Symptoms.—These are usually ambiguous or suggestive of tuberculosis, pleurisy or bronchitis. Thoracic oppression, coughing, stridor and *dyspnea* result from pressure on the trachea and vagus or wide substitution of tumor for lung tissue. Cyanosis, dyspnea, rapid breathing, emaciation and sweats may suggest miliary tuberculosis; this resemblance may also be noted at autopsy in miliary carcinosis; fever develops in over half the cases. Rokitansky's contention that pulmonary cancer and tuberculosis do not coexist is disproved by Wolff's 31 cases of cancer of the lung of which 23 had tuberculosis.

2. Sputum.—The sputum in over half the cases is rather characteristic. Altered blood gives it a reddish-brown color or it resembles raspberry jelly in hue and in consistency; it is sometimes grass-green or prune-juice in color. Occasionally macro- and microscopic bits of cancer tissue in characteristic nest-like arrangement are seen.

3. Physical Signs.—(*a*) *Inspection:* The patient usually lies on the affected side to give freer play to the sound lung. Marked *ectasia* is common; sometimes the diseased side is shrunken from lung cirrhosis or bronchial stenosis. (*b*) *Palpation* elicits *increased fremitus* if the tumor partially occludes the bronchi. (*c*) *Percussion* gives *increased resistance* and *irregular dulness,* increasing to absolute flatness, most often over an upper lobe or substernally. Flatness persisting after tapping, and blood-stained sputum without tubercle bacilli in a patient over fifty years, at once suggest pulmonary neoplasm. (*d*) *Auscultation* varies with the patency of the bronchus, giving *bronchial breathing* when it is open and weak or *absent breathing* when occluded.

4. Compression Symptoms.—Besides the early dyspnea and cough, (*a*) the heart is often luxated or its yielding auricles are crowded. (*b*) The venous trunks suffer compression, with cyanosis and edema of the face, neck and arms and with collateral dilatation of other veins of the thorax. (*c*) Other symptoms of compression are rapid or slow heart (vagus compression), recurrent laryngeal compression with phonic changes, dysphagia, intercostal neuralgia, brachial neuralgia or paralysis, and bronchial stenosis.

5. Metastasis.—This occurs in 85 per cent. of cases; there may also be extension, by contiguity, to the axillary or cervical glands.

6. Complications include hemoptysis from necrosis and cavity formation; gangrene and bronchiectasis; and pleurisy, which is usually serous (hemorrhagic in 12 per cent., even like venous blood, or rarely adipose or chyliform effusions); hydropic, fatty and granular cells or flocculi of tumor tissue, may be obtained by puncture. Carcinomatous nodes may develop at the site of puncture. The course is indicated by Marfan's types; the acute or galloping pleuropulmonary cancer, and the chronic type (the typical, bronchopulmonary, the mediastinal, and the pleuritic with or without effusion).

II. Sarcoma and Lymphosarcoma.—Sarcoma and lymphosarcoma are more frequent than cancer. They probably begin chiefly in the lymph nodes of the bronchi, and oftener on the left than on the right side. Extension in all directions occurs along the peribronchial tissues from the hilum. A primary, very malignant lymphosarcoma develops in cobalt miners of Saxony; 150 cases among less than 700 miners were described by Anke in 1884. Ulceration is less common than in cancer, but the symptoms are, in the main, identical. Sarcomatous tissue is occasionally voided in the sputum or aspirated by the exploring needle. The differentiation of malignant lung tumors must be made chiefly from aneurysm (*q. v.*) and pleurisy (*q. v.*). The *x*-rays may be of value. There are three main clinical groups, (*a*) in which a neoplasm, perhaps its exact type, can be diagnosticated; (*b*) in which some growth is present but differentiation from aneurysm is uncertain; and (*c*) in which the disease escapes detection or is only suspected.

Prognosis.—Death generally occurs in less than a year with cachexia or suffocation, but sometimes within a few weeks.

III. Other Tumors of the Lung.—Fibroma, lipoma, osteoma, hypernephroma and enchondroma are of solely anatomical interest.

Treatment.—It is only palliative—morphine for extreme dyspnea and cough. The *x*-rays and arsenic offer little encouragement.

CIRCULATORY AFFECTIONS OF THE LUNG.

I. Active Congestion.—As a secondary lesion acute hyperemia occurs commonly with tuberculosis, pneumonia, influenza, pleurisy or cardiac strain. French writers describe an *acute primary congestion* (*Maladie de Woillez*); some cases are apparently abortive pneumonia; perhaps it is due to attenuated pneumococci, which Carriére has demonstrated by puncture of the lung It develops like pneumonia, with chill, fever of 101° to 103°, pain in the side, dyspnea, cough and an abundant, viscid but not blood-stained sputum. Physical examination is said to elicit somewhat impaired resonance and weak or occasionally bronchial breathing.

II. Passive Congestion; Hypostasis.—In broken compensation, particularly in mitral stenosis, the lungs are distended, heavy, indurated, and pigmented a brownish-red; this is brown induration (*induratio pulmonum fusca*). The capillaries are turgid, the connective tissue is increased, the lungs are tinged with blood pigment and the sputum contains the so-called heart disease cells (see page 368).

Hypostasis is a passive congestion from weak heart, in typhoid and other fevers, old age, cachexiæ, in opium and other narcoses and in apoplectic or other comas; it *rarely occurs if the patient's position is changed every few hours.* The lung is dark red, engorged with blood and serum, and may also show aspiration pneumonia and atelectasis; the latter conditions are incorrectly called hypostatic pneumonia. There is moderate dulness over the lower lobes posteriorly, in apoplexy perhaps only on the paralyzed side; the breathing is more often weak than bronchial, and moist or crepitant rales are heard. Fever is no part of hypostasis.

Treatment.—Treatment of the fundamental lesion, phlebotomy, stimulation and frequent change of posture are indicated.

III. Infarct; Embolism.—Etiology.—Embolism, first observed by Laennec, is most frequently caused by (*a*) fibrinous coagula from the right heart, the frequency and mechanism of which have been discussed under Acute and Chronic Endocarditis and Septic Infections. Clots may also reach the lung from peripheral thromboses or rarer causes are (*b*) tumor or hepatic tissue, fat embolism after fractures and liver injury, air embolism or echinococcus cysts.

Pathology.—The embolus may obstruct a main branch of the pulmonary artery, straddle one of its bifurcations or lodge in a smaller vessel. Not every embolism produces an infarct, for the occlusion may be incomplete or collateral circulation possible. Occlusion of an end-vessel results in the hemorrhagic infarct, which is often wedge-shaped with its base toward the pleura, is airless, on section projects above the cut surface, and is attended by extravasation of blood into the lung, though the lung is not torn. This is known as a fibrinous or *simple infarct.* If the embolus contains pus or putrefactive microörganisms, it initiates *abscess* or *gangrene.* Infarcts occur in the right lower lobe in 46 per cent., right upper in 21, left lower in 15, left upper in 12 and right middle lobe in 6 per cent. They are often multiple. Small infarcts may be absorbed, but larger ones leave pigmented cicatrices.

Symptoms.—(*a*) Infarction of the main pulmonary trunk may result in immediate death, as from sitting up after childbirth (two-thirds of these cases die). In other cases death occurs in a short time from cerebral anemia and cardiac failure. The infarct may cover the greater part of the lobe involved, and a systolic murmur may develop over the pulmonary artery. (*b*) Infarction of a smaller vessel is often ushered in with chill, vomiting, dyspnea, cyanosis and fever, even with bland embolism. The sputum contains dark blood and a "stitch in the side" denotes pleuritic participation. Physical examination frequently elicits a pleural rub and later, in some cases, slight dulness, especially behind, between the spine and scapular line; crepitant rales and bronchial breathing are frequently present. Pyogenic or putrid clots cause abscess, gangrene, empyema or pneumothorax.

Diagnosis.—Sudden cardiac failure is very difficult to differentiate from embolic occlusion of the pulmonary trunk, especially when death occurs without hemoptysis. The etiology, sudden onset and circumscribed chest signs are of diagnostic importance. Treatment lies in

cardiac stimulation and prophylaxis—rest during endocarditis, cardiac insufficiency or phlebitis.

IV. Edema of the Lungs.—Etiology.—Pulmonary edema was described by Laennec (1819). In the etiology of this secondary, common, frequently terminal or agonal affection, *three important causes* stand in the foreground, viz., *heart, kidney* and *lung disease.* Edema is a serous transudation into the air cells and interstitial tissue. Classification of its mechanism is difficult: (*a*) *Stasis* due to heart weakness is its most frequent mechanism. Cohnheim and Welch demonstrated experimentally that it may result from failure of the left ventricle, with continued activity of the right ventricle (this explanation is not entirely satisfactory, for cases in which the pulse is strong; however, general cardiac anasarca may occur when the radial pulse is strong, though it is known that the heart must be weak). Weak heart may result from any disease of the endo-, myo- or pericardium. Sahli explains certain cases by (*b*) *paralysis of the pulmonary vessels.* Under (*a*) or (*b*) nephritic or toxic edema, edema from pleural paracentesis, the iodides, ether, adrenalin or pilocarpine may be included. (*c*) *Inflammatory edema, e. g.,* around fibrinous pneumonia and other pulmonary disease, is an exudation, not a transudation; the pulse is usually strong and there is often fever. It is possible that (*d*) *angioneurotic* edema or (*e*) *occult changes in the blood and bloodvessels* are causal factors. These varied mechanisms, most common in cardiac, renal and pulmonary lesions, may also operate in cachexia, anemia, cerebral disease, old age or innumerable other conditions.

Pathology.—Edema may be circumscribed or oftener diffuse. The lungs are large, heavy and spongy; they pit and on section, especially of the bases, a frothy serosanguineous fluid exudes.

Symptoms.—Symptoms usually come on *precipitately,* with dyspnea and cyanosis. The *sputum* is copious, foamy, somewhat resembling soapsuds or at times tinged with blood; it is rich in albumin and poor in mucus and cells. The *bubbling breathing* indicates serum in the alveoli. The second pulmonic sound is accentuated, the pulse is weak, the lung note is slightly tympanitic from relaxation and coarse and fine rales are heard everywhere. If the lower lobes are filled, the physical signs may closely resemble those of consolidation. Recurrent attacks are described. The *diagnosis* is determined from the sputum and lung findings.

Treatment.—Too often edema is the death agony itself. (*a*) *Prophylaxis* concerns, *e. g.,* the causal, cardiac weakness or anasarca in nephritis. (*b*) *Diffusive stimulants* are indicated hypodermically, as camphor, strophanthus and strychnine, and by mouth, coffee or champagne. (*c*) *Morphine* is almost specific. (*d*) *Venesection* relieves the right heart and is particularly indicated in hypertension. (*e*) *Stimulating expectorants* and *rapid purgation* by croton oil, ℿiij, on the tongue and artificial respiration are rarely of value. The author has obtained no results from the acetate of lead. Atropine and adrenalin are dangerous, adrenalin not infrequently inducing edema.

V. Hemoptysis.—Hemoptysis or hemoptoë refers to "spitting of blood," while profuse hemorrhage is known as pneumorrhagia.

Etiology.—(*a*) Laryngeal ulceration or inflammation; (*b*) tracheal ulceration or aneurysm bursting into the trachea; (*c*) severe inflammation or ulceration of the bronchi, cardiac stasis, bronchiectasis, putrid and fibrinous bronchitis, rupture into the air passages of aneurysm or anthracotic lymph glands; hemoptysis may also occur in the acute exanthemata, in blood affections as hemophilia, scurvy, purpura and leukemia, and in renal disease and cholemia; hemoptysis occurring as vicarious menstruation, mentioned by Hippocrates, is an established, though rare, variety; hemoptysis vicarious to suppression of hemorrhoidal bleeding must be regarded with scepticism. (*d*) *Lung disease is its most important* etiology, notably (i) *tuberculosis* (*q. v.*), which explains 80 to 90 per cent. of blood-spitting. It very rarely occurs under six years of age, as children seldom develop cavities. It occurs as capillary or profuse hemorrhage. Hemoptysis sometimes occurs epidemically among tuberculous subjects, referable to the pneumococcus. (ii) Infarcts are characterized by hemoptysis. (iii) Pneumonia, abscess and gangrene; (iv) tumors; (v) parasites of the lung, trauma; and (vi) syphilis and leprosy are more uncommon causes. (*e*) Hemoptysis nervosa is most rare, though authentic cases have occurred in cerebral traumatism, hysteria (where it has caused death) and epilepsy. (*f*) Toxic hemoptoë from mercury or iodides is very rare.

Symptoms.—Symptoms follow those of the causal tuberculosis, mitral stenosis or aneurysm, or develop in apparent health. Blood poured out into a cavity or into the lung substance may not be voided, in weak or moribund persons. Prodromes are unusual; *without warning*, a salty taste in the mouth, substernal warmth, a sense of something rising from the chest and coughing are followed at once by a few spoonfuls or more of blood, which also may be accidentally noted in a previously mucous sputum. The blood varies from the merest stain to 5 quarts; it may appear but once or repeatedly. It is generally bright red and well mixed with air. If the blood has clotted, the clot is mixed with air and perhaps with mucus. Casts of the bronchioles may be voided. In gangrene or abscess, the blood may be brown or buff-colored and the red cells disintegrated. Brown hemosiderin granules or, if the blood has remained as a clot two weeks, hematin crystals may occur.

Physical findings, other than those of the causal condition, are frequently lacking; if blood stagnates in a large bronchus, there may be bronchial breathing, bronchophony or dulness if the affected area measure 2 by 5 cm. In the bronchioles, blood gives rise to small rales, in the bronchi to coarser rales.

Complications.—Fever, aside from the causal disease, may result from resorption (ferment fever). Urobilin icterus, functional heart murmur, relaxation of the right heart, albuminuria and puffiness of the ankles are possible complications.

Diagnosis.—Examination of the nose, gums, throat and larynx excludes hemorrhage simulating hemoptoë. Differentiation between bronchial and pulmonary bleeding is decided by the history, later physical examination and clinical evolution.

Hematemesis	*vs.*	Hemoptysis.
(*a*) The blood is acid (unless a very large amount is voided).		Is alkaline.
(*b*) The patient always recognizes whether it is vomited or coughed up. (Blood from the lung is very rarely swallowed and then vomited.)		(Hemoptysis, very rarely, causes consensual vomiting.)
(*c*) Is often dark (though large amounts of bright red blood may be vomited, as in ulcer).		Nearly always bright (though when stagnating in a cavity it may become dark or buff).
(*d*) Is mixed with food, HCl, ferments, etc.		Mixed with air, which is also found in the clots. (Not infrequently tubercle bacilli or heart disease cells are found.)
(*e*) In 16 per cent. of cases blood is in the stools.		Is very rare in stools.

Prognosis and Course.—Spontaneous cessation is the rule. The immediate prognosis is good, as early death occurrs in 3 per cent. only, except in profuse pneumorrhagia, resulting from aneurysmal rupture (even then the patient may live years). Drowning of the patient in his own blood is rare. The final outcome depends on the etiology. In Ware's 386 cases 62 per cent. recovered.

Treatment.—Prevention.—In tuberculosis (*q. v.*) the patient should be kept quiet, the cough suppressed and exertion and vocal efforts avoided; in heart disease and marantic thrombosis, rest is indicated to obviate detachment of the thrombi.

Therapy.—*Absolute physical and mental quiet* should be enforced. Movement and talking are prohibited; coughing is suppressed by hypodermics of morphine, which allay the mental excitement (opiates are employed guardedly in very large hemorrhages, for bronchial occlusion may result). The patient is assured of a favorable outcome; no physical examination should be made except auscultation of the chest in front, for treatment takes precedence of diagnosis; if rales indicate the side of the hemorrhage, the patient should lie on that side to prevent aspiration into the contralateral bronchus.

Arterial pressure should be reduced by inhalation of 10 to 15 drops of *amyl nitrite,* which lessens tension in the pulmonary and arterial circulation. A liquid diet is ordered. Alcohol, coffee and tea should be interdicted. Even if the patient collapses cardiants should be avoided, for syncope is nature's hemostatic; the old practice of venesection in this way promoted thrombosis. Cardiac excitement is lessened by placing a large ice-bag over the precordium. Active purgation by croton oil is very beneficial.

Hemostatics do not control the lesser circuit, which is independent of drugs which operate on the arterial circuit. Ergot raises the blood-pressure in the lungs. Calcium lactate, gr. xxx, and common salt on the tongue are of some value (salt acts reflexly, by way of the vagus, on the pulmonary vessels). Horse serum may be used in urgent cases and the beneficial effects of turpentine have long been known. Nature, as a rule, effects the cure. Hippocrates employed ligature of the extremities, *i. e.*, binding tightly for a few minutes the vessels of the groin and axilla, to lessen the return flow of blood. Artificial pneumothorax, to compress the bleeding lung, is advocated in desperate and persistent hemoptysis.

DISEASES OF THE PLEURA.

PLEURISY.

Definition.—An inflammation of the pleura, secondary to tuberculosis or other lung disease, general and septicemic infections and disease in contiguous tissues. Hippocrates recognized the dry and humid types and tapped for empyema and pneumothorax. The first full description dates from Laennec.

Pleurisy constitutes 3 per cent. of internal maladies and is the most common of all postmortem findings (in over 50 per cent.). It is susceptible of division into infinite varieties, acute, subacute or chronic according to its course, dry or effusive according to its consistence, primary or secondary as to its cause, diffuse or circumscribed as to its distribution, and serofibrinous, hemorrhagic or suppurative as to its nature. It seems preferable to describe all its symptoms first and group special types later.

Etiology. — 1. Tuberculosis. — So-called primary, spontaneous or cryptogenetic pleurisy is usually tuberculous. Landouzy considers that 98 per cent. of all pleurisies are tuberculous and Le Damany proved by inoculation that 92 per cent. of "primary" pleurisies were tuberculous. Lord states that two-thirds of pleuritics have no obvious disease in other organs and three-quarters respond to tuberculin. The causal lesion may be recognized at autopsy in calcified bronchial glands or as small lung foci (*v.* page 459).

2. Diseases of the Lung Substance.—Pneumonia, abscess, gangrene, infarcts, tuberculosis, tumors, pneumothorax, etc., are etiological factors; according to Koplik, 95 per cent. of pleurisies in children follow pneumonia. It is less frequent in diseases involving the bronchi, as in grippe or measles.

3. Acute Infections.—Rheumatism, sepsis, endo- and pericarditis, scarlatina, and other acute infections may result in pleurisy. It develops in about 2 per cent. of typhoid cases.

4. Diseases of Adjacent Organs.—(*a*) *Intrathoracic* affections, involving the ribs, spine, esophagus, mediastinum, lymph glands, diaphragm and pericardium; and (*b*) *abdominal* affections, as of the stomach, spleen or liver (abscess, tumor or cirrhosis), subphrenic abscess, suppurative, carcinomatous or tuberculous peritonitis (in 25 per cent.), perinephritic abscess, etc., may excite pleural infection by direct local invasion or by the lymph and blood streams; in neglected appendicitis, hepatic and diaphragmatic invasion are the steps to pleural infection.

5. Tumors.—Echinococcus and endothelioma are the most important types; carcinoma, sarcoma, ecchondroma and rarely lipoma are also factors; pleural neoplasms are far more frequently secondary than primary.

6. Altered Conditions of the Blood.—Gout, nephritis and scurvy are often causative of pleurisy, as reduced physiological resistance predisposes to infection.

7. Terminal Pleurisy.—Various forms of sepsis are often fatal to subjects of chronic disease of the heart, liver and kidneys.

8. Age, Sex, Localization.—*Age:* most cases occur between the twentieth and fiftieth years, but no age is exempt. *Sex:* Eichhorst observed pleurisy in 3 per cent. of all his male and 2 per cent. of all his female cases, and found the following *localizations:* right-sided in 54 per cent., left-sided in 42 per cent. and bilateral in 4 per cent. Cases following nervous disease, cold or exposure, venous thrombosis or trauma can usually be brought within the above given classification.

Bacteriology.—The above etiological classification does not correspond to the bacteriological division. Suppurative pleurisy may result from the streptococcus, pneumococcus, tubercle bacillus or less often from the staphylococcus or typhoid or colon bacillus; serous pleurisy may be due to the tubercle or pyogenic organisms. The frequently negative cultural results favor the hypotheses that chemical products may induce pleurisy or that bacteria produce pleurisy and then die. It must be remembered that cultures are made, not from the pleura directly, but from the fluid effused from it. The organisms found are most frequently the pneumococcus, tubercle bacillus and streptococcus; less often the staphylococcus, pneumobacillus of Friedländer, typhoid, paratyphoid, colon, influenza and diphtheria bacilli, gonococcus, meningococcus, ray fungus, etc. (*a*) *Purulent pleurisy* (empyema) is due most often to the streptococcus in adults and the pneumococcus in children. About 10 per cent. of empyema is tuberculous; empyema giving no results culturally or by inoculation is usually tuberculous—a cold abscess of the pleura. Other bacteria (*v. s.*) are infrequent factors. (*b*) *Serous pleurisy* is usually tuberculous, as shown by the increasing percentage of successful inoculations. Tubercle bacilli when found, suggest a direct communication with a tuberculous focus in the lung or lymph glands; Eichhorst found them by direct examination in 15 per cent. of serous pleurisies, though inoculations were positive in 62 per cent. (*c*) So-called primary pleurisy which is non-tuberculous is usually pneumococcic. (*d*) Meta- and parapneumonic pleurisy is most frequently pneumococcic, but sometimes streptococcic and staphylococcic.

Symptoms.—Physical findings are absolutely essential for diagnosis and all subjective symptoms are ambiguous or may be wholly absent. In the main, all types have much the same signs which differ more quantitatively than qualitatively; serous pleurisy nearly always shows friction, and fibrinous pleurisy is always accompanied by some exudation.

Inspection.—(*a*) Inspiratory retraction of the intercostal spaces may result from imperfect ingress of air, pain or adhesions. Epigastric retraction during inspiration is due to contraction of the diaphragm. (*b*) The patient often lies during the dry stage on the sound side to avoid pain from pressure on the inflamed surfaces; later, he lies on the diseased side to allow freer expansion of the sound lung, especially when the exudate is large; the patient may sit up for breath, or assume the diagonal decubitus. (*c*) *Ectasia* (distention) of the affected side occurs especially in children, and is determined by mensuration and inspection. The interspaces are obliterated, the costal arch is immobile during respiration, the skin glistening, the spine curved toward the diseased side, the shoulder higher and the nipple and scapula are farther from the median

line. (The right chest is normally one or two centimeters larger.) The sound side exceeds, by three centimeters, its normal dimensions on account of compensatory emphysema. In some very large effusions the writer has observed an apparent retraction of the diseased side. (*d*) *Respiratory movement* of the affected side *is decreased* or absent both in dry and exudative pleurisy. Pain alone may reflexly decrease the respiratory expansion and variation on the same side may occur, there being more movement above and less below. The frequency of respiration is increased from lung compression, heart luxation, the low diaphragm, fever, pain or paresis of the intercostal muscles. (*e*) The *x*-rays show a dark shadow. (*f*) Inspection reveals cyanosis, dislocation of the apex and occasionally edema (*v. i.*). (*g*) *Absence of Litten's phenomenon* and (*h*) rarely pulsation of the effusion. (*i*) C. L. Greene describes a rhythmic lateral displacement of the heart most marked in medium-sized effusions. The heart approaches the affected side in inspiration and moves outward in expiration, movement often amounting to two inches, measured by fluoroscopic examination, percussion of the free cardiac border, or by inspection of the apex.

Palpation confirms inspection. (*a*) An up-and-down *friction-rub is felt;* it increases on pressure, which also increases the pain and tenderness, and it is the more readily distinguished the coarser the rub. The friction develops before the effusion, is often present with it and after it is absorbed. The rub is due to roughening of the pleura, which loses its glistening appearance and looks like glass upon which one has breathed; the two raw pleural surfaces scrape on each other. It may last a few hours or days, even disappearing when the patient takes a deep breath; or it may last for months or even years. (*b*) Pressure on a tender interspace may cause a sudden contraction of the rectus muscle on the side of lesion. (*c*) *Vocal fremitus,* normally greater on the right side, varies according to the level at which it is tested. (i) Above the effusion it is normal. (ii) Near the spine over the compressed, relaxed lung it is increased. (iii) *Below it is weak or absent,* from exudation of thick fibrin or fluid, though less change is observed in dry than effusive pleurisy. The condition of the lung and the bronchial secretion are important, whence before testing, the patient should cough and take a few deep breaths. The results are irregular in cases of loculated effusion and of adhesions bringing the lung closer to the chest wall in given places. (*d*) *By tapping with the finger tips the increased resistance* is clearly palpated. (*e*) Edema of the chest wall on the diseased side may be inflammatory in serous and purulent pleurisy, or mechanical (and more intense) from pressure on the azygos or hemiazygos veins. (*f*) Fluctuation is extremely rare. (*g*) Palpation determines the *dislocated apex beat,* the *dislocated liver or spleen,* the bulging of the diaphragm and sometimes a furrow between the diaphragm and liver (Stokes).

Percussion is negative in dry pleurisy, except when a very thick fibrin causes some dulness and weak breathing; after an effusion has resorbed, the residual plastic lymph maintains the dulness. In effusion (*a*) *dulness* is found typically between the fifth and eighth ribs in the left axilla or over the liver and posteriorly on the right side. Dulness

also results from old adhesions, or atelectasis of the lung. As in pneumothorax, emphysema of the lungs and calcification of the ribs may somewhat obscure the dulness of an effusion. In doubtful cases the apparently sound side should be percussed first. Thirteen ounces of fluid are necessary in the adult for recognition and four ounces in children, and sometimes six ounces, which produce a dulness two fingers in height, may be recognized, especially in children and women having thin thoracic walls. The effusion must be one inch thick to produce dulness. Before percussion the patient should breathe deeply several times to eliminate partial collapse of the lung and accumulated bronchial secretion. *Percussion is absolutely essential to diagnosis.* Not merely the audible dulness should be regarded, but also the *palpable sense of resistance,* on light percussion. In larger effusions, the *distinct flatness,* at first located posteriorly, increases along the spine and laterally in the axilla, and in massive exudates, may reach above the clavicles.

The dulness is highest posteriorly as the patient lies on the back. When he sits the line of dulness describes a parabolic curve (courbe de Damoiseau or the Garland-Ellis line) which is lowest behind and due to the uneven thickness of the chest wall or to adhesions. If the fluid should effuse with the patient out of bed, the upper level would be horizontal. Dulness is found one or two centimeters higher than the fluid, due to plastic exudate. (*b*) Right-sided effusions merge with the liver dulness, but the lower limit of left-sided effusions can usually be determined because they *invade Traube's semilunar space,* which lies between the heart's apex above and tenth rib below, representing the supplementary pleural space and is designated as the "half-moon" space because of the curve at its upper portion, produced by the apex; its tympanitic note is decreased or replaced by flatness in left-sided effusions (without adhesions). Dulness here may not be confused with the spleen, for there is no respiratory excursion in pleurisy, and the dulness lies more toward the heart than toward the spleen. (*c*) *Respiratory excursion and Litten's sign are absent.* (*d*) Change of the upper line of dulness with change of position, emphasized by most writers, cannot be established, because of adhesions at the upper level; the patient must lie hours in another posture before any alteration occurs.

The following findings are less important: (*e*) *A vertical strip of tympany* along the spine, often measuring 3 cm. wide, may be detected in a large exudate, due to compression of the lung. Tympany in the lower left chest results from the gaseous stomach and intestines. (*f*) Tympany over an upper lobe from compression or relaxation of the lung, was observed by Avenbrügger and Skoda—the *Skodaic note.* (*g*) *"Cracked-pot" resonance* occasionally appears on short, forcible percussion over the upper lobe, and results from sudden expulsion of air through the rima glottidis. (*h*) *William's tracheal note* is observed on the left more than on the right side and most clearly in the first and second interspaces. It is obtained chiefly when the entire side is dull and is due to transmission of percussion vibration from the fluid to the trachea, giving a tympanitic note. (*i*) *Grocco's sign is a small triangular dulness on the sound side*

along the vertebræ, due to displacement of the mediastinum and, with it, the sound lung, which is pushed away from the vertebral column by fluid in the lower chest; it also occurs in subphrenic abscess, cysts or ascites; Korányi first described this sign in 1897 and Grocco in 1902. (*j*) *Dislocation of other organs.* There is no parallelism between the amount of exudate and the amount of luxation, which, of course, is absent if the organs are adherent. The average increase of intrathoracic pressure is 10 mm. mercury (0 to 26). The *heart is usually dislocated in toto* and seldom suffers change of the direction of its axis. The right ventricle pulsates to the right of the sternum, even at the right nipple line, in left-sided effusion. In right-sided effusions the apex is pushed to the left and downward, for the diaphragm is lower. The *liver* is *less easily displaced* than the heart, but in right-sided effusions is pushed down (especially in empyema) and the left lobe tipped somewhat upward; both lobes are dislocated downward in massive pleurisies. In left-sided effusions the *spleen is dislocated downward* and forward, undergoes some torsion and may stand perpendicular to the costal arch.

Auscultation.—This is most important in dry and effusive pleurisy. (*a*) The *friction-rub* may be hard or soft, is superficial, *i. e.,* close to the ear and resembles the sound made by scratching the finger upon the ear or the crunching of snow under the shoe; it often develops in the first twelve hours. It is most frequent during inspiration, but is very often detected in expiration, but seldom during expiration alone. It may disappear after several long breaths which smooth the pleural roughening. It may be increased by pressure and its duration is subject to no law; Gerhardt heard a pleural rub which lasted six years. It is heard most frequently over the anterolateral portion of the chest, and is detected more clearly with the stethoscope than with the naked ear, but may be heard at a distance of several feet, as in a case seen with Dr. H. M. Richter. It may rarely be present in miliary tuberculosis and in tumors of the lung or pleura without actual pleurisy. It usually becomes less clear when exudation develops, and its reappearance indicates absorption or extension. (*b*) The *breath sounds* are modified not only by the effusion, but also by the condition of the lung, and the conditions of conduction in the pleura and thoracic walls. In general, and especially in men and children, the *respiratory sounds are weakened* even in fibrinous pleurisy, from repressed breathing and perhaps from pain alone; cog-wheel breathing is common. In moderate effusions three zones are observed: (i) Absence of breathing in the lower parts from compression of the lung by the exudate, which lessens the conduction of the vesicular sounds; (ii) bronchial breathing in the middle zone from moderate collapse of the alveoli of the lung, with fair conduction through the bronchi; (iii) an upper zone of sharp vesicular breathing. In larger effusions the lower zone extends higher and bronchial breathing may be heard near the clavicle or between the scapula and spine. In the largest effusions the breathing is indistinct everywhere (with metallic bronchial breathing when the lung is wholly collapsed). (*c*) The *voice sounds are not heard* over the fluid or over very thick, plastic exudate. They may persist where islets of lung adhere to

the chest wall. (*d*) *Bronchial breathing* and increased fremitus may be present, especially in children with empyema, under the following conditions: if the bronchi are patent, if there is no great fibrin deposit on the pleura and if the lungs are retracted but not absolutely compressed. The bronchial breathing may sometimes be as intense as in pneumonia. In adults increased fremitus may be observed between the spine and scapula where the lung is compressed. (*e*) *Bronchophony* depends upon the same conditions as bronchial breathing. (*f*) *Egophony*, which is an exaggerated bronchophony, was described by Laennec; it is found chiefly in middle-sized effusions, usually at the level of the fluid; in large effusions it is rare, the compression being too great. It is caused by the voice sounds passing through the compressed smaller bronchi to the chest wall; it may be present when the exudate is increasing or decreasing at a given point. (*g*) *Baccelli's symptom* of the "whispering voice," which he thought was more frequent in serous effusions, also occurs in other forms of pleurisy, in induration of the lungs, pneumonia, hydrothorax and cavities. (*h*) A *wide propagation of the heart tones*—if consolidation is excluded—suggests dislocation of the heart by an effusion. Murmurs may result from bending of the large vessels by dislocation of the heart, as well as accidental murmurs resulting from a heart weakened by toxemia.

Diagnostic puncture determines: (*a*) the existence of pleurisy, excluding pneumonia, hypostasis, tumor, etc.; (*b*) the character of the fluid; and (*c*) the prognosis. It is indicated in all doubtful cases. Puncture with aspiration is also an important therapeutic measure. *Negative results* may result from a thick, fibrinous exudate, from failure to reach an encapsulated effusion, as in interlobar pleurisy, and from the membrane being pushed in before the needle. In dry taps, after withdrawal of the needle, examination should be made to see that no fibrin or cells plug the needle. If a plug is obtained it should be examined under the microscope. No carbolic acid or alcohol should remain in the syringe or needle, which, by coagulating the serum, may explain some dry punctures. The puncture should be made with a large needle. *Positive results:* when the needle is introduced, resistance due to a callous pleurisy or indurated lung may be felt. After introducing the needle, the point may be freely movable, as in a cavity. In rare cases serum may be obtained from a high puncture and from a lower one a cellular deposit, the cells settling to the bottom; oftener puncture in different localities shows serum in one and pus in another sac—Galliard's polymorphous pleurisy. A pocket of sterile effusion often indicates a contiguous encapsulated empyema or a lung abscess. Bacteriological examinations made with 20 to 30 c.c. of the serum give frequent positive results. The special chemical, bacteriological, cellular and other characteristics will be discussed under individual forms.

X-ray Findings.—It is usually stated that fluid first accumulates under the lung, between it and the diaphragm. Englebach and Carmen found this was the case in but 25 per cent. of cases; in another 25 per cent. the fluid stood like a vertical column and in 34 per cent. laterally —nearer the chest wall than the mediastinum; the column was oblique

in 16 per cent. The *x*-rays confirm the lack of mobility on change of position and show how adhesions may prevent luxation of organs, invasion of Traube's space, respiratory excursion, etc. Small or deep pockets of fluid near the diaphragm or in the interlobar fissures are thus detected which may escape physical examination.

Symptoms.—Symptoms may be absent (latent pleurisy); if present they are not distinctive and there is no relation between them and the physical findings. (*a*) The *temperature* rise is usually gradual and without a rigor. Temperature is no positive index as to the character of the fluid. For weeks there may be no temperature, even in purulent exudates, especially in marantic and diabetic subjects and in terminal infections. When present it is more frequently subcontinuous or remittent than continuous. The average elevation is 101° to 102°. Exacerbations of fever are ominous, expecially when no change occurs in the physical findings. Recovery is not expected until the temperature has been normal for some time. (*b*) The *pulse* is increased; it is sometimes of the paradoxical type or unequal in the two radials. (*c*) *Dyspnea* seldom attends pleurisy, for effusions may attain enormous proportions without respiratory difficulty, particularly when they are gradual in development. Urgent dyspnea is usually referable to coincident pneumonia, pericarditis, arteriosclerosis, nephritis or bilateral pleuritis. (*d*) *Pain* is usually an initial symptom. It is in no way proportional to the physical findings; it may be absent when auscultation shows a marked rub. Its character is usually dull, tearing, stabbing and sometimes very severe, crampy or colicky. The pain is pleural (Cruveilhier) and is present in the diseased side, well down over the ribs, liver, epigastrium or low in the back, and sometimes may radiate to the sound side, into the arm, spine or abdomen, especially in children, in which case it may simulate appendicitis. (*e*) *Coughing*, an early and frequent symptom, results most often from pleural irritation. It may occur after tapping or change of posture. It is usually unproductive. (*f*) Other symptoms are anorexia, vomiting, headache, and difficult swallowing due to involvement of the foramen esophageum; the sweats of pleurisy rank third in importance after those of sepsis and rheumatism. The urine is scanty but increases during resorption, when peptonuria and transient albuminuria are often found. The *skin* may be pale and cachectic from purulent accumulations, red from high temperature, cyanotic from poor oxygenation or suddenly pallid from hemorrhage into the pleura, when also there is a sudden increase of fluid. *Emaciation* is common in chronic tuberculous or purulent pleurisy.

Special Forms.—1. Character of the Inflammation.—In 790 cases Eichhorst found serous pleurisy in 81, fibrinous in 12, purulent in 5, ichorous and hemorrhagic each in 1 per cent.

(*a*) *Serous pleurisy* is the prototype of pleuritis. The fluid is lemon-colored. Its specific gravity ranges between 1.015 and 1.023, which characterizes inflammation. Albumin amounts to 3 to 7 per cent.; sugar is frequently present. The fluid clots after puncture. All pleuritic exudates, serous and otherwise, contain white and red blood cells and endothelial cells, which may show fatty degeneration.

Cytodiagnosis.—Widal, Sicard and Revault (1900) insisted that *lymphocytes* are characteristic of primary pleural tuberculosis and afford diagnostic data long before the results of inoculation are available; the fluid must be examined before it coagulates; in other pleurisies the polymorphonuclears prevail and that over 50 per cent. lymphocytes indicates tuberculosis; the "lymphocytic formula" proves tuberculosis, the "polymorphonuclear formula" indicates pyogenic infection or pleurisy secondary to well-developed phthisis and the "endothelial formula" denotes irritation, transudation or malignancy. The tuberculous nature of pleurisy is established by inoculation tests (positive in 66 to 94 per cent.), by apical or other involvement, by the finding of tuberculosis in cases of sudden death in the course of pleurisy, by the usually intensely positive ophthalmoreaction (see page 164) and by the fact that 25 to 33 per cent. of cases become obviously tuberculous. It is well to suspect the tuberculous nature of all so-called rheumatic pleurisies and carefully watch the sputum and lungs. The leukocytes are not much increased in the blood.

(*b*) *Fibrinous pleurisy* usually shows some fluid, serous or purulent, if systematic punctures are made.

(*c*) *Purulent pleurisy* (empyema) usually begins as a serous exudate which soon becomes purulent. In contrast to serous pleuritis two-thirds of this group are secondary: (i) the streptococcic form, which is observed chiefly in adults, in which the thin fluid separates on standing into an upper serous and a lower denser layer, with slight fibrin formation; and (ii) the pneumococcic form, which is more common in children; the fluid is more homogeneous and viscid, like "laudable" pus, fibrin formation is abundant and the odor peculiar; it is more often accompanied by endocarditis, pericarditis and meningitis, and is more often febrile and more susceptible of spontaneous recovery. In Netter's series of empyema the streptococcus was causal in 47 per cent., the pneumococcus in 39, the tubercle bacillus in 11 and the staphylococcus in 3 per cent. Over 80 per cent. of juvenile empyemas are pneumococcic. It is remarkable that the pneumococcus has far less pyogenic propensities in the lung than in the pleura. Microscopically, various bacteria are present, polymorphonuclear and often fatty white cells, and sometimes cholesterin and Charcot-Leyden crystals. Greater visceral luxation and distention of the chest than in any other type may occur; Krause reported 22 pounds of pus. Leukocytosis and hectic fever are mentioned under symptoms.

Empyema pulsans (Walshe); Sailer collated 95 cases in 1904; 95 per cent. of all pulsating effusions are purulent; 93 per cent. are left-sided. A pulsating tumor as large as the fist may be noticed, which usually decreases during inspiration, or the effusion may simply pulsate behind the ribs; in either case the pulsation may be expansile, and usually lies between the second and fourth ribs, anteriorly, but may be seen lower or even in the back. Pulsation is probably propagated from the aorta. The apparent conditions for pulsation in an empyema are cardiac energy, massive exudation and paresis of the intercostal muscles. Empyema pulsans may be confused with aneurysm, which latter is much

slower in its evolution, is much oftener located above and to the right, and has its own signs, murmur and thrill; it may be confused with pulsating abscess, pneumonia, gangrene or tumor of the lung. The pulsation ceases after aspiration. Expansile pulsation of the chest wall has been observed without any organic change (Lafleur and Edwards).

Empyema may rupture externally, *empyema necessitatis,* usually along the sternum or lower ribs. Gravitating abscesses may result, as pulsating tumors in the groin and loin, confusable with aneurysm or spinal caries. Rupture into the air passages may cause suffocation during sleep; 25 per cent. of postpneumonic empyemas rupture into the bronchi. Fistulæ, hectic fever and amyloidosis are frequent sequelæ. Hippocrates knew of the "mouthful" expectoration and also the fact that pus may penetrate the lung like a sponge without perforation or pneumothorax (perforation into the lung without pneumothorax is caused by a small opening which allows the escape of fluid into the lung but not of air into the pleura, because of higher tension in the exudate). Empyema may rupture into the pericardium, peritoneum, esophagus, stomach, etc. Hellin (1905) found 114 cases of *bilateral empyema* recorded in the literature; 67 per cent. occurred in males and 90 per cent. in young persons under fifteen years old; the mortality was 30 per cent.

(*d*) *Putrid pleurisy* is usually associated with gangrene of the lung, pneumothorax, esophageal carcinoma, appendicular abscess and kindred lesions. The fluid is extremely fetid; fat, leucin, tyrosin, cholesterin and sometimes hematoidin crystals are found; the colon bacillus and other gasogenic bacteria are frequently present.

(*e*) *Hemorrhagic pleurisy* is usually serofibrinous and the exudate is tinged with blood. Five to six thousand red cells per c.mm. are necessary to produce a reddish tinge. Its character is seldom suspected before puncture. (i) Most cases are *tuberculous* and Dieulafoy speaks of them as having "hemoptysis into their pleuræ." After puncture more fluid is frequently formed but recovery is possible. (ii) Some cases are *cancerous;* hemorrhagic pleurisy is found in but 12 per cent. of pulmonopleural cancer; pain, dyspnea, luxation and rapid pulse are frequent symptoms; the fluid is dark red and contains little fibrin; a case of Desnos was punctured thirty times in six months. (iii) Some cases occur in *liver cirrhosis,* in nephritis and in hemorrhagic fevers or exanthemata; profuse hemorrhage into the pleura was called pleural hematoma by Wintrich and pachypleuritis hemorrhagica by Chouppe.

(*f*) *Chyliform pleurisy* (*v. i.* CHYLOTHORAX).

2. LOCATION OF THE EXUDATE.—(*a*) *Pleuritis diaphragmatica* produces notable subjective disturbance, as pain which may resemble angina pectoris, pain with tenderness in the epigastrium, between the tenth rib and the xiphoid; tenderness in the lower interspaces near the spine; tenderness over the cervical portion of the phrenic nerve, with pain reflected along the acromial branches of the cervical nerves to the shoulder. An inspiratory spasm of the upper part of the rectus muscle; dysphagia from inflammation of the foramen esophageum; if left-sided, vomiting and singultus may be noted, and if right-sided, icterus is not uncommon.

Cough may occur spontaneously or on pressure. Dyspnea is usually great, the temperature is generally normal, the lower chest moves but little, the respiratory murmur is very weak and friction may be heard over the liver or in Traube's space. If there is no exudate the diaphragm may remain high. Fluid, accumulating between the diaphragm and lung without reaching the chest wall, causes no dulness. Fibrinous is more common than serous effusion.

(*b*) *Interlobar pleurisy* occurs most frequently in pneumonia or tuberculosis. Previous pleurisy may favor this localization by obliterating the general pleural space. As Laennec noted, it is almost always suppurative. Its early diagnosis is uncertain, for the fever, rales, hemoptysis, cough and dyspnea suggest a lesion of the lung, *e. g.*, lung abscess from which differentiation is sometimes impossible; tenderness is frequently elicited along the interlobar fissure; later a zonular surface dulness is found conforming to the fissure, bounded above and below by a normal or somewhat over-resonant note. The heart may be pushed to the right in left-sided localization, but the liver is not luxated in right-sided localization. Interlobar pleuritis may be shown clearly by the *x*-rays. Large effusions may break internally, pus suddenly appearing in the sputum, but spontaneous recovery is infrequent. Puncture may infect the lung during the withdrawal of the needle. The outlines of the lobes are shown in Figs. 33–35.

(*c*) *Mediastinal pleurisy* is most often tuberculous or pneumococcic, and more frequently purulent than serous. The symptoms of lung compression are less distinctive than those of the compression of mediastinal tissues. There may be dyspnea, dysphagia, intrathoracic oppression, a pertussis-like cough, enlarged thoracic veins, deviation of the trachea, and dulness between the scapula and spine; the *x*-ray picture may be suggestive. The sudden onset and febrile course differentiate it from aneurysm, tumor and adenopathies. Without operation the outlook is unfavorable.

(*d*) *Peri-* (*para-*) *pleuritis* is inflammation outside of the parietal pleura, and is usually purulent. Most cases are *actinomycotic*. Fistulæ and the evacuation of the characteristic sulphur granules are common. The symptoms are more local and irregular than those of pleurisy; fluctuation is more frequent; heart dislocation and lung compression are rare; the respiratory excursion is free, and heavy percussion elicits lung resonance beneath the dulness. Metastatic inflammation, perforation and burrowing are common.

3. Pleurisy in Children.—In children pleurisy is attended by more toxemia, higher temperature, pulse and respiration. Bronchial breathing is more frequent; there is less dulness, less luxation and less friction. It often follows pneumonia, where, in the majority of cases, the empyema is more benign; tuberculosis is less frequently causative in children.

Issue.—1. Resorption.—Even purulent pneumococcic pleurisy may exceptionally resorb in part without operation or rupture, leaving inspissated caseous accumulations. Serous pleurisies may be absorbed after three to six months, during which time persistent friction is common over the lower chest. A proteolytic ferment in the white cells dissolves

the fibrin and the serum is taken up by the lymph- and bloodvessels. Dieulafoy reports a case of serous pleurisy, in good health after ten years, which was tapped over 100 times and 230 liters removed.

2. Adhesions.—The thickness of pleural adhesions is more important than their extent. Universal obliteration of the pleural sac by thin

Fig. 33

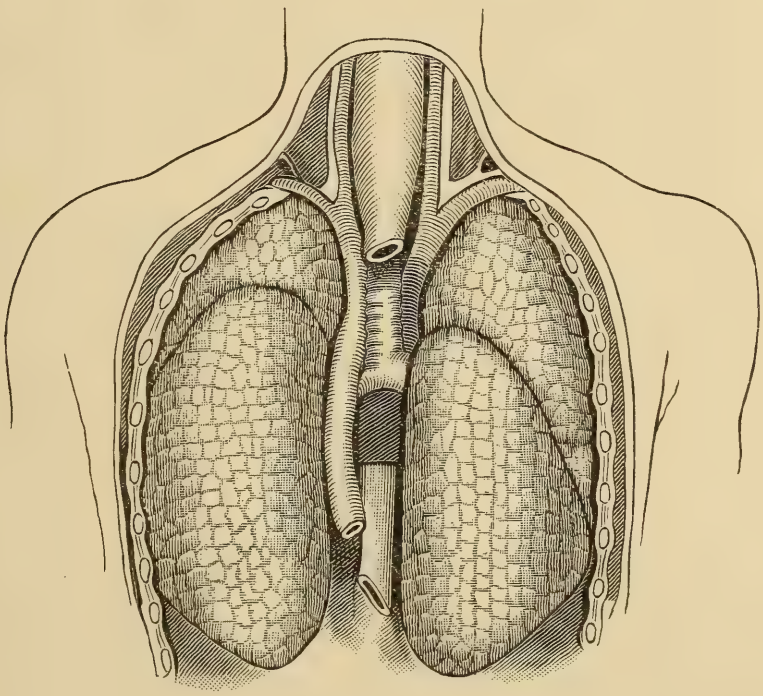


Fig. 34

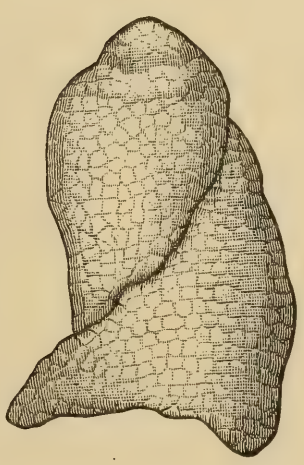
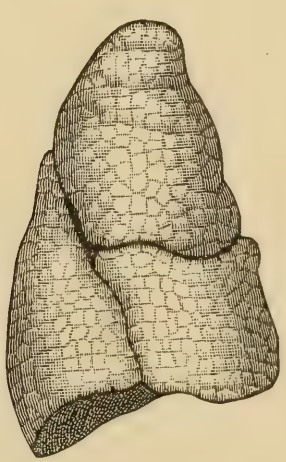


Fig. 35



Figs. 33, 34, and 35.—Cuts from Dieulafoy, showing: Fig. 33, the posterior surface of the lungs and their interlobar fissures; Fig. 34, the lateral aspect of the left lung; and Fig. 35, that of the right lung.

adhesions is of no consequence. Compensatory emphysema, pain, thoracic oppression, obliteration of the complementary pleural space, dulness, decreased vocal fremitus and breathing, failure of Litten's phenomenon and stagnation of bronchial secretion may occur when the adhesions are thick, and stasis may ensue. Peritonitis, mediastinitis

and obliterative pericarditis may complicate the case. In some instances the pleura calcifies or ossifies, leaving a deposit measuring 3 cm. or more in thickness.

3. SUDDEN DEATH may result after exertion, coughing, urination or defecation; it results chiefly from such extreme venous compression that the venous trunks can return little blood to the heart. Less frequent causes are cardiac paralysis or thrombosis, pulmonary edema, and rarely bending of the aorta, pulmonary embolism, rupture of fluid into the bronchi, which may drown the patient during sleep, and death from hemo- or pneumothorax or brain embolism.

4. RETRACTIO THORACIS is characterized by increased resistance, decreased fremitus, decreased circumference of the affected side and often by bronchiectasis. The percussion-note is dull and the breathing is usually decreased, but sometimes bronchial. The causative factors are atmospheric pressure, retraction of the pleural scars, or pleurogenous interstitial pneumonia, also known as pleuritis deformans, which sometimes lobulates the lung. Retraction is generally a slow process but the writer has seen marked deformity develop in three weeks.

5. CHANGES IN OTHER ORGANS.—Retraction may raise the liver and the lung may shrink away from the heart, the spleen and the heart may lie higher and Traube's space may be increased. Organs displaced by exudate may in rare cases remain luxated after the exudate is absorbed. Pleural callosities may produce recurrent laryngeal paralysis which may also follow pressure by an exudate or adenopathy. Other complications are acute and chronic infiltration in the lung; extension to other serous surfaces; septicopyemia; stasis in the liver, stomach, extremities, etc.; chronic nephritis; amyloid degeneration and various visceral or miliary tuberculosis. Scagliari reports 47 cases of brachial paralysis.

Diagnosis.—The physical findings only are final; symptoms are most unreliable. Diagnosis of the malady is the first step, and the second is the determination of its cause.

DIFFERENTIATION.—1. *Pleuritis sicca* is recognized only by feeling or hearing the friction. (*a*) The *crepitant rale* is largely inspiratory, and is heard as a number of small crackles at the end of inspiration; it is altered or removed by coughing and deep inspiration, but the pleural rub is increased by pressure and deep breathing. It may disappear after a number of deep inspirations, which make the two surfaces temporarily smooth. (*b*) It must be differentiated from *sacculated breathing* and the physiological, bilateral crackling in the chest muscles on deep breathing. (*c*) *Intercostal neuralgia* is intermittent, occurs in typical attacks and is limited to the interspaces; Valleix's points are present at the angle of the ribs, in mid-axilla and anteriorly, and correspond to the exit of the three perforating branches of the nerve. Intercostal neuralgia is increased less on deep breathing. (*d*) In *caries of the ribs* pain is localized directly over one rib and not between the ribs. (*e*) Differentiation from *rheumatism* of the thoracic muscles may be difficult, for both affections induce pain on breathing, tenderness and disturbed breathing; there may be a history of exposure or repeated attacks; there is no fever or cough; movement increases the pain more than in pleurisy.

(*f*) The rub in *pericarditis* (*q. v.*) more distinctly depends upon the cardiac movements. In pleurisy along the left border of the heart, over the lingual lobe, on holding the breath the pleuritic rub is heard near the heart, but decreases after a few heart beats. On deep inspiration the pleural rub ceases and the pericardial rub is increased; pericarditis most commonly produces a rub over the conus pulmonalis arteriosus.

2. *Pleuritis humida* (pleuritis effusiva) must be distinguished (*a*) from *pneumonia* (page 74). (*b*) *Tumors of the lung* produce a characteristic exudate; this is often brown, sometimes chyliform, and contains fat globules and large, degenerated cells which may show mitoses; the glands above the clavicle may be enlarged, as well as those in the mediastinum, which produce recurrent laryngeal paralysis. Tumor particles are rarely obtained by puncture or in the sputum, and are only found in carcinoma, for sarcoma rarely ulcerates. A trocar rather than a needle should be used. Unverricht observed the development of carcinoma at the seat of puncture. In tumors there is less displacement, less invasion of Traube's space and less widening of the chest than in pleurisy, and the breathing is often bronchial, the flatness intense and the fremitus increased. (*c*) *Hydrothorax* (*q. v.*), usually described as bilateral, may be unilateral (right-sided), especially in disease of the liver, kidneys and heart. The fluid readily shifts its level on change of posture, which does not occur in pleurisy. The claim that salicylic acid, iodine and potassium iodide pass readily into transudates and but slowly into exudates, is disproved. In hydrothorax the specific gravity is below 1.014, the albumin is 1 to 3 per cent. and the sediment shows few white cells and some endothelial cells. Cytology, etc., may point to hydrothorax, while inoculation reveals tuberculosis. (*d*) The presence of *hemothorax* is determined only by puncture.

3. *Circumscribed pleurisy* requires differentiation (*a*) from *pericarditis;* pleurisy shows a more irregular outline, the apex beat is not altered and there is no pericarditic friction. (*b*) *Lung cavities* show dulness when full of secretion and resonance when it is voided; the fluid obtained by puncture (under a wrong diagnosis) has a higher specific gravity and a larger percentage of fat. (*c*) Differentiation from *splenic tumors* is determined by respiratory excursion and palpation of the lower edge of the spleen. (*d*) *Liver enlargements* rarely occur solely upward, save in in echinococcus and in abscess of the convexity; these are prone to occur anteriorly, and pleurisy more frequently posteriorly. In tumors of the liver there is usually respiratory excursion. (*e*) *Subphrenic abscess* (*v.* Localized Peritonitis) may be intraperitoneal (as from liver abscess) or extraperitoneal (*e. g.*, perinephritic). The liver stands higher in the chest because of the paretic diaphragm. There may be a history or physical signs of an abdominal disease. The needle reaches the pus only when thrust deep in the lower interspaces. The pus frequently has a fecal odor. The manometer shows increased pressure during inspiration and decreased pressure during expiration, the converse of the findings in pleural effusion. (*f*) *Abscess of the liver:* Pleurisy, serous or suppurative, may complicate abscess of the liver. Differentiation is easy when abscess of the convexity is located forward; when it is situated poste-

riorly, aspiration alone will distinguish it; the pus shows liver cells, amebæ, cholesterin or bile. (*g*) 43 cases of *pleural echinococcus* are reported; 11 per cent. of hepatic cysts reach the lung and pleura.

Course and Prognosis.—1. The *course:* Acute cases last two, subacute three to eight and chronic eight or more weeks. The onset is no index of the issue. No cycle is seen in any form of pleurisy. Friction along the lowest limits of the pleura and in front rarely attends great effusions and therefore, after the exclusion of tuberculosis, carcinoma or pyemia, the prognosis is relatively good. In the average case dry pleurisy precedes and succeeds exudation. Persistence, recurrence and exacerbations render the prognosis unfavorable. The author has seen recovery of secondary carcinomatous pleurisy.

2. The *immediate mortality* is under 5 per cent., and depends upon the *etiology* and the *nature of the fluid.* Fibrinous pleurisy is the most favorable type, except when it occurs at the apex of the lung. The outlook in effusive forms is most favorable in metapneumonic serous or purulent pleurisy; next most favorable in serous pleurisy due to the staphylococcus, less favorable in staphylococcus empyema, and least favorable in tuberculous, fetid or putrid forms. It is usually poor in hemorrhagic, chyliform, peracute and bilateral (generally hemorrhagic or purulent) effusions. It is more favorable in effusions relatively rich in solid constituents; effusions containing little solids are apt to recur. Since Hippocrates, the belief has prevailed that right-sided are less favorable than left-sided effusions. *Sudden death* is not infrequent. The author saw 3 cases in one year.

3. Opinions vary as to the *ultimate prognosis;* 87 per cent. of cases become tuberculous in one or two years (Fiedler); 33 per cent. die in five years (Bars); 15 per cent. die of tuberculosis; 90 per cent. are in good health after two to five years; and 80 per cent. are healthy after five years (Cabot).

Treatment.—In *fibrinous pleurisy,* or in the fibrinous or first stage of exudative pleurisy, *pain* is the main indication; morphine may be given in severe cases, especially with hard coughing; the ice-bag, poultices, cups and hot fomentations give little relief; blisters cause as much discomfort as the disease itself; the pleura should be splinted by four strips of *zinc oxide adhesive plaster* two inches wide, overlapping the median line before and behind by about three inches, and running around the chest horizontally and obliquely; these should be applied firmly during expiration, that the lung in its lower part and the pleural surfaces may be well immobilized. Personal experience with the salicylates has not been satisfactory. *The patient should be kept in bed.*

In *serous pleurisy* the patient should be kept in bed and the cough and fever treated expectantly. *Laxatives* are given solely to obviate straining at stool which may induce cardiac weakness; they do not reduce the effusion. If at the end of the second week the effusion remains stationary, various measures are indicated; Schroth's treatment limits the fluid ingested to one quart daily, but the method gives no results, as exudation is an active, not a passive, process; tincture of iodine is seldom beneficial, and when old is distinctly a local irritant; potassium

iodide, digitalis, drastics to produce copious evacuations, cotton jackets and sweats are useless, and pilocarpine is dangerous. Transudates may be removed in this way but not exudates.

ASPIRATION.—Trousseau's indications were (*a*) the vital indication, when life is threatened by a large exudate; (*b*) moderate effusions with slow resorption, and (*c*) persistent or residual exudates. The author believes that *early puncture* and aspiration are indicated in every case, where two quarts or more (2000 or 2500 c.c.) of fluid are effused, whether the temperature is still high or not, and whether dyspnea is present or not; this amount of effusion causes dislocation of the heart, liver or spleen, compression of the lung, torsion of the large vessels, or ectasia of the chest; high intrathoracic pressure may cause sudden death, sometimes without warning symptoms. Early puncture does not irritate the pleura or favor recurrence or suppuration. Puncture was first performed by Hippocrates and exploited by Schub and Skoda (1841), but aspiration dates from Bowditch of Boston (1848).

Method of Aspiration.—After the usual surgical antisepsis, a small trocar or large aspirating needle is introduced, which has an elbow connecting with a rubber tube, to siphon the fluid to a vessel below or connecting with a bottle which can be exhausted by a pump. There is no rule as to the site of puncture, which must be governed by the physical signs; adhesions which hold the lung to the surface vitiate any set law; fluid is usually obtained low down in the chest between the scapular and postaxillary lines. The trocar is introduced perpendicularly to the chest wall and not obliquely, in order to avoid injuring the intercostal artery lying above the rib, as a result of which the author twice saw fatal hematoma and hemothorax. Hypodermics should be at hand, against a possible syncope. The pump is necessary in but 10 per cent. of cases, for the fluid can usually be siphoned readily; a tube is attached to the needle or trocar, filled with boric acid solution, clamped with an artery forceps and unclamped, after its lower end is placed in a basin containing the same solution and held at a lower level. The fluid, if pumped, should be withdrawn gradually; caution is necessary lest air be pumped the wrong way, *i. e.*, into the pleura. The blood-pressure during aspiration should be controlled by the sphygmomanometer. The amount removed need not be great, as moderate relief of intrapleural tension often promotes absorption; it is estimated arbitrarily at one quart (1000 c.c.) to three pints (1500 c.c.). Complete or nearly complete evacuation may cause the accidents enumerated below and is thought by Litten to induce miliary tuberculosis. Aspiration should be *discontinued* when the exudate becomes bloody from lung injury, when friction develops, when pain is felt or when from congestion of the relaxed lung coughing begins. *Repetition of puncture;* a second puncture is necessary in 25 per cent. and a third in 5 per cent. of effusions. *Favorable results* are relief of pressure on the lung and absorption of the exudate, strengthening of the pulse, and return of luxated organs to their place. *Unfavorable results* are (*a*) syncope due to cerebral anemia from reflux of blood to the expanding lung which contained little blood before it was relieved by the puncture, a relatively uncommon occurrence; (*b*) pulmonary

edema—albuminous expectoration—which is due to the sudden withdrawal of too much fluid (2 to 5 liters) at one tapping; it should take a half-hour to evacuate one and a half quarts or 1500 c.c. (Fräntzel); edema pulmonum is treated (and prevented) most successfully by hypodermics of morphine gr. $\frac{1}{4}$; Sears records 10 deaths from simple puncture; Capps and Lewis ascribe sudden death or syncope to the irritation of a pleural reflex; (*c*) sudden death from pulmonary embolism or cerebral embolism; the latter results from discharge of clots from vessels in the relaxing lung, and causes the so-called pleuritic hemiplegia; (*d*) injury to the heart, liver or spleen; (*e*) pneumothorax and subcutaneous emphysema may occur. Doubtless some, if not most, instances of pneumothorax after tapping are primary pneumothorax with secondary pleurisy; the tapping opens up the closed point of rupture and pneumothorax again dominates the clinical picture. (*f*) The author twice witnessed an extensive hemothorax and subcutaneous hematoma after pleural puncture, in which the trocar was impatiently thrust in and out during an unsuccessful thoracocentesis. Naunyn writes of death following puncture of an atheromatous intercostal artery. (*g*) Fibrinous bronchitis has followed paracentesis. (*h*) The needle has been broken by moving the patient during tapping. (*i*) Anaphylaxis has followed tapping. When repeated punctures fail the advisability of operation should be considered, as in empyema. Various modifications are suggested: Holmgren advises two openings, one to admit air and the lower to withdraw fluid; others inject nitrogen to replace the fluid withdrawn, adrenalin to constrict the vessels, etc.

Gilbert's autoserotherapy consists of aspirating 1 c.c. (10 to 20 c.c.), but before the needle is withdrawn entirely, reinjecting the fluid into the subcutaneous tissues; the results are uncertain.

Empyema in all cases necessitates operation (practised by Euryphon and after him by Hippocrates). Rarely, patients may recover spontaneously. A day or two before operation aspiration is indicated when the fluid is under high tension. Some surgeons advocate treating tuberculous empyema without operation, as Volkmann treated other cold abscesses. The mortality in Velpeau's cases was 100 per cent. and Dupuytren himself "chose to die of empyema at the hands of God rather than to die at the hands of man under operation." In Schede's series the mortality of tuberculous empyema was 77 per cent., of metastatic 32 per cent., of metapneumonic 13 per cent. and of primary 8 per cent.

After-treatment is directed toward prevention of thoracic retraction, to which end gymnastics and deep breathing are essential. A patient having suffered from a "primary" effusive pleurisy should be treated as one with latent tuberculosis.

PNEUMOTHORAX.

Definition.—Pneumothorax is an accumulation of gas or air in the pleural sac. It was named by Itard of Paris (1803) and was described fully by Laennec (who in 1819 made the first diagnosis) and Skoda. Hippocrates described succussion. As it excites pleurisy in 90 per cent.

of the cases, the following varieties are distinguished: *pyo-*, *sero-* and *hemopneumothorax.*

Etiology.—1. *Diseases of the lung and pleura* cause about 95 per cent. of all cases. In Biach's collection of 918 cases in Vienna, pulmonary *tuberculosis* caused 77 per cent., gangrene 7 per cent., empyema 5, trauma 3, bronchiectasis and abscess each 1 per cent., the etiology was unknown in 2 per cent. and the remaining 4 per cent. were due to emphysema, infarct, thoracocentesis, echinococcus and contiguous disease perforating into the pleura, as gastric or esophageal ulceration or caries of the sternum. (*a*) Acute is more important than chronic tuberculosis in which there are protecting pleural adhesions; pneumothorax develops in 5 per cent. of tuberculous cases. (*b*) Ulcerative lesions of the lung may erode the pleura and admit air (*v. s.*). (*c*) Trauma or stab wounds occur far less frequently than one would expect, and here hemopneumothorax is the type. (For "pneumothorax following paracentesis," see page 468.) (*d*) Empyema may rupture into the lung without allowing ingress of air from the opposite direction. (*e*) Rupture of a healthy lung by tearing away of the lung, as pleural adhesions give way in coughing, muscular straining or violent coughing is very exceptional.

2. *Perforative lesions in the mediastinum,* esophagus, peritoneum, stomach or colon and subphrenic abscess are uncommon causes (*v. s.*).

3. The view held by Oppolzer and Biermer that pneumothorax may occur *spontaneously* (without rupture) has received confirmation by discovery of the *Bacillus aërogenes capsulatus* and other gasogenic organisms (Welch, Lewy). It is always possible, however, that the point of rupture in the lung has healed; the fact remains that air enters the pleura chiefly from the lung or from a gas-containing organ.

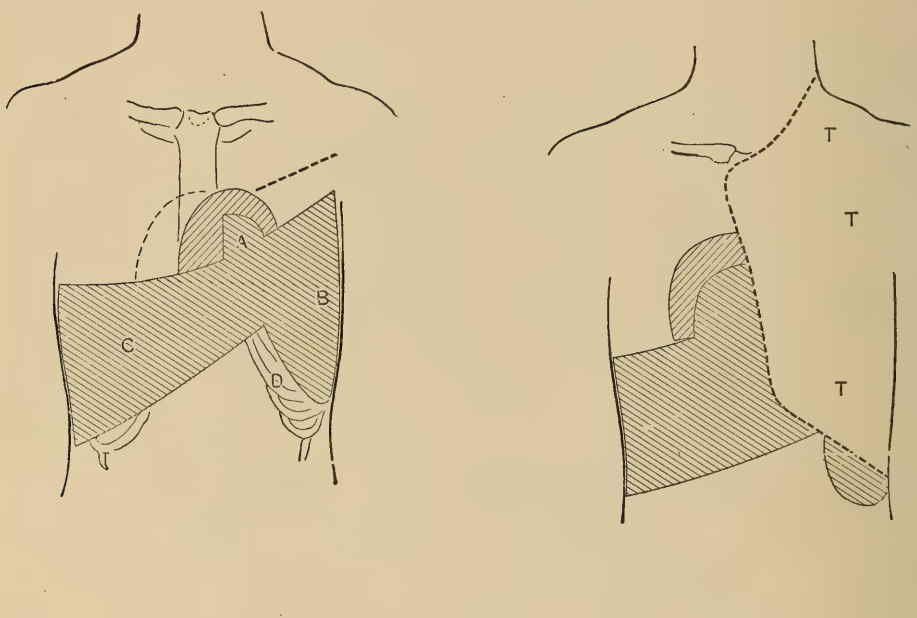
Pneumothorax occurs largely in *adults* and four to seven times as frequently in *males* as in females. Cozzolino (1906) found only 41 cases in children; of these but 40 per cent. were tuberculous.

Pathology.—When the chest is opened, air or gas escapes with a force sufficient to blow out a candle. The volume of air may exceed 2000 c.c., and its tension, studied first by Wintrich and Weil, collapses the lung—overcoming the normal negative pressure of 3 to 6 mm. Hg., due to its elasticity—dislocates the heart and mediastinum and depresses the diaphragm and subphrenic organs. The gas may be odorless or fetid. The pathological and clinical findings vary (*a*) as the gas is encapsulated or occupies the entire pleural cavity; (*b*) as the air is aseptic (producing a pure pneumothorax, or one with slight exudation) or is infected by the organisms of the primal disease which excite serous, purulent or putrid pleurisy; (*c*) as the *air-containing* cavity is "closed," "open" (into a bronchus, or sometimes also externally) or possesses a valve, allowing passage of air in one but not in the opposite direction (*v. i.*).

Symptoms.—The *onset* is usually *sudden,* occurring spontaneously or following coughing or exertion; it is characterized by *sudden pain in the side, cyanosis, dyspnea* and *collapse,* due to reflex action on the vagus; the onset may resemble angina pectoris, pulmonary embolism or abdominal perforation; there may be sudden evacuation of pus from the

breaking into the lung of an empyema; urticaria occasionally develops; decreased diuresis, albuminuria, stasis and subcutaneous emphysema are sometimes observed. The onset may be insidious—with no symptoms and with only physical signs; indeed pneumothorax sometimes constitutes an unexpected postmortem finding. In other instances the history or course may justify a presumptive diagnosis, as in a case clearly tuberculous; and in still other cases, a small, latent caseous focus may burst and thus produce its first symptom.

Physical Findings.—1. INSPECTION.—(*a*) The *affected side is distended* 2 to 4 inches and *immobile,* and contrasts sharply with the widely moving sound lung. (*b*) The *interspaces* are *obliterated.* (*c*) The *heart* and *liver* or spleen are *dislocated.* (*d*) There is *dyspnea* and *cyanosis.* (*e*) The



I II

FIG. 36.—I. Left-sided effusive pleurisy, showing relative heart dulness (*A*), effusion (*B*), liver flatness (*C*), and fluid impinging on Traube's half-moon space, of which but a small part remains resonant (*D*). In larger effusions reaching, *e. g.*, up to the dotted black line, the relative heart dulness may be pushed over to the dotted line (over *C*). II. Left-sided pneumothorax marked by tympany (*T*, *T*), pushing over the mediastinum and also the heart to the right, and pushing down the left lobe of the liver and the spleen.

attitude is characteristic; the patient most often lies *on the affected side* to afford ample play to the sound lung; sometimes there is orthopnea and if the point of rupture opens into the lung or bronchus a peculiar decubitus may be assumed in order to drain the pleura. (*f*) The *x*-rays show the *lung flattened* near the spine, the *diaphragm depressed* and *motionless,* and the shadow of the effusion.

2. PALPATION.—(*a*) *Vocal fremitus is absent,* unless adhesions hold the lung to the chest wall. (*b*) The *chest wall feels resistant* and (*c*) sometimes *splashing,* fluctuation or a "pillow sensation" is noted. (*d*) The *dislocated apex beat* and the edge of the liver or spleen are felt.

3. PERCUSSION.—Results vary according to the tension of the gas and chest wall: (*a*) In *open pneumothorax* the note is tympanitic or even metallic when percussion is made on a solid object, as a coin; this

bruit d'arain is found in 75 per cent. of cases; cracked-pot resonance and Wintrich's change of note may be elicited when the mouth is opened. (*b*) In *closed pneumothorax* the note is full and loud; it is tympanitic when there is moderate pressure and the intercostal muscles are relaxed; again the note may be *dull* if the tension is great or if the chest muscles are spasmodically contracted, as in sudden pneumothorax (vagus reflex action). (*c*) *Flatness* over the lower thorax usually results from pleural effusion. The author has seen tympany over the entire side, in great pleural effusion. Skoda said that the fluid is twice as much as we anticipate. (*d*) On change of posture a *changing level of the fluid* is readily elicited, the gas being found above, like a spirit-level, and the fluid below. (*e*) The *pitch* over the gas-distended part *changes with change of posture* (Biermer's change of note). It becomes deeper as the patient sits, because the vertical dimension of the chest is increased by the pushing down of the diaphragm. (*f*) The *heart dulness* is *replaced by resonance* in left-sided pneumothorax, and is found to the right of the sternum; in right-sided pneumothorax the luxation is less and toward the left axilla. (*g*) The *liver* or *spleen dulness* is *pushed* well *downward.*

4. Auscultation.—(*a*) The *breath sounds* are usually *absent* in the closed variety of pneumothorax, and this, with ectasia and hyperresonance, is most characteristic. In the open and sometimes in the closed form, the breath sounds are *bronchial,* amphoric or metallic, the voice sounds, especially under the clavicle, are metallic, and coincident rales in the bronchi are also metallic. Amphoric sounds were referred by Laennec to a fistula but probably are due to the air acting as a sounding board. (*b*) Air passing through the point of rupture may produce curious *snapping or whistling sounds;* the point of rupture is oftenest between the second and fourth interspaces and the mammary and axillary lines. (*c*) The *gutta cadens* (the metallic "falling drop") may be due to rales or dripping of fluid from fibrin shreds as the posture is changed. (*d*) The Hippocratic splashing (*succussio Hippocratis*) was carefully described by Laennec and is elicited by placing the ear to the chest and sharply shaking the patient; in one of James's cases it was heard over a large clinical amphitheatre; three patients called the author's attention to a "splashing in the chest"; it is heard in three-quarters of the cases, usually before dulness develops. (*e*) The *heart tones* may be *metallic,* and the heart's action may even cause splashing sounds. Hellin collected 54 cases of double pneumothorax; it is not incompatible with life.

Diagnosis.—The ectasia, resonant note, suppressed or amphoric breathing, luxations and succussion are characteristic.

Type.—Dislocation occurs in all types, though it varies in degree. (*a*) In the *open form* Wintrich's change of note, metallic sounds, fistula murmur, periodic expectoration of large amounts of pus, or of methyl blue injected into the pleura, moderate cardiac and other dislocations and no manometric increase of the tension in the pleura are distinctive. (*b*) The *closed type* is distinguished by increased manometric tension, absence of breath sounds, marked ectasia, change of note on sitting but not on opening the mouth and marked luxations. (*c*) In the *valvular form* air may enter the pleural cavity but cannot escape back and there-

fore pus cannot be voided. Some of the auscultatory findings of the open type are noted. The manometer shows increased pressure.

DIFFERENTIATION.—(*a*) *Cavities* may be simulated by circumscribed pneumothorax, but the interspaces are sunken, the fremitus is increased when the cavities are empty, luxation is absent and the Hippocratic succussion and coin sound are extremely rare. (*b*) *Pleurisy* can be distinguished on careful examination. (*c*) *Meteorism* is at once excluded by filling the stomach or colon with water. (*d*) *Diaphragmatic hernia* is very frequently confused with pneumothorax; out of 481 cases only 12 were recognized. The *x*-rays determine the condition. Respiratory excursion is present and the tympany and metallic sounds are accompanied by symptoms of incarceration, as vomiting, colic, obstipation and indicanuria. The intestines push the heart to the right. Ninety per cent. of cases are left-sided. (*e*) *Pyopneumothorax subphrenicus* (*q. v.* under PERITONITIS, LOCALIZED FORMS), results from abdominal lesions, such as cancer, ulcer, appendicitis, disease of the gall tracts, etc., whose histories are suggestive. Thoracic symptoms, as cough, sputum and dyspnea, are not conspicuous early in the process and the lungs may show perfect excursion. The manometric pressure is increased during inspiration and decreased during expiration; the converse is true in pneumothorax. By the *x*-rays the diaphragm is above the fluid, while in the pneumothorax it is below the fluid.

Prognosis.—The prognosis varies with the cause, the condition of the lungs and the character of the fluid. Cases in a healthy lung following exertion, trauma or infarct are relatively favorable. The closed and open are more auspicious than the valvular type. In tuberculosis, pneumothorax seems to arrest the tuberculous focus if it is small and if no mixed infection occurs; even in these cases complete recovery is infrequent. Eichhorst observed one case which lasted for five years. Gabb's case recovered and relapsed four times, the attacks being two to four years apart. West observed a mortality of 70 per cent., of which three-quarters died within two weeks, nine-tenths in less than a month and one case in twenty minutes.

Treatment—The results are disappointing. (*a*) Narcotics and stimulants are imperative at the time of the rupture, for collapse, dyspnea and cyanosis. (*b*) Aspiration is of most value in the serous forms; early aspiration may open a healing fistula by relieving the pressure which closed it. As a rule aspiration is resorted to only when pressure menaces the heart and lungs, and when pyothorax threatens sepsis. (*c*) The radical operation is indicated in putrid or purulent pneumothorax. Leyden reported 66 per cent. and Richardière 50 per cent. of recoveries.

HYDROTHORAX.

Definition.—Hydrothorax is a serous transudation into the pleura.

Etiology.—(*a*) *Stasis*, from cardiac, pulmonary or vascular factors, is a frequent cause. In failure of the right heart the superior cava is imperfectly drained, which congests the azygos and hemiazygos, leading in turn to pleural transudation. It may occur alone but is oftener

part of a general dropsy. Local venous obstruction from mediastinal tumor or aneurysm may induce hydrothorax. (*b*) *Blood and vascular changes* resulting from multitudinous anemic, marantic or cachectic states, as nephritis, cancer and blood diseases. The chief factors are cardiac and renal.

Symptoms and Diagnosis.—1. The *symptoms* of hydrothorax do not include pain or fever. The condition accentuates the dyspnea attending the causal stasis or cachexia. The amount of fluid varies from ounces to quarts (or even to two gallons).

2. The *physical signs* are those of moderate effusive pleurisy, except that the rub is absent, the fluid shifts with change of posture and produces a moderate compression of the lungs and luxation of the heart, liver and spleen. The transudate is usually described as bilateral, but in over half the cases it is unilateral (adhesions obliterating the opposite pleura) and often right-sided, in cardiac, renal and hepatic disease, by pressure on the azygos or pulmonary veins by the dilated heart or cava or the luxated root of the lung.

3. The fluid is clear, slightly green, possibly moderately blood-tinged if it develops during the death agony, and shows a specific gravity of 1.010 to 1.015; at the most it develops only light delicate flocculi of fibrin; the albumin ranges between 1 and 3 per cent. Centrifuging brings down only a few leukocytes, endothelial cells and red disks, but no bacteria. Right hydrothorax may prove very obstinate, because a low grade of inflammation eventually supervenes.

The treatment is causal; aspiration often prolongs life.

HEMOTHORAX.

Blood effusion into the pleura is a rare malady. It may be caused by trauma, scurvy or allied blood diseases, aneurysmal rupture, ulceration of the pulmonary or pleural veins and by various necrosing lung diseases. It may occur with pleurisy, pneumothorax or thoracocentesis. Its symptoms are essentially those of hydrothorax; it is suspected only by a sudden onset with signs of acute anemia, and is proved only by the exploring needle. Its treatment is symptomatic, stimulants, ice applied to the chest, horse serum for hemostasis or operation for trauma.

CHYLOTHORAX.

A very rare affection, in which true chyle or chyliform fluid is found in the pleura. Its etiology is disputed; (*a*) in genuine cases chyle containing sugar is found (though sugar has been found in many forms of serositis and in most transudates); only 60 cases are recorded. In 11 cases (of which 4 died and 9 were right-sided), traumatic rupture of the thoracic duct has been found. In others the duct was obstructed by thrombosis of the subclavian vein or by glands and tumors. (*b*) In a second group, the pseudochylous (chyliform or adipose), there is no chyle and the milky appearance is due to fatty or minute albuminous granules held in suspension. This variety is chiefly tuberculous or

carcinomatous. It has been thought that old empyemas may become chyliform. The characters of both forms of fluid will be considered under chylous and adipose ascites, with which they are frequently associated.

PLEURAL TUMORS.

Pleural tumors are nearly always secondary by contiguity or metastasis to pulmonary, mediastinal or extrathoracic growths. Carcinoma and sarcoma are the most frequent forms; enchondroma, lipoma, hypernephroma and dermoids are very uncommon. Of primary endothelioma of the pleura, 50 cases are reported, but it is considered by some as solely inflammatory; clinically it presents the picture of chronic pleurisy plus occasional metastatic deposits in the liver, lungs, kidneys, muscles and lymph glands. The signs are those of lung tumor plus those of pleurisy; the diagnosis of primary tumor is impossible.

DISEASES OF THE MEDIASTINUM.

MEDIASTINAL TUMORS.

Mediastinal tumors are very rare. They are usually primary. Sarcoma is the most common form and then carcinoma; lipoma (7 reported cases), substernal struma, thymic growths, fibroma, myoma, osteoma, enchondroma, echinococcus, dermoid (64 cases collected by Christian, 1908), teratoma, gumma, tubercle, leukemic, pseudoleukemic and chloromatous adenopathies are rarer forms. Sixty per cent. occur in males between twenty and thirty years of age.

Symptoms.—Symptoms develop gradually. 1. *Local signs:* (*a*) Bulging may be present in the jugulum or under the sternum, especially in lymphosarcoma. (*b*) There may be pulsation, imparted by the heart, aorta or its branches; it is rarely expansile. (*c*) The tumor or secondarily involved glands may be palpable deep in the jugular notch. The vocal fremitus varies. (*d*) The sternum may be exquisitely tender from erosion. (*e*) Dulness may be elicited under or near the sternum, or, if the posterior mediastinum is involved, in the back; the dulness may blend with the heart, suggesting aneurysm or effusive pericarditis, though far more irregular. (*f*) Sometimes there is bronchial breathing, or if the bronchi are closed the breath sounds may be absent.

2. *Compression symptoms,* as in aneurysm, may predominate in tumors of the posterior and middle mediastinum, though local physical findings may be slight or absent. (*a*) Paroxysmal asthma and orthopnea are common; dyspnea is the earliest and most frequent symptom, due partly to compression of the vagus and partly to tracheal, cardiac and pleuritic involvement. Other vagus symptoms are brazen cough from paralysis of the vocal cords, tachy- or bradycardia, hiccough, vomiting or esophageal spasm. (*b*) The trachea or bronchi may be compressed from without

or occluded by growth into their lumina, in which case the symptoms of tracheal or bronchial stenosis may be pronounced. Tracheal tugging and shrinking of one side of the chest (instead of bulging) may be noted, perhaps with dulness of the right apex. The sputum may contain blood, tumor tissue or hair. The larynx may show lateral deviation. (*c*) The arteries of the neck may show a systolic stenotic murmur, but the veins are more often compressed, especially the innominates or the superior cava, which cause bilateral or unilateral edema and, less often, cyanosis of the face, neck and arms; enlargement of the veins on the thorax indicate the collateral circulation. (*d*) The heart may be compressed and dislocated, downward and outward if the growth emanates from the anterior mediastinum, forward if from the posterior mediastinum. Dislocation of the liver and spleen are less common. (*e*) Compressive dysphagia may cause death from inanition. (*f*) Inequality in the pupils is uncommon. Neuralgia, muscular paralysis or even spinal paraplegia may result. Boring pain is rare.

Diagnosis.—A diagnosis is positive from the signs or presumptive from the symptoms. Dyspnea, ectasia, irregular substernal dulness, venous stasis, bronchial stenosis, heart luxation and the skiagram make the diagnosis probable; puncture may withdraw tissue or dermoid material; the sputum sometimes voids tumor particles, hooklets or hair. Deep aneurysm (*q. v.*) is difficult to eliminate. The irregular dulness excludes pericarditis. Tumors of the lung and pleura produce less pressure symptoms, though such differentiation is rather academic.

Prognosis and Treatment.—The prognosis is unfavorable, as death from inanition, compression, exhaustion or pneumonia results within a few months. Benign tumors run a longer course, sometimes five to seven years, or even forty in Lebert's dermoid. Treatment is palliative. A few successful operations are reported; in 20 operated cases of dermoid, 70 per cent. recovered (R. S. Morris).

MEDIASTINITIS.

Acute inflammation is uncommon, but may follow various local lesions, as pleurisy, pericarditis, pneumonia, acute lymphadenitis and kindred processes, by contiguity or by metastasis. (*a*) Acute non-suppurative cases begin with chill, fever, pain in the mediastinum and tenderness anteriorly over the sternum or high in the back; there may be edema over the sternum; dysphagia, dyspnea and vague cardiac symptoms may develop. A presumptive diagnosis is made from acute inflammatory symptoms with mediastinal localization. The prognosis is grave and the treatment is symptomatic; an ice-bag should be applied over the sternum and opiates should be given for pain. (*b*) Of *mediastinal abscess*, Hare (1899) collected 115 cases in most of which the anterior mediastinum was affected. Acute cases, most often traumatic or sequential to eruptive diseases, were more frequent than chronic cases, which were usually tuberculous (*v.* MEDIASTINAL PLEURISY).

Symptoms.—The symptoms are mediastinal, as in simple acute mediastinitis, and general or septic. A throbbing retrosternal pain is common

and dyspnea may result from pressure by large pus pockets. Edema, fluctuation, pointing or rupture in the upper interspaces or in the jugular notch may be noted. Fatal hemorrhage may follow simultaneous erosion of the aorta and bronchus. Internal rupture also occurs into the air passages, esophagus or pleura, or deep burrowing to the abdomen. The exploring needle is of great diagnostic aid in doubtful cases. Auvray (1904) found in the literature 36 operations with 33 recoveries.

(*c*) *Chronic mediastinitis* is discussed under Adhesive Pericarditis.

MEDIASTINAL HEMORRHAGE.

Small hemorrhages of no clinical importance may occur in icterus, hemorrhagic blood diseases or acute infections. Larger hemorrhages may follow trauma or erosion of the large vessels, both of which overshadow the mediastinal incident.

INTERSTITIAL EMPHYSEMA.

(See DIFFERENTIATION OF EMPHYSEMA.)

SECTION IV.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Etiology.—Simple, acute stomatitis is the most frequent form. In children it may develop with dentition, rude cleansing of the mouth and digestive disorders, especially in the poorly nourished; it is part of the exanthemata and syphilis. In adults it results from hot food, alcohol, tobacco, iodine, mercury, arsenic, etc., acidulous vomiting, carious teeth, throat disease, uncleanliness, etc.

Symptoms.—Symptoms are chiefly redness, swelling and increased secretion, and in the acute forms pain which makes eating difficult. The increased and usually acid saliva irritates the lips or chin if it flows over them. The gums are turgid (*gingivitis*) and the tongue broadened, lax and indented by the teeth, as is also the buccal mucosa. The tongue is coated. Vesicles sometimes form and break down into small erosions. The whitish areas sometimes seen are epithelial thickening. Fever, thirst and general symptoms are absent, except in some infantile cases. Acute stomatitis lasts but a week or two. Transient stomatitis is physiological in the newborn.

Treatment.—In children the mouth should be gently washed after each nursing. Boracic acid is excellent and safe. In adults the teeth should be cared for. Cold water or ice mitigates pain. Tincture of myrrh and of rhatany (equal parts) are excellent. Stronger antiseptics may be used in adults but children may swallow them; for instance, 2 per cent. chlorate of potash, or 1 per cent. phenol; erosions may be touched with 1 per cent. silver nitrate.

STOMATITIS ULCEROSA.

Etiology.—Its bacteriology is unknown. In some cases the microorganisms of Vincent's angina are found. Epidemics may occur in asylums or prisons. It may be induced by malnutrition, uncleanliness, oral or dental disease, phthisis, leukemia, diabetes, toxic factors (mercury) and dentition. (See Acute Pharyngitis and Diagnosis of Diphtheria.)

Symptoms.—The symptoms are those of catarrhal stomatitis plus necrosis and ulceration. The gums, especially about the incisors and canines, are red, swollen and spongy; they bleed, secrete pus and ulcerate; over the ulcers a membrane develops. The teeth are exposed and loosened. The tongue and cheek ulcerate less often and the pharynx is nearly exempt. The breath is fetid, the saliva is increased, the submaxillary lymph glands and salivary glands are swollen and mastication is almost impossible. Fever, prostration and signs of sepsis may mark the severest cases. Acute cases last one or two weeks and chronic cases weeks or months.

Varieties.—(*a*) *Mercurial stomatitis* may be industrial or therapeutic. The author saw a case in which five grains of calomel caused necrosis of the tongue, which rotted off at its root. (See INTOXICATIONS.) Mercurial and other stomatitis may cause "erosions" of the enamel in the developing teeth of children; they become pitted, discolored and transversely furrowed. (*b*) In *Riga's disease,* described by Riga (1881) in South Italy, there is a pearly induration between the tip of the tongue and frenum; it appears with the first dentition, sometimes ulcerates, is occasionally epidemic, is often observed with the status lymphaticus and is invariably fatal. (*c*) In *nursing women* ulcers may develop from the lymph follicles of the lip and cheek; they usually indicate malnutrition, sometimes are painful and respond to tonics and hygiene. (*d*) *Herpetic or pemphigoid stomatitis* is seen in neurotics and is frequently recurrent (Jacobi's stomatitis chronica neurotica). (*e*) Bednar described (aphthous) *white plaques* on the posterior part of the hard palate near the alveolar border of the jaw, which may ulcerate; they occur in the first three months of life, and are apparently due to sucking. Fatal cases are due to hemolytic streptococci. (*f*) *Parrot's ulcers* occur in marantic newborn children on the hard palate near the median line; they seldom heal.

Treatment.—Potassium permanganate 1 to 1000 relieves the *fætor ex ore.* Necrotic areas may be touched with silver nitrate solution or equal parts of tincture of the chloride of iron and glycerin. Potassium chlorate gargles may be combined in children with gr. x–xx every day internally and twice the amount for adults. Opium and belladonna are indicated by pain and salivation.

APHTHOUS STOMATITIS.

Etiology.—It may occur sporadically or epidemically. It is probably contagious; when due to milk it ceases on its withdrawal. Besides the causes of stomatitis (*v. s.*), it occurs chiefly in children between ten and thirty months old.

Symptoms.—Fever, thirst, salivation and pain in the mouth precede the round, yellowish-white and slightly elevated areas as large as a pea on the tongue and less often on the lip and buccal mucosa. The mucosa is slightly reddened about them. Attempts to scrape them off produce bleeding. They are areas of superficial necrosis into which exudation of fibrin and round cells occurs (stomatitis fibrinosa disseminata). Vesicles

are no part of the process, though they may occur on the skin near the mouth. Occasionally there is tumefaction of the lymphatic and salivary glands. They last one or two weeks, and recurrence is frequent. The *treatment* is that of stomatitis.

PARASITIC STOMATITIS.

Etiology.—*Thrush, muguet, soor* or *stomatomycosis oïdica* is due to the Oïdium albicans, closely allied to the saccharomyces, which consists of branching mycelia and ovoid granular and refracting spores. It occurs chiefly in young or marantic children whose mouths have been improperly cared for, especially among the poor and in institutions. Thrush may develop in tuberculous and diabetic adults.

Symptoms.—A few punctate, whitish spots develop on the tongue, cheek and soft palate; they become yellowish as the areas grow or fuse. The fungus is at first deposited on the epithelium, between the layers of which it proliferates and penetrates. The mouth is dry or moist from increased salivary flow. In severe cases the fungus may reach the pharynx, larynx, esophagus and even the stomach and intestine. It is generally limited to areas with flat epithelium, and epithelia of the cylindrical and ciliated order usually escape. It may, in exceptional cases, cause esophageal occlusion, bronchopneumonia, cerebral, renal, and other embolism. The *outlook* is good except in marantic subjects.

Treatment.—*Prophylaxis* is most essential in regard to *sterilization of bottles and nipples* and maintenance of the general health by fresh air and early treatment of dyspeptic disorders. Stomatitis may persist until a wet-nurse is secured. Cleansing the mouth with *borax*, sodium bicarbonate and lime-water is most effective. If unusual extension occurs a teaspoonful of a resorcin solution (1 to 200) may be given every two hours.

Stomatitis may in rare cases result from the sarcina, leptothrix, pneumococcus, gonococcus, etc.

GANGRENOUS STOMATITIS.

Etiology.—Gangrene of the cheek, *noma* or *cancrum oris*, is a rare affection. Of Brun's 415 cases, only 11 were over fifteen years of age and only 6 infants; the author saw noma in a man forty years old. More cases occur in girls. *Malnutrition* causes some cases; 50 per cent. follow measles; typhoid, scarlatina, etc., are less frequent factors. An organism resembling that of diphtheria (Bac. necroseos) and those of Vincent's angina may be seen.

Symptoms.—Ulceration of the gums or buccal mucosa near the angle of the mouth is followed by rapid necrosis, gangrene and extensive brawny edema of the face. The fetor is intense and much tissue is frequently sloughed off. Perforation of the cheek, necrosis of the jaw and extension to the throat, orbit or ears are not uncommon; 70 per cent. die within a week with septic symptoms as fever, weak heart, diarrhea,

colitis and meningitis or lobular pneumonia. The mortality was 92 per cent. in Springer's collation.

Treatment.—Treatment is ineffectual. A full diet, alcohol (salvarsan) and disinfectants applied to the necrotic focus are indicated; excision, the live cautery and caustics are employed without success and sometimes aggravate the necrosis.

DISEASES OF THE TONGUE.

ECZEMA.

In eczema the epithelium thickens and desquamates, leaving red, smooth and circular, serpiginous or geographical areas, which heal centrally and extend peripherally. It may cause local irritation or worry the patient, who may think he has syphilis or cancer. Again, it is an accidental finding. Its cause is unknown, its course is chronic and its treatment by silver nitrate is quite inadequate.

LEUKOPLAKIA.

Leukoplakia (psoriasis, ichthyosis, keratosis) closely resembles the "geographical tongue," in connection with which it is often described. Irregular, smooth, pale plaques of thickened epithelium measuring 1 or 2 centimeters, develop on the tongue and less often on the lips, cheeks or tonsils. Eighty to ninety-eight per cent. occur in men; tobacco, alcohol, gout and gastric disease are probable factors; Fournier's experience with 324 cases convinced him that 80 per cent. came from syphilis and 20 per cent. from tobacco. Its symptoms are slight, its course very chronic and its treatment uncertain; salvarsan is suggested. Sodium salicylate, 10 per cent.; chromic acid, 5 per cent.; strong silver nitrate; corrosive sublimate (1 to 500 parts) and the galvanocautery are recommended. Excision is necessary for papillomatous or epitheliomatous outgrowths which are said to develop in one-third to one-half the cases.

ACUTE GLOSSITIS.

Acute glossitis may be localized or diffuse, and ends in suppuration in 33 per cent. It follows burns, erosions, trauma or the entrance of pyogenic cocci through the lymph follicles. The painful, swollen tongue may protrude beyond the lips. In 145 cases the mortality was 3 per cent. (Bennett, 1906). Eating, speaking and oral breathing are impossible. Incision is indicated.

GLOSSITIS DESICCANS.

Glossitis desiccans is a rare, chronic, intractable affection in which deep indentations of the margins of the tongue and consequent lobulation

occur. *Melanoglossia* (*nigrities linguæ*) results from proliferation of the filiform papillæ at the base of the tongue (*melanotrichia linguæ*, black-haired tongue); 50 cases are on record; the hair is removed by a 10 per cent. salicylic acid solution or by curetting. The *mucor niger* may cause a black coating on the tongue.

DISEASES OF THE SALIVARY GLANDS.

PTYALISM, SALIVATION, HYPERSECRETION, SIALORRHEA.

Etiology.—The chief causes are (*a*) disease of the mouth, stomatitis, dentition; (*b*) toxic substances, as jaborandi, tobacco, mercury and iodides; (*c*) nervous affections, as trigeminal neuralgia, paretic dementia or hydrophobia; a distinction is drawn between oversecretion and lesions like bulbar paralysis, in which the saliva cannot be swallowed; (*d*) reflex salivation, which may occur from diseases of the tympanum through the chorda tympani, from gastro-intestinal disease (ulcer, pancreatic lesions, etc.) by way of the vagus and sympathetic nerves and from genital conditions, as pregnancy.

Symptoms.—A pathological salivary flow of over twelve quarts has been recorded. The salivary glands are tense, swollen and hard. The reaction of the saliva may become neutral or acid; it may contain ropy mucous or scattered pus cells; the ptyalin and potassium sulphocyanide disappear. The saliva may trickle into the larynx during sleep, and cause coughing; it may flow into the stomach and produce vomiting of an alkaline or neutral vomitus, especially in the morning; it may stream from the lips and redden the skin.

Prognosis and Treatment.—Both depend on the causal affection. In nervous and reflex types potassium bromide and iodide are often valuable. Severe cases necessitate opium and belladonna.

XEROSTOMIA.

Hyposecretion or arrested secretion occurs in diabetes insipidus and mellitus, in some cases of contracted kidney and as a neurosis or a neurotic symptom (Hutchinson, 1888). Women are more often affected (80 per cent.). The mouth is dry, red and glistening, interfering with speaking, chewing, swallowing and digestion of carbohydrates. The tongue may cleave to the roof of the mouth. *Treatment* consists in painting the mouth with iodine, potassium iodide and glycerin (1, 10, 100 parts), pilocarpine hydrochloride, grain $\frac{1}{12}$ to $\frac{1}{8}$, and galvanism to the parotid.

PAROTITIS.

Besides specific parotitis (mumps), infection may reach the salivary glands by way of their ducts or the blood stream. *Acute parotitis* may be

simple or oftener suppurative (staphylococcic). (*a*) Any acute infection may be complicated by parotitis, particularly typhoid. (*b*) Paget drew attention to diseases of, or operations on, the alimentary and genito-urinary tracts, peritoneal disease, pancreatitis, abdominal trauma, etc.; many of these cases are clearly attenuated sepsis. Zezas, in 1910, collected 162 cases of postoperative parotitis; the mortality is 33 per cent. (*c*) Facial neuritis and the starvation therapy of gastric ulcer may cause parotitis.

Chronic parotitis sometimes follows epidemic or symptomatic parotitis, poisoning by mercury or lead, syphilis or chronic nephritis. Mikulicz described a "chronic symmetrical hypertrophy of the salivary and lachrymal glands," which may be independent or symptomatic of pseudoleukemia, leukemia, etc. There is a round-cell infiltration, epithelial degeneration, and structures like giant cells.

Treatment.—In acute parotitis, leeches, cold and later, hot fomentations and surgical incision, if there is pus formation, are indicated. Chronic forms respond slowly to iodine or mercurial ointment, and Mikulicz's disease to arsenic and *x*-rays.

ANGINA LUDOVICI.

Ludwig's angina is a streptococcic infection beginning in the submaxillary gland, and extending to the floor of the mouth and the cervical cellular tissue (Ludwig, 1836). Trauma may be its immediate antecedent, but most cases are secondary to typhoid, diphtheria and kindred infections. The inflammation spreads rapidly, causing fever, redness, pain and swelling under the tongue, cellulitis and sometimes gangrene in the neck (*cynanche gangræneuse*). Mastication and deglutition may be impossible. The parotids are sometimes invaded. Resolution is very exceptional and external pointing of pus is not likely to occur, whence, without early surgical intervention, septic and pyemic complications or laryngeal and pharyngeal edema or phlegmon (of which it is the analogue) very frequently develop. The mortality is 40 per cent.

SIALODOCHITIS FIBRINOSA.

Inflammation of the salivary ducts with formation of membrane (Kussmaul) results from infection ascending from the oral cavity and is characterized by fever, tenderness, pain and tumefaction of the gland. The duct is kept patent by pressure or sounding.

SIALOLITHIASIS.

Stones of calcium phosphate or carbonate occur usually from stagnation of the salivary secretion by foreign bodies, bacterial invasion or stricture following ulceration. Roberg found stones in Wharton's duct fifty times, in the submaxillary gland twenty-eight times, in Stenson's duct six times and in the parotid gland twice. Bendixen collected 216 cases (1908).

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Etiology.—Acute, simple angina may be caused by (*a*) rheumatism, cold or exposure; (*b*) infections, either acute (measles, scarlatina) or chronic (syphilis); (*c*) gout, dyspepsia or smoking; (*d*) in many cases the cause is obscure, probably always mycotic; no single virus is found. It is most common in youth.

Symptoms: (*a*) *Local:* The throat is red, glazed and streaked with mucopus which is easily wiped away. Swelling or vesicles on the soft palate and uvula, superficial erosions, and swollen mucous follicles are occasionally seen. In some cases the local findings, hidden in the nasopharynx, are very slight in proportion to the local pain and general symptoms. A scratching sensation in the throat may radiate into the posterior nares or ear. The patient hawks, but raises little, unless there is coincident rhinitis or laryngitis. Swallowing is rather painful, slight deafness is common, the speech is slightly nasal and the angular lymph nodes are painful. (*b*) *Constitutional symptoms* are usually slight. At the onset there are chilliness, moderate fever, aching in the muscles and in some individuals extreme cutaneous hyperesthesia, which may be falsely attributed to changes in the deeper parts of the body. With or without coincident tonsillitis, there may be high fever, and in labile individuals, marked nervous intoxication. Herpes facialis occasionally develops. Complications, as acute endocarditis or nephritis, are very uncommon. In a few days convalescence is complete.

Treatment.—(*a*) *Local:* Painting the pharynx with a 20 per cent. silver nitrate solution affords local and general relief. (*b*) *General:* Dover's powder and acetylsalicylic acid, each ten grains, may be exhibited for unusual pain, calomel, followed by salines, for elimination and a 1 per cent. phenol gargle for the throat.

The *pneumococcic sore throat* may be suppurative, pseudodiphtheritic, follicular, like tonsillitis, erythematous or herpetiform; its onset is severe, it produces a thicker and more adherent membrane than does the streptococcus and the prognosis is generally poor.

CHRONIC PHARYNGITIS.

Etiology.—(*a*) Recurrent pharyngitis; (*b*) tobacco or alcohol; (*c*) abuse of the voice, as in clergymen, venders, etc.; (*d*) extension from chronic nasopharyngitis; (*e*) chronic nephritis, syphilis, etc. These factors are most common in adolescence and middle life.

Symptoms.—The nasopharynx is red and lined with turgid venules; the pillars are relaxed and the drooping uvula may tickle the tongue or epiglottis, causing chronic coughing. The sides of the pharynx may show isolated whitish epithelial thickenings or more often, reddish, hypertrophied lymph follicles (*pharyngitis granulosa*). The pharynx is dry and burning, exciting hawking efforts which are unproductive and may

cause morning vomiting. Slight bleeding occasionally causes unnecessary worry. Middle-ear involvement is common, through the Eustachian tube. *Pharyngitis sicca* is an atrophic type and marked by a pallid glazing of the pharynx wall and crust formation.

Treatment.—(*a*) The *causal factors*, as straining of the voice, alcoholism or smoking, etc., are corrected. Alkaline laxatives are frequently beneficial. (*b*) *Galvanopuncture* of the turgid veins or hyperplastic follicles is indicated; improvement is probably more frequent than aggravation by overzealous treatment. (*c*) *Local medical treatment* is less efficacious.

℞—Phenolis . . . . . gr. xx
Acidi tannici . . . . . ʒss
Glycerini . . . . . ʒij
Aquæ rosa . . . . . q. s. ad. ʒv
M. et ft. gargarismus.
S.—As gargle, properly diluted.

℞—Tincturæ guaiaci . . . . . ʒiv
Liq. potassæ . . . . . ʒss
Alcoholis . . . . . ʒj
Aquæ cinnamomi . . . . . q. s. ad. ʒiv
M. et S.—One teaspoonful in one-half glass of water as gargle.

(*d*) For *atrophic* pharyngitis the best formula is:

℞—Iodi . . . . . gr. iij
Potassii iodidi . . . . . ʒss
Glycerini . . . . . ʒj
M. et S.—Massage thoroughly into the pharynx.

PHLEGMONOUS PHARYNGITIS.

Angina phlegmonosa invades the submucosa (Senator). It is usually primary but may be metastatic. The throat is painful, swollen and edematous, the neck enlarges and suppuration occurs with high fever, sepsis, dysphagia and dyspnea, especially when the subject lies down. The inflammation may be hemorrhagic or necrotic. Phlegmonous angina is the analogue of suppurative submucous laryngitis and angina Ludovici. Without surgical intervention or early spontaneous rupture into the throat, death occurs from septicopyemia, mediastinitis or glottis edema.

RETROPHARYNGEAL ABSCESS.

(*a*) The *primary* form occurs in children under two years of age, with dysphagia, a nasal voice and retropharyngeal suppuration, as a visible protrusion or a palpable fluctuation on the posterior pharyngeal wall; dyspnea, cyanosis and inspiratory retraction of the chest develop. It is primarily a lymphadenitis. (*b*) Secondary types are less common in caries of the cervical spine, diphtheria, erysipelas, scarlatina and suppurative inflammation in the retropharyngeal lymph glands, or rarely by metastasis. Unless surgical measures are instituted or spontaneous rupture occurs early, death results from asphyxia, mediastinitis, glottis stenosis, inhalation pneumonia or sepsis. After incision the head should be promptly thrown forward to obviate occlusion of the air passages.

PHARYNGEAL ULCERATION.

Syphilitic and tuberculous ulceration have been considered. Follicular ulcers are superficial and, like all ulcers in the mucous membrane only, heal without a cicatrix. Ulceration also occurs in diphtheria and the diphtheroid group, due chiefly to the streptococcus. Malignancy too, causes ulceration.

Vincent's angina (see page 85) constitutes 2 per cent. of all anginas including diphtheria. It occurs especially in children between six and ten years, medical students and servants in anatomical laboratories. Tobacco, tuberculosis, syphilis, scarlatina, measles and the eruption of wisdom teeth are predisposing factors. The two causative organisms were first described by Rauchfus (1893): (*a*) The *fusiform bacillus* has pointed, sometimes rounded ends, is sometimes bent, measures 6 to 12μ in length and may be flagellated; (*b*) the *spirillum* or Spirochete darticola is long, delicate and twisted. They stain with carbol-fuchsin but not by Gram's method.

Symptoms.—In the first period, there is *congestion* and edema. Then a grayish-yellow *membrane* forms which is friable, cheesy and usually removable. Under the membrane, an *ulcer* develops; it is usually single, oval and has an irregular edge and an uneven granulating floor.

The general symptoms are lassitude, indigestion, vomiting or epistaxis; fever over 100° is uncommon and complications rare (albuminuria, enteritis or noma). The breath is fetid and the cervical nodes enlarge. Lesions are recorded in the larynx and bronchi alone. Syphilis and diphtheria require differentiation. It is communicable within narrow limits. The affection heals under applications of iodine, pencilling with silver nitrate and gargles of hydrogen peroxide or chlorate of potash. Salvarsan locally has given rapid results. Mercury is contra-indicated (*v.* page 226).

DISEASES OF TONSILS.

ACUTE FOLLICULAR OR LACUNAR TONSILLITIS.

Definition.—An acute mycotic parenchymatous inflammation of the tonsils, usually attended by decided constitutional reaction.

Etiology.—(*a*) *Bacteriologically,* the streptococcus is the most frequent cause, then the staphylococcus, pneumococcus and diphtheria bacillus (at the time of diphtheria epidemics, but such cases are a subtype of diphtheria). (*b*) The virus of *rheumatism* probably gains access to the blood by way of the tonsils; affections associated with rheumatism, as erythema nodosum, chorea and endocarditis may follow acute tonsillitis. Tonsillitis may begin the rheumatic cycle, as described under Rheumatism in Children, or it may appear later with endocarditis, pericarditis, arthritis and chorea. Tonsillitis is contagious. (*c*) *Cold and dampness* are directly predisposing causes, largely in the spring. (*d*) Tonsillitis occurs

chiefly in youth and adolescence. (*e*) *Individual predisposition* depends on some unknown factor and one attack favors recurrence. (*f*) *Poor hygiene* and possibly sexual activity, especially in young married people, may be conducive to tonsillitis (Shepard).

Symptoms.—(*a*) The *onset* occurs with chilliness or rigor, severe *pains in the back* and limbs, *fever*, rising within a day to 103° or even 106° in children or susceptible adults, *pain in the throat*, particularly on swallowing, and tenderness of the glands at the angle of the jaw. (*b*) The *tonsils are swollen* and show on the deeply injected surface three to ten yellowish-white points, corresponding to the lacunæ; these crypts contain bacteria, epithelium and leukocytes and are small abscesses; the tonsil is the seat of serocellular infiltration. Occasionally there are small superficial areas of necrosis, slight erosions or even membrane. The voice may acquire a nasal twang, the pain increases for a few days and the angular glands remain painful and tender, whence the head is often held rigidly forward and the jaws are opened with difficulty. Pain in the ear is common. (*c*) The *constitutional symptoms* (*v. s.*) also include anorexia, coated tongue, hyperesthetic skin, depression, accelerated pulse and respiration; in two adult cases seen by the writer, retraction of the neck with headache, delirium and vomiting was mistaken for meningitis. Herpes facialis is frequent.

"Septic Sore Throat."—English writers have long believed that milk conveys streptococcic tonsillitis; such epidemics were observed in Christiana in 1908 and, in 1911–12, 1043 cases developed in Boston with 5 per cent. mortality and 10,000 cases in Chicago. In 75–87 per cent. of cases, infection was traced to certain dairies, where bovine mastitis and human sore throat prevailed. The disease is due to streptococci (of a type halfway between the S. pyogenes and S. mucosus) or to pneumococci. The local findings are tonsillitis with a milky gray membrane; the onset is severe, or first mild with later severe symptoms—fever 102–106°, bradycardia (40–90), prostration, chill, moderate leukocytosis; later marked lymphadenitis, which suppurates in about 10 per cent., and suppurative tonsillitis in 5 per cent.; and finally septic generalization in the severerst types, with peritonitis particularly, empyema, meningitis, arthritis and endocarditis.

Complications.—Complications are uncommon. Acute pericarditis, endocarditis, functional heart murmurs, febrile albuminuria, otitis and erythema (from the toxins or medication) are not very infrequent. Convalescence is complete within a week, though marked prostration and considerable residual swelling remain for some time. Repeated attacks may cause chronic nephritis or endocarditis.

Diagnosis.—The typical case cannot be confused. (*a*) *Diphtheria* produces a white membrane, with a pathognomonic tendency to spread to the uvula, soft palate or pharynx; it leaves bleeding raw points when it is stripped off; it recurs after removal; it sometimes extends to the larynx; it is followed not infrequently by paralysis, responds to serotherapy and shows the Klebs-Loeffler bacillus. (*b*) *Tonsillitis necrotica* leaves an ulcer; some cases showing the fusiform bacillus and spirillum of Vincent and others are alleged to be diphtheritic; in some cases there

is much fever, in others none; the general reaction is severe. (*c*) The possibility of incipient *scarlatina* should be kept in mind.

Treatment.—1. Prevention.—The subject should be hardened by cool baths or sponges, and tonsillectomy should be performed.

2. Local Measures.—After cocainizing the throat, a small probe, bent at right angles near its end, is introduced into each swollen crypt to secure free drainage; then undiluted Dobell's solution is applied on bits of cotton to each crypt, followed by 20 per cent. *silver nitrate* solution; the results are often immediate, sometimes abortive. Squeezing out the crypts with a spatula is painful. Hot fomentations may be applied to the neck.

3. Constitutional Treatment.—In a small number of cases sodium salicylate, given as in rheumatism, affords relief. Hydrarg. biniodide, gr. $\frac{1}{500}$, every fifteen minutes for ten doses may abort the process; tr. guaiaci ammoniat., ♏x–xl, every two hours is unsatisfactory. Ac. acetylsalicylicum and Dover's powder, āā gr. x, relieve pain in the throat, body and limbs. Aconite and belladonna are beneficial in children with high fever, but less so in adults; they are given as in coryza or influenza.

SUPPURATIVE TONSILLITIS.

Etiology.—The etiology is almost identical with that of the follicular type. Suppurative tonsillitis is most common in adolescence, and for unknown reasons individual disposition is an important factor.

Symptoms.—The symptoms resemble those of the follicular type but are more severe from the onset; one, or less often both, tonsils are greatly swollen, tense, edematous, and deep red. The pain often radiates to the ears. The swollen tonsils may come in contact; one swollen tonsil may reach over to the sound side. There is often coincident stomatitis, a free salivary flow, and edematous palate and uvula. Swallowing causes great pain, the jaw cannot be depressed because of the swollen, painful angular glands, and the speech is nasal. The toxemic prostration is often profound, the fever ranges between 103 and 105 degrees and the pulse between 100 and 140.

Incision in the first two or three days evacuates only blood and serum. In a few days fluctuation is felt anterior to and above the tonsil (peritonsillar abscess). Introduction of a probe into the early incision is followed by escape of pus and immediate relief. The abscess may break spontaneously, usually forward and rarely toward the throat, when, in rare cases, suffocation or aspiration pneumonia may follow. Very rarely ulceration may reach the carotid with fatal hemorrhage. Minute suppurative foci may cause septicemia.

Treatment.—(*a*) *Tonsillectomy* is indicated after an individual tendency is manifested. (*b*) An ice-bag over the neck is beneficial. Local applications are uncertain. It is good practise, early in the course, to introduce a knife, *with extreme care,* covered with adhesive plaster to within half an inch of its point, above and inside the tonsil; blood and serum escape, tension, edema and pain are relieved and the later abscess more readily points toward the cut; the wound is opened with a blunt probe every

day. (*c*) *Salicylates* help, and possibly hasten maturation. *Opium* and *coal-tar products* relieve the pain to some extent. (*d*) In exceptional cases intubation or tracheotomy is indicated, because of great swelling. (*e*) The diet is necessarily liquid. (*f*) In *convalescence*, iron and strychnine overcome the resulting prostration and anemia.

CHRONIC TONSILLITIS.

Synonyms.—Chronic inflammation; tonsillar hypertrophy; hyperplasia of the pharyngeal or lingual tonsils; adenoids; aprosexia.

Etiology.—(*a*) Repeated *acute attacks* of tonsillitis cause chronic hypertrophy in some cases, and in others the tonsils gradually indurate and shrink. (*b*) Waldeyer's "Schlundring" of *lymphadenoid* tissue consists of the *two tonsils*, the adenoid tissue in the nasopharyngeal vault ("*adenoids*" in the common acceptation) and the *lingual tonsil*. Variously combined hypertrophies of these structures may be noted, occasionally as congenital growths, but most frequently from the third year until puberty. Adenoids are found in 1 per cent. of children. Rickets, the lymphatic constitution and acute infections involving the throat predispose.

Symptoms.—1. Local Changes.—(*a*) From the *enlarged tonsil* may be expressed cheesy plugs, which occasion an extreme *fœtor ex ore;* calculi may result from a deposit of lime salts. The pharyngeal mucus is increased, sometimes tinged with blood and expectorated with difficulty. (*b*) *Adenoids* occur alone or with tonsillar hyperplasia. They are papillomatous, vascular and range up to the size of a bean. (*c*) Hypertrophy of the "*lingual tonsil*" often disturbs deglutition. Enlarged tonsils and adenoids produce the following changes by stenosing the posterior nares:

2. Sequences.—(*a*) *Oral breathing* is the earliest symptom and is first noticed at night, when sleep is disturbed by night terrors (*pavor nocturnus*), by disordered and irregular breathing, for a time stertorous and then interrupted, asthmatic seizures or Balne's paroxysmal cough. (*b*) The *palatal vault* is *high* from atmospheric pressure in the mouth, and the transverse measurement between the upper teeth is diminished; there is less room for the teeth to erupt. (*c*) The *anterior nares are small* and retracted and the voice is nasal (*rhinolalia claustra*); the consonants *n* and *m* are pronounced with difficulty. Bloch associated oral breathing with stuttering. The sense of smell or taste is obtunded. Hearing is impaired by pressure of adenoids on the Eustachian tubes, or retraction of the tympana from low atmospheric pressure in the nasopharynx. (*d*) The *facies* is characteristic; it is *vacuous* and *apathetic*, the nose is pinched, the mouth open and the lips swollen. The "adenoid habitus" may be observed as a stigma of degeneracy. (*e*) In chronic cases the cerebral reaction is slow, the memory tardy, the disposition sullen and the power of concentration diminished (aprosexia), due perhaps to some cytotoxin elaborated in the lymphoid tissue. (*f*) *Nervous symptoms* embrace headache, habit-spasm, night terrors and enuresis nocturna. (*g*) *Development* is sometimes retarded. (*h*) *Deformity of the thorax* was noted

PLATE XIII

FIG. 1

FIG. 2

Types of the "Adenoid" Facies. (Posey and Wright.)

by Dupuytren (1828). The pigeon- or *chicken-breast* is the most common. The sternum juts forward, from which the ribs slant sharply backward; at the level of the diaphragm there is a circular depression, and inspiratory retraction is caused by the contraction of the diaphragm. The *funnel-breast,* marked by a deep depression of the lower part of the sternum, is less commonly due to adenoids, and like the chicken-breast also results from other causes. The emphysema chest may result from adenoids. (*i*) Mouth-breathers may develop collapse-induration resembling apical tuberculosis (see page 158). (*j*) The cervical lymph glands are enlarged and the blood evidences decreased hemoglobin, increased white cells and distinct lymphocytosis. (Plate XIII.)

Diagnosis.—The oral breathing, adenoid facies, mental disturbance, palpation of the adenoids in young children and their rhinoscopic detection in older ones, and the inspection of hyperplastic tonsils determine the diagnosis. In the *prognosis* of untreated cases, the possibility of deafness, mental deficiency, retarded growth, arthritis, endocarditis, nephritis, asthma and emphysema should be borne in mind. Acute recurrent tonsillitis, diphtheria and other infections are more likely to develop and produce disproportionate symptoms. Rarely the hyperplastic tissues may atrophy spontaneously.

Treatment.—The general health should be maintained by out-door life, an ample diet, hydrotherapy and iron. Hypertrophied tonsils should be removed when they harbor chronic infection, or when growth is retarded, etc. Adenoids causing symptoms should be removed early under ether anesthesia; chloroform frequently causes death in the *status thymicus* or lymphoid *constitution.* Oral breathing, persisting after operation, is a habit hard to break; at night a bandage should be used to close the mouth.

DISEASES OF THE ESOPHAGUS.

INFLAMMATION, NECROSIS, ULCERATION.

I. Esophagitis.—This is an uncommon finding at autopsy. Its *causes* are (*a*) mechanical, as from foreign bodies, strictures or dilatation of the esophagus; (*b*) chemical, as corrosive poisons or acid vomitus; (*c*) hypostatic, in cardiac or pulmonary affections; (*d*) rarely extension from pharyngeal or laryngeal inflammation; (*e*) acute infections, as smallpox and diphtheria. Esophagitis is physiological in the newborn after the ingestion of the first meals of life. Its pathology is local or diffuse, acute or chronic redness, swelling and hypersecretion. Special forms are follicular catarrh, sometimes resulting in ulcers or retention cysts; exfoliative esophagitis; the fibrinous form, observed in acute infections and uremia; genuine diphtheria; the pustules of smallpox; the toxic form from caustics or acids; only 12 cases are on record of the single bulging submucous abscess or diffuse submucous suppuration which perforates into the esophagus which then presents a sieve-like aspect;

forms due to the Oïdium albicans or ray fungus. In chronic cases the epithelium may thicken (leukoplakia) or hypertrophy (papilloma).

Symptoms.—Only in marked cases is there *pain*, continuous, substernal or elicited by speaking, or movement of the spine. *Dysphagia* is constant in marked inflammations. Mucus, blood, pus or eschars, depending on the nature of the inflammation, are brought up by gagging efforts. Fever is common in the more severe types, in which the local symptoms are wholly obscured by the causal diphtheria, smallpox, etc. In inflammation due to acids or alkalies, the history, local symptoms and eschars in the mouth and pharynx are distinctive.

Treatment.—Treatment is palliative. Narcotics are given for pain, demulcents as acacia and ice-cream and a liquid diet for local irritation, or nutrient enemata are given.

II. Necrosis.—This may be due to corrosive poisoning; pressure from struma, aneurysm, sounds left in the esophagus to dilate strictures; or "bed-sores of the gullet," described by Dittrich (1850) in laryngeal perichondritis, due to pressure of the larynx upon the gullet. *Esophagomalacia* is softening due to regurgitation of the gastric acid, chiefly after death, or during the death agony.

III. Ulceration.—Aside from cancer, follicular ulceration, caustic ulcers, syphilis and tuberculosis, there is the *peptic ulcer* first described by Cruveilhier, who identified it with ulcer of the stomach. Forty cases were collected by Tileston (1908). It appears in four conditions (*a*) by extension from the round ulcer of the cardiac orifice, (*b*) in ulcers at the pylorus or duodenum, which produce gastric dilatation, (*c*) in multiple hemorrhagic infiltrations of the esophagus, stomach and duodenum and (*d*) in alcoholism and arteriosclerosis. Its chief symptom is *dysphagia* (50 per cent.); its pathology, complications and treatment are those of gastric ulcer.

STENOSIS OR STRICTURE OF THE ESOPHAGUS.

Stenosis is the most frequent esophageal disease. The esophagus is a closed tube only in its cervical segment, and its cardiac orifice is not physiologically closed.

Etiology.—(*a*) *Interstitial factors, i. e.*, in the wall of the esophagus, are the most important; *cancer* causes 90 per cent. of all stenoses; *ulceration* by caustics and acids comes next; then stenosis by diverticula or pouches which, bulging out of and beside the esophagus, exert upon it a lateral compression; rarer ulcerative stenoses result from healing gummata, tuberculous lesions, smallpox and peptic and diphtheritic ulcers; muscular spasm causes many cases; congenital stenosis or atresia, of which Dickie (1906) collected 76 cases, occurs chiefly as a *cul de sac*, incomplete development or fistulous communications with the trachea or bronchi. (*b*) *Extra-esophageal* factors, compressing the tube from without, include *aortic aneurysm* or abnormal branches of the aorta; vertebral tumor, cold abscess, exostosis or deformation; anthracotic lymphadenitis and malignant lymphoma; thyroid or thymic enlargements; medias-

tinitis, pericarditis and pulmonary neoplasms. (*c*) *Intra-esophageal* factors are the rarest, as foreign bodies, pedunculated polyps, etc.

Stenosis occurs chiefly (*a*) at the lower third of the esophagus, (*b*) near the level of the bifurcation of the trachea or (*c*) high in the esophagus. The stenosis is more often single than multiple and more often annular than longitudinal; it varies from slight stenosis to complete atresia. Above the stenosis the tube is dilated and its musculature is hypertrophied to force food through.

Symptoms.—*Dysphagia* may be secondary to aortic aneurysm and vary from time to time with the pressure exerted upon it; in cicatrizing ulcers dysphagia is the only symptom. In advanced stenosis the food and later the fluids *regurgitate* after a short time if the stenosis is low, or at once if the stenosis lies just below the pharynx. The *regurgitated food is neutral*, often fermented and contains round cells, mucus or blood, but never HCl nor pepsin. Esophageal *pain* is common. *Singultus* is frequent when the lesion lies near the diaphragm. Dyspnea may result from pressure upon the air passages by the dilated portion of the esophagus above the stricture.

Signs and Diagnosis.—The chest should be examined for aneurysm, retrosternal struma and lung tumor and the finger should be introduced down the throat to exclude lesions situated high in the esophagus or larynx. *Esophagoscopy* may give excellent results if done by an expert. Local signs are elicited in three ways:

1. Sounding.—A soft sound is far less dangerous at least for the first examination in cases of acute esophagitis, aneurysmal compression and ulcerating cancer; as there may be other strictures lower down, the sound should be passed into the stomach. The length of the esophagus is 10 inches or 25 cm.; from the upper incisor teeth to the esophagus, 6 inches or 15 cm.; from the teeth to the cardia, 16 inches or 40 cm. (19.7 inches or 50 cm.). The length of the cervical portion is 2 inches or 5 cm., of the thoracic, $6\frac{3}{4}$ inches or 17 cm., and of the abdominal part, $1\frac{1}{4}$ inches or 3 cm. From the teeth to the level of the tracheal bifurcation is 9 inches or 23 cm.

2. Auscultation.—Physiologically, two murmurs are heard with the stethoscope six to eight seconds after the beginning of deglutition, first a short and then a longer murmur. In marked stenosis the swallowing sound is heard at a point just above the lesion, of value when aneurysm is suspected and sounding is feared. In the cervical part the noise results from the movements of the pharynx; in the thoracic segment, to the left of the spine from the first to ninth dorsal vertebra, there is a delayed first murmur, and a gurgling, spurting character in both murmurs; in the lowest segment the murmurs are heard above the cardia, at the tenth dorsal vertebra.

3. X-rays.—The *x*-rays outline the dilatation (*v. i.*).

Prognosis.—Cancerous stricture may ulcerate, relieving the local symptoms, though cachexia increases. Strictures from caustics often improve. Where the cause cannot be relieved, the same symptoms result as in esophageal cancer (*q. v.*). Perforation into the lungs, pleuræ, air passages,

mediastinum or vessels may follow. Aspiration pneumonia, which often becomes gangrenous, is not infrequent.

Treatment.—The remediable etiological factors are few. Foreign bodies can sometimes be removed by long forceps, with the direction of the esophagoscope. In cancerous and aneurysmal strictures, dilatation by sounds is attended by the risk of perforating the esophagus. Sounds should be introduced beside the index finger, with the patient's head thrown slightly backward; severe gagging may be overcome by painting the pharynx with cocaine, though it is often as much psychical as esophageal. The subject should first swallow some olive oil to facilitate passage of the sound, which is held like a pen and pushed with exceeding gentleness. Trousseau's ivory olive points are excellent, but care is necessary, and successively larger sizes should be used. Sounds may be left in the esophagus for some minutes or an hour, or even permanently, but pressure necrosis may then result. It is at times necessary to introduce filiform bougies. Silver balls (2 to 7 mm. in diameter) with threads attached, may be swallowed and carefully pulled out after remaining in over night. When the obstruction is high, external esophagotomy is advisable. After opening the stomach, dilatation may be employed from below. Impermeable strictures call for gastrostomy.

DILATATION. DIVERTICULUM.

Etiology.— *Dilatation involves the entire circumference; diverticulum involves but part of it.* Secondary *dilatation* may occur above stenosis of the esophagus or above the cardiac end of the stomach. It seldom extends more than an inch and a half (4 cm.) above the stricture; it may follow enteroptosis, ulcer or pyloric stenosis. Marked dilatation above the cardia, Luschka's so-called "fore-stomach" is a local form, usually congenital. The *diffuse idiopathic dilatation* results from *cardiospasm* (Purton, 1821). It is seen chiefly between the years of twenty and forty. Most clinicians incline to Meltzer's view, that *cardiospasm* is due to abolition of cerebral inhibition on the cardia; Kraus reports a case with great atrophy of both vagi; others accuse a primary esophagitis or the angular insertion of the esophagus in the stomach. Cardiospasm produces a great hypertrophy of the cardia muscle, even six times its normal thickness, and diffuse total dilatation so that the esophagus may measure 46 cm. instead of 25 cm. in length, or 30 cm. in circumference. Though Zenker collected but 18 cases in 1878, in 1912 Plummer published 91 personal cases. The mucosa is catarrhal, thickened or eroded, and the muscularis is hypertrophied.

Symptoms.—There is gradual or sudden *dysphagia,* which may be overcome by effort; the patient feels that the food does not reach the stomach. *Regurgitation* of the food, especially the portion last swallowed, occurs soon after eating or lying down; it may measure two quarts, is neutral, decomposed if retained for some time, and contains no hydrochloric acid, pepsin or rennet. The regurgitated food may again be swallowed. Vomiting is impossible. The pressure of the stagnant food or fluid in the gullet may produce dyspnea or palpitation. The *sound* moves freely

from side to side, but meets temporary resistance at the cardia, though pressure usually overcomes an obstructing cardiospasm. One sound, introduced into the stomach, may recover HCl, pepsin, etc., but another, introduced *to* but not *beyond* the cardia, recovers fluid or food with a neutral reaction; methylene blue solution poured through the first tube returns blue; that recovered by the second tube is colorless. Distention of the esophagus with water gives *dulness*, to the left of the sixth to the ninth dorsal vertebra, sometimes above the left costal arch, or even in the epigastrium. The esophagoscope reveals the dilatation; a distinct shadow is seen by using the *x*-rays after the patient has eaten potatoes mixed with bismuth. The *second swallowing bruit* may be absent or sounds like fluid running into fluid. Marasmus, constipation and often death result, but the clinical course may cover decades.

Treatment.—Treatment is unsatisfactory. Olive oil ℥ss before meals may help the fluid (or the sound) to pass the cardia. Continued use of the stomach-tube may cause esophagitis; its life-long use is often required. Lockwood's pneumatic bag may be introduced empty on a sound into the stomach, then inflated and withdrawn gently through the cardia. Gastrostomy and rectal feeding may be indicated. Operation should not be delayed too long—entering the stomach and divulsing the cardia.

Diverticulum involves only an arc of the esophageal circumference. Two types are distinguished: 1. *Zenker's pressure diverticulum* (pulsion-diverticulum) of which Rosenthal (1902) collected 180 cases. It is found chiefly in men (77 per cent.), and is a pouch which forms on the posterior esophageal wall, near its junction with the pharynx, and consists only of the mucosa and submucosa—a bottle-shaped esophageal hernia. Its onset is very gradual, for the muscle, probably congenitally weak, yields before intra-esophageal pressure, trauma, foreign bodies and the stagnating food. The early *symptoms* are signs of pharyngo-esophagitis, hawking and dysphagia, and food entering the sac rather than the esophagus; the axis of the sac, which dips between the gullet and the spine, is roughly parallel with that of the esophagus; many of its symptoms are those of a high esophageal stenosis. The food stagnates and regurgitates with a fetid odor. Cooper describes a succussion splash. In 33 per cent. of cases a tumor appears in the supraclavicular region, which is dull when filled with food or tympanitic when distended with carbon dioxide; it sometimes can be smoothed out, whereon the food regurgitates. The murmurs on swallowing are protracted in 50 per cent. A sound usually enters the sac, and another, if slightly bent, enters the gullet, which differentiates a diverticulum lower in the gullet, which is even less common. Examination by the esophagoscope is generally negative, but the *x*-rays show a characteristic shadow. There may be pressure on the heart, trachea, vagus, recurrent laryngeal nerve and brachial plexus. The clinical course is protracted, even to forty-nine years, and the evolution is inanition in over 50 per cent., gangrene (15 per cent.) or perforation with cervical phlegmon. *Treatment* includes feeding by means of the tube, rectal nutrition, gastrostomy and surgical resection, first performed by von Bergmann, with 20 per cent. mortality.

2. *Rokitansky's traction-diverticulum,* found in 3.5 per cent. of autopsies, is due to extra-esophageal traction, which draws part of the gullet out into a funnel-like form. Over 80 per cent. are due to indurative periadenitis around caseous or anthracotic lymph glands. Cicatricial mediastinitis from pleuritis, pericarditis or vertebral disease produces the same effect. It usually, therefore, occurs at the level of the tracheal bifurcation; it begins early in life, is usually single, less marked on the anterior or lateral wall, rarely exceeds 5 to 10 mm. in depth and is rarely discovered except with the esophagoscope. Its chief danger is ulceration at its apex and perforation, with resulting gangrene of the lungs, fatal hemorrhage, pneumothorax or phlegmonous mediastinitis.

CANCER OF THE ESOPHAGUS.

Etiology.—Its frequency is rated at 0.25 to 1 per cent. of autopsies, ranking third among cancers (stomach 33, uterus 15, and esophagus 6 per cent.). Scars, ulceration and traction are promoting causes; 75 per cent. occur in males and 80 per cent. between the fortieth and sixtieth year and 8 per cent. occur before the fortieth year.

Pathology.—Cancer of the esophagus is single and primary; cancer of the stomach, pharynx or mediastinum rarely involves the esophagus secondarily. Most cases are squamous *epithelioma,* arising from its pavement-epithelium; scirrhous, colloid and medullary forms are rare. Most cases (57 per cent.) develop in the lowest third of the esophagus, 33 per cent. in the middle, and 10 per cent. in the upper third; its favorite sites are near the cardia, tracheal bifurcation level and back of the cricoid cartilage. The muscularis sometimes hypertrophies above the cancer, where some dilatation may occur.

Symptoms.—Signs of *esophagus stenosis* (*v. s.*) develop in varying grades; stenosis generally advances slowly but occasionally is sudden. It may lessen as the cancer ulcerates. Its symptoms are those of stenosis, as *dysphagia,* rather indefinitely located pain, regurgitation of food and perhaps also of bloody mucus or necrotic cancer tissue. Its signs are also identical, as obstruction on sounding, fluoroscopic findings, detection by the esophagoscope of a papillomatous or ulcerated neoplasm, and rarely tumor particles found in the fenestræ of the tube. Salivation (Roger's syndrome) was noted in 22 cases. *Cachexia* develops; constipation, indicanuria and acetonuria are frequent. Extension, by contiguity, occurs to the spine, mediastinum and cardia, or in 60 per cent. by *metastasis;* the mediastinal lymph nodes are usually invaded, the supraclavicular often, and upper cervical seldom.

Complications.—There may be pressure on the recurrent laryngeal, vagus, mediastinal vessels, sympathetic ganglia or brachial plexus. *Perforation* occurs in over 50 per cent., most often into the air passages, when aspiration pneumonia or gangrene may follow; into the lung, pleura (ichorous pleurisy or pneumothorax resulting), aorta (fatal hemorrhage), heart, pericardium and mediastinum. Pyemia, tuberculosis and thrombosis occur in some cases. *Death* is usual within thirteen months, with emaciation, subnormal temperature, small, slow pulse,

slow, shallow breathing, flattened abdomen and perhaps acidosis, coma and fever.

Diagnosis.—The diagnosis is based (*a*) on a stricture of the esophagus, which in 90 per cent. of all cases is carcinomatous; (*b*) on cachexia, nodes, metastases and perhaps tumor particles on the sound. Other tumors, as lipoma, myxoma, myoma or fibroma are of little clinical importance, except accessible fibropapillomata of the upper gullet. Von Hacker (1908) collected 21 cases of sarcoma; 81 per cent. were in the lower thoracic segment.

Treatment.—The treatment is that of stricture, nutritive enemata, feeding by the stomach-tube, gastrostomy and narcotics. Less than 10 per cent. are accessible to surgical interference. As to gastrostomy, 45 per cent. do not live longer than a week and 90 per cent. not more than one hundred days.

PERFORATION, RUPTURE; HEMORRHAGE OF THE ESOPHAGUS.

1. *Perforation* may result from causes within or without the esophagus. Internal causes include ulcers, foreign bodies, diverticula, cancer and use of the sound; external causes are aneurysm, gravitating abscess, lung cavities or gangrene, suppurating lymph glands or trauma. It is marked clinically by mediastinitis and cervical cellulitis, which are usually phlegmonous. Esophageal fistulæ may communicate with the air passages, from which by the laryngeal mirror fluid or food is seen to be expectorated; a communicating tuberculous cavity may become flat when the patient drinks or eats, and the flatness changes to tympany when the food or drink is expectorated; this is tested by giving the patient milk, which will be brought up from the larynx. Aspiration gangrene, pneumopericardium, pneumothorax and fatal hemorrhage are common causes of death.

2. *Spontaneous rupture* (so-called) is a very rare affection, there being only 39 cases on record (Cohn, 1908). It is often confused with esophagomalacia, in which the rupture is not longitudinal or clean-cut as in genuine rupture. The first case, and one of the clearest, was reported by Boerhaave (1714); in all cases, except Boerhaave's, the rupture was longitudinal. It follows overeating or severe vomiting, and most victims have been alcoholics. There is great and sudden pain over the lower esophagus (where most ruptures occur) and over the xiphoid, hematemesis, collapse and subcutaneous emphysema. Death results from collapse, phlegmonous mediastinitis, pleuritis or pneumothorax.

3. *Hemorrhage* may follow esophageal ulceration, external or internal trauma, toxic esophagitis, softening and aneurysmal or other perforations. Varicose veins in the lower esophagus, first described by Le Derberden (1837), most often complicate liver cirrhosis, less often syphilis and pylethrombosis. The diagnosis and localization of the bleeding-point is only suggested by the history and previous physical findings. *Treatment* consists chiefly of absolute quiet, morphine and rectal feeding.

MOTOR AND SENSORY DISTURBANCES OF THE ESOPHAGUS.

(See AFFECTIONS OF THE VAGUS under NERVOUS DISEASES.)

DISEASES OF THE STOMACH.

ACUTE GASTRITIS (ACUTE CATARRH, ACUTE DYSPEPSIA).

I. Simple Gastritis.—Etiology.—It is a common primary and secondary affection. (*a*) Its most important *mechanical* cause is improper food or improper eating; *rich food,* an excessive amount, *partial mastication,* washing down food with water, hurried eating when very hungry, articles containing bacteria or ptomaines and excessive ingestion of cold water and fruit are accessory factors. (*b*) *Thermal* factors include too hot or too cold foods, especially when taken on an empty stomach. (*c*) *Chemical* causes; aside from toxic gastritis (*v. i.*), *alcohol* ranks foremost; autotoxins may excite acute gastritis, as in nephritis, cholemia or gout. (*d*) Some cases are clearly *infectious;* the condition may occur in variola, typhoid, grippe and pneumonia, as a secondary manifestation; occasionally it is an independent febrile affection, as in the Christiana epidemic reported by Hausemann (1880), in which 6000 persons were affected within three weeks (Gärtner's Bacillus enteritidis, paratyphoid bacillus or Bacillus botulinus). (*e*) Stasis from diseases of the heart, lung or liver, tuberculosis, cancer, chlorosis and affections of the higher alimentary mucosa are predisposing factors. In some cases a nervous, familial or even hereditary tendency is noted, "the delicate stomach."

Symptoms.—1. Digestive Symptoms.—These are sometimes absent, as demonstrated by Beaumont in the celebrated St. Martin case of gastric fistula. The *appetite* is usually *lost* or perverted. The *tongue is thickly coated* with white fur and dry mucus. The *mouth is dry* and there is a disagreeable taste and *thirst,* even though the salivary flow be increased. Hiccough, *eructations,* a sense of gastric distention, pressure or *pain* and tenderness over the stomach are common. Nausea and *vomiting* characterize the more marked cases; the vomitus is pale yellow, bitter and contains little or no hydrochloric acid, though lactic, butyric and fatty acids are common; there is much mucus, frequently flecked with blood. Gastric intolerance, pain, nausea and vomiting summarize the situation. Few uncomplicated cases come to autopsy; the mucosa is red, swollen, smeared with adherent mucus, sometimes slightly eroded or dotted with ecchymoses; the dilated vessels, seen by the gastroscope or through a gastric fistula, are less conspicuous at autopsy for the blood leaves the organ after death; microscopically the main findings are leukocyte emigration, granular degeneration and swelling of the mucous and other glandular cells.

2. General Symptoms.—Herpes is an occasional finding. *Constipation* is the rule. The *urine* is *scanty* because of the vomiting; it deposits a lateritious sediment and sometimes contains indican or acetone. Frontal *headache,* vertigo, intercostal neuralgia and depression are common; convulsions (acetonemia) and other marked nervous manifestations occur infrequently in sensitive individuals. Fever is uncommon; when present, it is irregular and ephemeral.

Diagnosis.—With fever or constitutional symptoms it is excellent practise to regard acute gastritis as a symptom, until typhoid, exanthematous disease or nephritis is excluded. Confusion is possible with a tabetic crisis, the vomiting of pregnancy, hysteria, appendicitis or gall-bladder disease.

Treatment.—(*a*) *Prophylaxis* embraces the etiological factors. (*b*) *Complete rest of the stomach* is the first indication; no rectal feeding is required for twenty-four to forty-eight hours, by which time the gastric equilibrium is restored; warm water by rectum unloads the bowels and allays thirst. (*c*) If *gastric irritation* persists, the stomach is washed out by the *stomach-tube* (using warm water with sodium bicarbonate); lavage is better than calomel, salines and apomorphine or ipecac which irritate the inflamed mucosa. *Epigastric fomentations* alleviate pain.

℞—Phenolis		gr. iv
Ac. hydrocyanici dil.		ʒss
Bismuthi subnitratis,		
Cerii oxalatis	āā	ʒiss
Mucilaginis acaciæ	q. s.	
Aquæ menthæ piperitæ	q. s. ad.	ʒij

M. et fac emulsum. S.—One teaspoonful without dilution every half to one hour for six to ten doses, or till vomiting ceases.

Opiates give relief, but secondary nausea is an insuperable obstacle to their use. (*d*) *In convalescence,* dilute hydrochloric acid ♏v–xv and bitters are given after meals, to stimulate the gastric secretion. The food should be simple and thoroughly chewed.

II. Gastritis Toxica (Venenata).—Etiology and Pathology.—Acute toxic gastritis is caused by various alkalies, acids and metallic poisons. Sulphuric acid produces a gray eschar, nitric acid a yellow one, alkalies a brown, copper a greenish-blue, silver a deep black eschar and phosphorus produces an opaque, milky stain. The pathological changes are severest in places with which the corrosives remain longest in contact, as the pylorus and posterior wall; they range from epithelial desquamation and catarrh, to hemorrhagic infiltration, diphtheroid patches, suppuration and ulceration.

Symptoms.—They are those of simple gastritis, but more severe; pain is intense in the mouth, throat, esophagus and stomach, and the vomitus is often bloody and the epigastrium tender.

Prognosis.—The outcome varies; (*a*) collapse after a few hours or days is frequent, with small pulse, shallow breathing, subnormal temperature, bloody, albuminous urine, etc. (*b*) The gastric symptoms subside and death occurs later from blood or visceral changes. (*c*) Stricture may develop, or (*d*) there may be atrophy of the secretory glands.

III. Phlegmonous Gastritis.—Etiology.—Only 131 cases are reported since Borel's in 1656. It is an acute suppurative gastritis, usually caused by the streptococcus, which usually enters some small erosion at the cardia and infiltrates the submucosa and other coats diffusely or circumscribes as a mural abscess. It occurs secondarily (75 per cent.) in sepsis, acute infections, or in local diseases as gastric cancer; 85 per cent. occur in adult males. Alcoholism and traumatism are predisposing factors.

Symptoms.—Besides an acute *gastritis*, the vomitus sometimes contains pus, the epigastric pain is intense, with meteorism and many symptoms of peritonitis, which is a most common complication. In a few cases an *epigastric tumor* marks the circumscribed variety. *Sepsis* causes the high fever, the pronounced status typhosus, the small pulse, occasional icterus and petechiæ.

Diagnosis and Treatment.—A *diagnosis* is almost impossible, the *outcome* is fatal in 96 per cent. during the second week and the *treatment* is symptomatic, viz., rectal nutrition, stimulation, narcotics, and if the affection is suspected, operation. Some cases heal by rupture into the stomach with cicatrization.

IV. Diphtheritic Gastritis.—Genuine diphtheria is very rare. Diphtheroid gastritis, caused by streptococcic and other infections, may develop after gastritis toxica, pneumonia and kindred maladies.

V. Parasitic Gastritis.—This rare form is due to Oïdium albicans, favus, anthrax, larvæ of insects and tineæ.

CHRONIC GASTRITIS.

Chronic catarrh is characterized pathologically by degeneration of the parenchymatous and infiltration of the interstitial tissue, and clinically by mucus formation and decreased secretion.

Etiology.—(*a*) The factors are those of acute gastritis, from repeated attacks of which chronic inflammation may result. Abuse of drugs, especially cathartics, *alcoholism* and improper eating, *eating hurriedly* or at irregular hours, inadequate mastication, washing down the food with fluids, excessive use of coffee, tea, ice-water, pastries or fried foods are salient factors. Chewing and smoking tobacco, stomatitis, carious teeth and pyorrhea are important causes. Most cases occur in adult males. (*b*) It often accompanies other gastric diseases, as cancer or ectasia, or (*c*) tuberculosis, anemia, stasis, nephritis, gout, diabetes, syphilis, amyloidosis, etc.

The acid form is usually caused by excesses in eating, drinking and smoking; the subacid form by the abuse of spirits.

Pathology.—The pathological changes are observed chiefly in the *pyloric* region. The vessels are dilated, imparting a reddish-brown color, which later changes to gray. The mucosa is swollen, lax and covered with tenacious mucus or sometimes punctate hemorrhages or erosions. In a few cases the mucosa is rough, even papillomatous (*gastritis polyposa, état mamelloné*). The muscularis is occasionally hypertrophied near the pylorus. Histologically, the glandular cells show granular, fatty or mucoid degeneration; the parietal cannot be distinguished from the principal cells, the glands are often large or cystic and the tubules show ramifications; the glandular structures may completely atrophy (see Achylia). The interstitial tissues show leukocytic infiltration.

Symptoms.—The symptoms begin *insidiously*.

1. Oral Symptoms.—The *tongue* is generally *coated* with moist white fur, reddish at its tip and indented on its edges, which (like salivation)

may also result from alcoholic or nicotine stomatitis. There is frequently a bitter, sour, *salty taste* in the mouth and sometimes a marked *fœtor ex ore*. The throat frequently aches.

2. Gastric Symptoms.—(*a*) Gastric pain is less frequent than a sense of *distention* or *pressure after eating*, or sometimes when the stomach is empty; actual pain is characteristic of the alcoholic type. Soup and fluids, unless they are cold, generally cause no distress; *coarse or hard foods*, as beefsteak, bread, smoked meat, cabbage, hard-boiled eggs or cheese, cause distress. (In nervous dyspepsia, any kind of food may cause discomfort.) Tenderness is much less than in cancer or ulcer. (*b*) *Belching* indicates atony. Eructations of hydrochloric acid are rare. (*c*) Nausea and vomiting are infrequent, except in alcoholics, in whom the morning vomiting (*vomitus matutinus potatorum*) consists chiefly of saliva and mucus swallowed during the night.

3. Gastric Chemism.—Our knowledge dates from the introduction of the stomach-tube by Kussmaul and Leube and especially the work of Riegel, Ewald and Boas. After washing out the stomach the Ewald test breakfast is given, consisting of a piece of dry bread and a cup of weak tea without sugar or cream. In an hour the gastric contents is withdrawn (the patient compressing the abdomen and straining) by Ewald's aspiration bag. Innumerable *errors result from confidence in a single examination* and assigning undue importance to the gastric chemism alone. It is best to examine the contents without filtration.

(*a*) The *gross examination* shows coarse particles of food when the hydrochloric acid is absent or greatly reduced, or fine subdivision when it is present in fair amounts. A rancid odor denotes fatty acids, and a foaming appearance, fermentation. *Mucus* is seen in practically all cases; most clinicians do not recognize a simple gastritis without mucus formation, though Ewald distinguishes (i) simple gastritis in which the acid and ferments are decreased; (ii) a mucous gastritis, in which the acid is greatly decreased, the ferments are present and there is much mucus; and (iii) an atrophic gastritis (*v.* Achylia). Mucus usually indicates a reduction in the hydrochloric acid. Normally there is little gastric mucus, though rather more with an amylaceous than with a proteid diet. Mucus of gastric origin is intimately mixed with the food and is not yellow, nummular, pigmented or mixed with air or cells, as is swallowed mucus.

(*b*) The *HCl is reduced.* Of Günzburg's solution (2 gm. phloroglucin and 1 gm. vanillin dissolved in 30 c.c. of absolute alcohol), a few drops are added to an equal amount of gastric contents and heated, without boiling, in a porcelain dish, when a carmine-red color develops, indicating free hydrochloric acid; the solution should be kept in a dark bottle and not over two months, for it deteriorates. Boas's test gives the same reaction and is less expensive: resorcin 0.5, sugar 0.3, dilute alcohol q. s. ad. 10 gm.

Total free HCl. 10 c.c. of gastric contents plus a few drops of the indicator (0.5 per cent. solution of dimethylamidoazobenzol in strong alcohol) are placed in a beaker and, if free HCl is present, the yellow color imparted by the indicator will change to bright red; a decinormal solution of

caustic soda is then added drop by drop until the color of the indicator has been changed from red to bright yellow; the number of c.c. of decinormal solution used is multiplied by 0.00365 and again by 10 to give the amount of free HCl. Often the result is expressed in terms of the number of c.c. of decinormal solution used; *i. e.*, if 4 c.c. were employed, $4 \times 10 = 40$, the free HCl. The usual amount is 30 to 40.

The *total acidity* (HCl, lactic and other organic acids, acid phosphates, albumins, albumoses and peptones) is estimated by adding two drops of a 1 per cent. alcoholic solution of phenolphthalein (as an indicator) to 10 c.c. of the gastric contents; to this is added, drop by drop, a decinormal solution of caustic soda until the red color, due to the alkali acting upon the phenolphthalein, is no longer even deepened in hue by it. As 4 to 8 c.c. of the decinormal solution are usually required, 4 to 8×10 = normal total acidity; *i. e.*, 40 to 80 expressed in terms of the decinormal solution (corresponding to 0.15 to 0.25 per cent. HCl). In most cases determination of the total acidity is sufficient. The bread of the test breakfast (phosphates chiefly) accounts for a total acidity of from 5 to 8; this figure indicates that the gastric glands are atrophied.

In chronic gastritis the *hydrochloric acid* is *found decreased* or absent. A few cases, usually light or incipient, show a total acidity of 40 to 50; this form is called gastritis acida, though its separation as a type is scarcely justifiable.

(*c*) *Rennet and pepsin* are rarely absent, though usually *decreased;* 25 c.c. of gastric juice will dissolve 0.05 gm. of serum albumin in one hour, of fibrin in one and one-half hours and of egg albumen in three hours. A few drops of hydrochloric acid liberate the enzyme (pepsin) from the pro-enzyme (pepsinogen). If no rennet is present the filtrate will not coagulate milk.

Lactic acid is seldom found. Leukocytes and epithelial cells are found at times, and in rare cases shreds of gastric mucosa (gastritis exfoliativa, Parisier).

4. Other Symptoms.—(*a*) *Vertigo e stomacho læso*, gastric vertigo, is probably neurasthenic rather than gastric. Dyspeptic dyspnea probably results from pressure of the distended stomach on the heart. Headache, physical and mental depression, intercostal neuralgia, tender points between the shoulders or over the lower cervical spines and palpitation are common toxemic symptoms. (*b*) The bowels are *constipated;* diarrhea and tympany at times result from marked gastric fermentation or extension of the catarrh to the gut, when catarrhal icterus may result. Boas and Schmidt find undigested connective tissue in the movements. (*c*) The *urine* is reduced and shows a lateritious deposit, phosphates and oxalates. (*d*) The general nutrition is maintained if the intestines functionate well. (*e*) The motive and absorptive power of the stomach may be impaired, normal or even exaggerated, the food being hurried into the intestine.

Diagnosis.—Its cardinal points are its steady, chronic course, the presence of mucus and decreased secretion. (For its differentiation, *v. i.* Ulcer.)

Treatment.—1. Prophylaxis.—Recurrence is prevented by treatment of the causal conditions.

2. Hygiene.—Hygiene concerns regularity in living, moderation, recreation, rational exercise and careful mastication.

3. Diet.—Everyone "has the stomach he deserves." Slowness of eating, drinking only after meals and thorough mastication are frequently curative without medicinal treatment; insalivation of the food is vital, since the ptyalin operates for nearly an hour in the unchurned contents of the cardiac end of the stomach. As Ewald states, "die Diät der Magen-Kranken fängt im Munde an." The *teeth* require care, as pyorrhea, carious teeth and fitting of proper plates Food should be taken in moderate amounts, and the stomach given sufficient rest. The relish of food excites the flow of the gastric juice, the "appetite juice" (Pawlow).

Kind of Food.—"Easily digestible food" is less important than careful mastication and insalivation. In severe or alcoholic cases equal parts of milk and Apollinaris water, may be tried, to which grains x each of sodium bicarbonate, sodium chloride and magnesium oxide are added. Predigested milk is indicated in severe catarrh with much mucus formation or atrophy. The diet should be mixed, but proteids are superior to carbohydrates, which are prone to ferment. The *proteids* given are chopped lean meats, scraped beef, boiled mutton, veal, roasted steak, broiled tenderloin, pigeon, chicken and eggs boiled one minute. Beef-tea, fresh beef, hard-boiled eggs, pork, game, fish, warmed-over (therefore hardened) meat, turkey, goose, duck, smoked or cured meat (except bacon) and cheese must be interdicted. *Carbohydrates* are withheld when there is obstinate fermentation. Those allowed are arrow-root, well-cooked sago, tapioca or rice, white bread, zwieback and toast; those occasionally allowed or tried later are mealy potatoes, spinach, pea or bean soups; those forbidden are substances containing much cellulose, as hard or brown bread, oatmeal, coarse vegetables, uncooked green vegetables, most fruits, pies and pastries. Soups (containing meat extract), dextrin, maltose, dextrose, and proteoses, reaching the pyloric region and duodenum, liberate a hormone (gastrin), which, absorbed and carried by the blood to the gastric glands, excite secretion from them. Carbohydrates should be eaten before the proteids, as they leave the stomach earlier. *Fats* are a necessary part of the diet, especially moderate amounts of butter and cream; greasy soups are injurious. Liberal quantities of salt may be used; condiments in moderate amounts are helpful. Tea, coffee, alcoholic beverages and smoking should be interdicted or restricted.

4. Gastric Lavage.—Lavage is considered when there is formation of mucus, fermentation, deficient secretion and delayed digestion, but in the writer's experience is seldom indicated as there is no stagnation of food. Morning lavage is best for removal of mucus, and lavage before the evening meal for delayed digestion or fermentation. Warm water is used, to which sodium bicarbonate (1 to 100) is added to dissolve the mucus, or salicylic acid (1 to 1000) to modify fermentation. The stomach is washed until the water returns clear. When the stomach-tube is contra-indicated, the patient may drink before breakfast two glasses of warm

water to which a teaspoonful each of sodium chloride and sodium bicarbonate has been added—essentially the same as drinking the Kissingen, Wiesbaden and other waters. Sodium bicarbonate acts locally and sodium chloride locally and systematically, for small doses increase the hydrochloric acid and large doses reduce it. Carbonated waters stimulate secretion and neutralize abnormal acids.

5. Medical Treatment.—(*a*) The first indication for *hydrochloric acid* is to convert pepsinogen into pepsin, whence it is useless in atrophic gastritis save as it stimulates pancreatic and possibly biliary secretion. Its second indication is as a digestant; we employ 20 drops of the dilute acid every thirty minutes until ℥j–ij have been given; it dissolves the glutin covering on vegetables and some connective tissue in meats. Its third indication is as an antizymotic, which Boas holds is its sole indication; it should not be given until about half an hour after meals, during which period the saliva is acting upon the carbohydrates; its fourth indication is its appetizing action. (*b*) *Pepsin* should be given as a powder, for solutions rapidly deteriorate and are strongly alcoholic; many forms on the market are inert. Pancreatin is usually ineffective. Taka-diastase is beneficial in some cases. (*c*) *Bitters*, best exhibited before meals, increase the appetite and flow of saliva, gastric and probably also the pancreatic juice. Strychnine and nux vomica are the best drugs and are expecially valuable in alcoholic cases; bitters are given in solution, as their taste is their chief mode of action.

℞—Strychninæ sulphatis . . . . . gr. j
Acidi nitrohydrochlorici . . . . . ℥j
Tr. gentianæ comp. . . . . ℥iv
M. et S.—One teaspoonful half an hour after meals in half a glass of water.

Condurango bark (fluidextract ℥ss) is prepared by macerating the bark in cold water. Some bitters, as gentian, may irritate the stomach and induce diarrhea. (*d*) *Antifermentatives* often cause irritation. Phenol gr. j, creosote ♏j, thymol gr. ss–j, resorcinol gr. ij–v, after meals, act on the stomach and salol gr. x and betanaphthol gr. x (in capsule because of its bitterness) are intestinal antiseptics. (*e*) *Gastric sedatives* are seldom indicated except in the hyperacid type (see page 55). Silver nitrate is sedative, astringent and alterative, but may cause nephritis or argyria; it is given in pill form, half an hour before meals, gr. ¼ (perhaps with small doses of opium or belladonna). Zinc oxide, gr. iij–v, before meals, is valuable in alcoholic gastritis. Extract of belladonna, gr. ¼, relieves pain, especially in hyperacid gastritis. In pyrosis of fatty and other organic acids, alkalies may be employed, sparingly, sodium bicarbonate, creta preparata and magnesium carbonate, each grains x, after meals. Spasm from gas, erosions near the pylorus, or overloading of the stomach is modified by tincture of asafetida ℥j, extract of belladonna gr. ¼ and subnitrate of bismuth gr. x. (*f*) Faradization, massage and hydrotherapy, are less valuable than in gastric atony and sensory neuroses of the stomach. Cathartics cause irritation and constipation recurs; hot water, colonic flushings and perhaps the mildest saline aperients are indicated.

Achylia Gastrica.—Etiology.—The term was introduced by Einhorn to designate total suppression of gastric digestion. Achylia occurs secondarily in cancer of the stomach, various gastritides, cancer of distant organs, diabetes, pernicious anemia, tabes and as a functional disorder. In rare cases it develops as a seemingly primary affection, possibly as a congenital condition. It occurs oftenest in middle or advanced life. Atrophy of the stomach was described by H. Jones, Flint and Fenwick.

Pathology.—Pathologically two main forms exist, (*a*) *cirrhosis ventriculi*, in which the stomach is very small and its walls measure several centimeters in thickness, and (*b*) *phthisis ventriculi*, in which the walls are thin or "wasted" and the stomach is lax or dilated. The glandular structures are wasted (anadenia). In neurotic achylia there is no anatomical change.

Symptoms.—Symptoms may be absent if gastric motility is good and the intestinal glands are normal. If the same process exists in the gut diarrhea, marasmus and death result.

1. Gastric Symptoms.—None are pathognomonic. Pain may be absent, moderate or in some cases lancinating, like that of hyperchlorhydria. The appetite is variable. When vomiting is present the vomitus contains coarse, poorly digested particles.

2. Test Meal.—The recovered food is coarse; the total acidity corresponds to the amount in the food ingested. No HCl is found (achlorhydria), no pepsin (therefore no peptones), no rennet (therefore no coagulation of milk), no blood and according to most writers no mucus; mucus may be due to metaplasia of the gastric mucosa to one resembling that of the intestine, the cells of which secrete mucus; as they in turn atrophy the mucus disappears. Lactic acid is found but rarely. The gastric *motor power* is usually *increased* and the stomach is emptied in less than an hour. In the contracted cirrhotic form, a sound is introduced into the stomach with difficulty, hydrochloric acid is present, the stomach cannot be distended with gas and its capacity is enormously decreased; in the phthisic form ectasia may occur.

3. Other Symptoms.—Headache, vertigo and constipation are the rule; in some cases diarrhea may be the sole salient symptom, simulating an intestinal lesion; these morning movements are inoffensive and usually painless. Symptoms like those of pernicious anemia or like gastric cancer mark some cases.

Prolonged observation may be necessary to exclude the atrophic gastritis of cancer and amyloid gastric disease. In one instance the autopsy showed only atrophic gastritis, whereas clinically hematemesis, achylia, lactic acid, edema, ascites and extreme emaciation had led to a suspicion of cancer.

Prognosis.—Life may be prolonged for years by compensating intestinal digestion, but recovery is impossible after atrophy develops.

Treatment.—Treatment is necessarily palliative. Thorough mastication of food is imperative. Cellulose is to be avoided and thick, plain, leguminous purées should be given. Butter is well tolerated. The meals should be small and given every three or four hours. HCl should

be given in full doses (ℳxx–xxx), stimulating the pancreatic flow and controlling the pylorus (thus averting diarrhea). Water relieves the pain, which resembles hyperchlorhydria.

DILATATION OF THE STOMACH; MOTOR INSUFFICIENCY.

Gastrectasia is only a symptom, and is understood as a more or less lasting dilatation with motor insufficiency.

I. Acute Dilatation.—Acute dilatation was first fully described by Fagge (1883); 217 cases are recorded (Laffer, 1908).

Etiology.—(1) Most cases follow abdominal trauma or anesthesia, affecting the vagus (45 per cent.); gall-bladder operations are the most frequent type. The two cases seen by the writer were in women operated on for fibroids of the uterus. (2) It may follow acute infections as pneumonia or typhoid; or (3) mechanical causes; in one of Kolisko's autopsies the author saw an enormously dilated stomach in a man who on a wager had taken a large number of charged siphons. Unconscious aërophagia is Mathieu's explanation. (4) Traction or obstruction of the superior mesenteric artery in the duodenal mesentery is the most favored etiological factor, though no proof exists that it is not a result rather than cause. (5) Most cases occur between twenty and thirty years of age.

Symptoms.—The stomach is enormously distended; there is vomiting of brown or black material which often runs out of the mouth, may be very copious, sometimes offensive and rarely fecal. Succussion sounds are frequent. The abdomen is greatly distended and often painful. There is much collapse.

Prognosis and Treatment.—Sixty-four per cent. die, usually within five days. The indications are: the knee-chest position or lying on the face or the right side; interdiction of all food and fluid; early and frequent use of the stomach-tube, which may be left permanently in the stomach; rectal feeding; atropine (under which the writer observed one recovery), or eserine gr. $\frac{1}{40}$; salt solution under the skin, and other measures for shock; gastrostomy or gastro-enterostomy is contra-indicated.

II. Chronic Dilatation and Motor Insufficiency.—Various grades of distention and atony may occur; dilatation may be associated with increased power of the gastric musculature or with atony; and motor insufficiency (inability of the stomach to propel food into the intestine) is not necessarily, though very often, associated with dilatation of the stomach. Dilatation and motor insufficiency are most prevalent in middle or advanced life.

Etiology.—1. Pyloric Stenosis.—Its causes are twofold, *malignant and benign*. It is most often *cancerous*, but may follow round or corrosive ulcers, gastritis leading to hypertrophy of the pyloric musculature (stenosing gastritis), benign tumors, as pedunculated polyps, and congenital stenosis. (In pyloric obstruction in nurslings, the cause is either pylorospasm or hypertrophy of the pyloric musculature, from its repeated irritation.) It may also result from congenital duplicature, volvulus of the stomach, angular bending of the fixed pylorus or from cholecystitis. Garré collected 25 cases of tuberculous pyloric obstruction.

Stenosis of the duodenum produces essentially the same results as pyloric stenosis and may follow duodenal ulcer, diverticulum or twisting. The pylorus may suffer compression stenosis from tumors of the liver, colon, kidney or pancreas. Pyloric stenosis may be associated with intestinal stenosis (chiefly from round ulcer). Temporary or permanent pyloric spasm from fissure, erosion, ulcer, hyperchlorhydria or from appendicitis may cause obstruction. The pathological sequence of pyloric stenosis is retention of food within the stomach and hypertrophy of its musculature in attempts, sometimes partially successful and at other times vain, to force the food through the narrow or closed pylorus. Dilatation of the stomach may be considered as a disturbed compensation, analogous to hypertrophied and dilated bladder from prostatic enlargement or hypertrophy with dilatation in obstructive valvular disease. In all three instances the hypertrophied muscle eventually degenerates.

2. Muscular Weakness.—Motor insufficiency, atony or myasthenia gastrica constitutes, in contrast with the first group of causes, a functional and, generally speaking, a more reparable type. (*a*) *Atony dependent on general causes* may occur in acute infections, anemia, rickets and neuroses. The tendency may be acquired or congenital. (*b*) *Atony may result from gastric conditions,* as cancer, gastritis, hypersecretion, peritoneal adhesions, cholecystitis with pyloric adhesions, diaphragmatic pleurisy and diastasis of the recti muscles. *Overloading the stomach* with heavy meals, indigestible foods, vegetables, carbohydrates, beer, milk in large quantities, much water ingested with heavy meals, as in diabetics, swallowing of foreign bodies by insane subjects, etc., may develop muscular insufficiency. The stomach is usually enlarged and shows a lax, often wasted and thinned, musculature but seldom contains residual food.

Symptoms.—The symptoms differ greatly, ranging from absolute pyloric stenosis to the least motor weakness, and are partly mechanical, partly chemical.

1. Gastric.—(*a*) The appetite is variable; the tongue is sometimes coated, sometimes clean. (*b*) There is usually thirst and dryness of the throat, most frequently in hypersecretion. (*c*) Singultus, *pyrosis* of fatty acids, oppression over the stomach and eructation of gas are frequent. Gases are never formed without motor insufficiency; they include hydrogen sulphide, hydrogen, oxygen, nitrogen, carbon dioxide and even inflammable gases. (*d*) *Vomiting* is *always present in pyloric obstruction* and marked motor insufficiency. It occurs easily and is often rather belched up than vomited. It frequently occurs during the night or at intervals of days; five quarts may be evacuated. *Large amounts argue directly for stagnation and dilatation.* The vomitus is acid, sour and often greatly decomposed. Three layers are noted: a brownish foam, a grayish-brown fluid and a sediment of food, stones and seeds, which may have lain for days in the stomach. Gas often bubbles up as the vomitus stands. If gastric juice only is found, it indicates hypersecretion with motor insufficiency. Chemically, butyric acid, peptones and, in cancer, lactic acid, are found; when the obstruction lies in the duodenum below the papilla of the common duct, bile is also present. Micro-

scopically undigested meat or starch, in benign stenosis sarcinæ and in cancer the Oppler-Boas bacilli and yeast are found. HCl is absent in cancer. (*e*) *Pain* is cramp-like, boring or burning.

2. Motility and Resorption.—(*a*) The *chemism* depends on the causal affection (see Ulcer, Cancer, Hyperchlorhydria, Hypersecretion). Hyperchlorhydria and hypersecretion are usually present except in malignancy and are sequential rather than causative. (*b*) *Motility* is best tested by *Leube's test dinner*, consisting of 13 ounces beef soup, 7 ounces beefsteak, one or two ounces bread and 7 ounces water, which should leave the stomach physiologically in not more than seven hours; jam is added, for seeds are a ready index of stagnation. If food is found later, stagnation is present; if found in the morning when Leube's meal has been given at night, there is great stagnation and fermentation. Fluid is not absorbed by the stomach, whence the measure of its residual amount is important. There is more fluid in the stomach than was ingested, which is accounted for by hypersecretion, transudation, mucus and perhaps regurgitation from the intestine or hemorrhage. (Other tests are less reliable—the oil test, administering 100 c.c. of olive oil, 70 to 80 per cent. of which should leave the stomach in two hours; and the salol and potassium iodide tests.) In general a homogeneous chyme means that the motor function is normal; coarse particles occur when there is motor insufficiency.

3. Physical Signs.—(*a*) *Inspection* in a good light is very important. A visible prominence of the dilated organ may be seen below the navel, sometimes showing clearly the abnormally low upper and lower curvatures, the upper curvature never being seen normally. In obstruction a peristaltic wave passing from left to right or an antiperistaltic wave in the opposite direction often tells the whole story. Peristalsis disappears when the gastric musculature is exhausted or infiltrated, *e. g.*, by carcinoma. "Stiffening" of the stomach is an infallible sign of pyloric stenosis. The *x*-rays show the outline of the viscus after ingestion of bismuth, hyperperistalsis, stasis, etc. (see Ulcer and Cancer). (*b*) *Palpation* confirms the above findings and also elicits splashing (*clapotage*) of gas and water in the stomach; it occurs in healthy stomachs. The hand may palpate the final gush of gastric contents into the gut, or outline a tumor (adhesions, callus, pyloric spasm, cancer). (*c*) *Percussion:* The normal vertical dimension of the stomach is 10 to 14 cm. and its average capacity 1600 c.c. Filling the stomach with water and percussing, with the patient standing, outlines both the lesser and *greater curvatures*, which may reach to the pubes. Siphoning the water and then distending the stomach with carbon dioxide, replaces the flatness by tympany, though distention is dangerous. When doubtful, the colon may be distended with gas or fluid. The greatest distention is at the fundus. The note changes with change of posture, the fluid being in the lower and the gas in the upper parts; the most experienced have confused enormously dilated stomachs with ascites and ovarian cysts and have introduced a trocar; in Jadon's case the stomach contained 45 quarts. Leube palpated the lower end of the stomach by means of a rigid sound; this practise is abandoned. (*d*) *Auscultation* elicits the splashing which may be heard by the patient,

and sometimes metallic heart tones, crepitation, hissing of gas or a dripping sound as the patient drinks.

Complications.—(*a*) *Constipation* is the rule, because less fluid passes the pylorus; in the exceptional diarrhea, sarcinæ may be found in the stools. (*b*) The *urine* is often *alkaline,* its chlorides lessened and its amount reduced as a result of acid vomiting; acetone and diacetic acid are occasional. (*c*) The skin is dry and emaciation common, even in benign forms because food is imperfectly absorbed or lost to the organism by fermentation and possibly by tissue-destroying toxins. (*d*) Autointoxication may develop, causing headache, vertigo, drowsiness, psychoses, polyneuritis, tetany, slow pulse and dyspnea. Tetany (Neumann and Kussmaul) occurs oftenest in hypersecretion or stenosis from cancer or ulcer; 88 per cent. of cases die.

Diagnosis.—The *vomiting, impaired motility,* as shown by Leube's test dinner, the *visible outlines, x*-rays, and the low lesser curvature which is not seen normally, distinguish gastrectasia easily; its cause may be determined with difficulty only. The stomach may be physiologically large (*megalogastria*), but then there is no motor insufficiency, no vomiting and no stagnation. *Gastroptosis* will be considered later. Stenosis may be intermittent, latent and misinterpreted (stenose méconne du pylore); a test of the digesting or fasting stomach may reveal nothing between the attacks when muscular compensation is good. The *degree* of *stasis* is determined by washing out the stomach at varying times after the test dinner. Retention of food after twelve hours almost certainly indicates ulcer or cancer. In *pyloric stenosis* there is greater dilatation than in simple motor insufficiency (or atony) and marked peristalsis always indicates obstruction. The benign must be differentiated from the malignant type; *benign pyloric obstruction* runs a more remittent, longer course, over two years, even fifteen years; HCl and the ferments are always present, often increased and sarcinæ are often, and lactic acid seldom, present; there is more supernatant fluid and the total acidity runs 70 to 100 or over, and the free HCl 50 to 80; while in *cancer* the march of symptoms is progressive and achlorhydria, cachexia, anemia with leukocytosis, hydrops, pyloric tumor, the Oppler-Boas bacillus and metastases develop with relative rapidity. The *spasmodic* form runs an intermittent course. The *prognosis* depends on the etiology and amenability to treatment; spasmodic types are curable; relative stenosis may somewhat improve. *In infants* with spastic obstruction or hypertrophied pylorus the onset within the first month of life is characteristic, with forcible vomiting, though the child be carefully fed, emaciation, visible peristalsis, constipation, and usually a palpable pylorus. The 23 cases of volvulus of the stomach (Mühlfelder, 1911), showed gastric meteorism, difficulty in introducing the stomach-tube, singultus, symptoms of ileus, sinistrocardia, dysphagia and thoracic pains.

Treatment.—1. *Etiological factors* should be treated.

2. *General measures,* to give tone to the musculature are *abdominal massage,* only when ulceration and inflammation are excluded; *drugs,* especially strychnine and atropine, and an ice-bag to the epigastrium; *hydrotherapy; faradization* of the stomach; external is seemingly as

efficacious as intragastric application; and *abdominal binders or supports*. This treatment avails only in atony.

3. *Gastric therapy.* (*a*) *Gastric lavage* removes the fermenting contents, relieves the pressure exerted by accumulated food and fluid, cleans off the mucus, lessens vomiting and auto-intoxication, and may permanently help atony and temporarily mitigate stenosis. The stomach is washed with luke-warm water until it returns clear; salicylic acid and sodium bicarbonate may be added, as in chronic gastritis; the patient soon welcomes the relief afforded; washing two hours before the evening meal is preferable, to insure less stagnation through the night. (*b*) *The food* should be well divided, nourishing not voluminous, and given with moderate quantities of water, in small amounts and at intervals of four hours. The diet consists of meat, eggs, leguminous purées, thoroughly cooked rice, small amounts of butter (fats and carbohydrates are more prone to fermentation), and an ounce or two of olive oil in pylorospasm and moderate benign stenosis. A dry diet was advised by Van Swieten, but moderate amounts of fluid aid in propulsion of food into the gut. A fluid diet is indicated in great pyloric stenosis and when the HCl is decreased; meat may be given if the acid is not decreased and the pylorus is not greatly stenosed. Recently there has been a reversion in favor of two fair-sized meals a day, giving the stomach time to empty itself and rest. Water is not absorbed by the stomach; indeed, absorption from the stomach of sugar, peptone, dextrin and alcohol is attended by actual excretion of water into the stomach. In pronounced cases peptonized milk and water are given by rectum to supplement gastric feeding. In all cases careful individualization is indicated; the degree of insufficiency, *i. e.*, the time required for evacuation, should be tested by Leube's meal and the patient fed accordingly. (*c*) *Posture:* The patient should lie on his right side one or two hours after eating, thereby relieving pressure upon the greater curvature and directing the food toward the pylorus. (*d*) *Medication:* (See CHRONIC GASTRITIS, ULCER, HYPERCHLORHYDRIA). (*e*) *Thiosinamin:* (see page 553).

4. *Surgical intervention* is imperative in severe or malignant stenosis and when medical treatment fails. It embraces (*a*) pylorectomy, (*b*) pyloroplasty, (*c*) gastro-enterostomy. In 153 operations for benign stenosis Moynihan had only two deaths. In the pyloric stenosis of infancy the mortality under medical treatment is 72 per cent., under surgical 17 per cent. (10 per cent., H. M. Richter). In 24 operations for tetany with pyloric stenosis 21 recovered (McKendrick).

CHANGES IN FORM, SIZE AND LOCATION OF THE STOMACH.

Form.—The *antrum cardiacum* is a congenital bulging of the esophagus just below the diaphragm and the fore-stomach is a congenital dilatation above the diaphragm. There also exists a congenital narrowness of the pylorus as well as hour-glass stomach.

The acquired *hour-glass deformity* is more common and may result from spastic hypertrophic muscle, *ulcer*, especially cancer developing upon ulcer, adhesions, abdominal tumors, peritoneal tuberculosis,

twisting of the stomach or hernia of it through the mesocolon. It may coexist with pyloric stenosis. Randolph and Thomas observed 2 cases in one family. The pyloric sphincter normally contracts so vigorously as to give an hour-glass appearance in many *x*-ray plates.

Symptoms may be lacking in the lesser degrees of deformity; in the higher grades they are those of stenosis. (1) Only a part of a given amount of water introduced into the stomach may be recovered (Wölfler's first symptom). (2) After the stomach has been washed till the water returns clear the fluid may suddenly become clouded (Wölfler's second symptom). (3) The stomach is apparently emptied by the stomach-tube and yet splashing is obtained, from the cavity nearest the pylorus (Jaboulay's paradoxical dilatation). (4) Inflation by carbon dioxide produces distention first of the left and later of the right half of the epigastrium (von Eiselsberg's first symptom). (5) Normally when the stomach is distended and the gas passes through the pylorus into the bowel, there is a sharp gurgle over the pylorus; in the hour-glass stomach the gurgle is heard two to four inches to the left of the median line (von Eiselsberg's second symptom). (6) On distention by carbon dioxide Moynihan found tympany only over the upper part of the stomach and not over its lower half. (7) When the stomach is distended a furrow may appear (Eichhorst and Schmid-Monard). (8) Filling the stomach with water and illuminating it by a light introduced into the stomach, the cardiac portion appears translucent and the pyloric part dark (Ewald). (9) A rubber balloon, introduced into the stomach and then inflated through the stomach-tube, fills or outlines a part only of the stomach (Hemmeter).

Treatment is surgical. Moynihan successfully operated on 23 cases by single or double gastro-enterostomy, gastroplasty, gastrogastrostomy, partial gastrectomy and dilatation of the stenosis.

Size.—Megalogastria and microgastria are usually accidental findings if not associated with other gastric disease. Microgastria may be congenital, or result from spasm, infiltrating cancer or chronic *stenosing gastritis* (linitis plastica). The latter condition is a diffuse or circumscribed cirrhosis of the stomach. Many cases of carcinoma are brought under this caption, even after microscopic examination. The connective-tissue increase may be localized, as at the pylorus, or diffuse, involving the entire stomach, particularly in its submucous coat. The localized form gives the usual symptoms of pyloric stenosis. The generalized type is attended by pain, vomiting and the toleration, by the stomach, of small amounts of food; a rounded, or sausage-shaped tumor is felt in the epigastrium. The prognosis is grave unless surgery is invoked.

Location.—The stomach at birth is vertical; in adult life three-quarters or the whole stomach is to the left of the median line.

Gastroptosis.—(See ENTEROPTOSIS, page 555).

ULCER OF THE STOMACH AND DUODENUM.

Definition.—Ulcer is characterized pathologically by a "punched out" funnel-like ulcer, oftenest near the pylorus, and clinically by localized pain and tenderness, hematemesis and hyperacidity.

Frequency.—It is found in 2.5 to 5 per cent. of all autopsies.

Etiology.—The causation is obscure. (*a*) *Age.*—Most ulcers occur between twenty and sixty years of age, with about equal frequency in each decade. (*b*) *Anemia* and chlorosis are predisposing factors. Anemia and (*c*) hyperacidity are usually regarded as important factors; the writer believes that hyperacidity is only a result. (*d*) As a normal access to the stomach of arterial blood prevents its autodigestion, it is thought that interruption in the local circulation is the cause of ulceration (embolism and arterial spasm). Embolism and ulceration follow burns (13 per cent.). However, ulcer often occurs at an age when arterial changes are rare, though syphilis, alcoholism, arteriosclerosis, hard work, cardiac, renal and hepatic diseases apparently cause some of the cases in later life. (*e*) *Occupation.* Many cases occur in servant girls and in cooks; (*f*) trauma in cooks, shoemakers and tailors is an accessory factor; the tasting of hot foods and pressure against the stomach; the pressure of corsets, the swallowing in certain trades of particles of iron, porcelain and glass; and imperfectly masticated or improper food, may be brought under this caption. External trauma is a distinct factor. (*g*) *Bacterial necrosis* may cause ulcer, as in Dieulafoy's cases of pneumococcic hemorrhagic ulceration of the stomach. (*h*) The idea was recently advanced that ulcer results from lack of anti-enzyme, which normally resists necrosis or digestion. Operative figures indicate that duodenal ulcers are twice as frequent as gastric, but these statistics represent a narrow group of cases; Mayo's figures give 64.5 per cent. duodenal, 32.5 per cent. gastric and 3 per cent. both.

Pathology.—Scars indicating healed ulcers are three times as frequent at autopsy as open ulcers. The ulcer is known as *ulcus simplex* to distinguish it from malignant or syphilitic ulceration; *ulcus rotundum*, as the smaller ones especially are round; *ulcus pepticum* and *perforans*. The peptic ulcer occurs almost exclusively in the stomach, lower esophagus and upper duodenum. Its general morphology was first thoroughly described by Cruveilhier (1829) and Rokitansky (1839); it has a "punched out," clean-cut appearance; in contour it is round or oval in small, and irregular in large, ulcers; it is usually small, but may even measure four by seven and a half inches; it is funnel-shaped in old cases; its apex is eccentric and directed toward the serosa, its sides are terrace-shaped and its floor is clean. In 90 per cent. the ulcer is *single;* as many as 34 ulcers have been reported. Extension is more common in acute than chronic types and more toward the peritoneum than laterally. Microscopically there is no infiltration with round cells. Its *localization* in 90 per cent. is near the pylorus on the posterior wall or lesser curvature; its *complications*, as hemorrhage and perforation, and its *sequelæ*, as pyloric stenosis and deformations, will be considered under clinical symptoms.

Symptoms.—The *general clinical picture* varies, cases being typical, doubtful or latent.

In the *typical cases* there is an epigastric pressure, which later becomes a cutting, *localized pain;* this is observed one and a half to two hours after eating, and ceases only with vomiting or passage of the food into

the bowel. The appetite is usually good; *hyperacidity* and hypersecretion are noted in the test meal or the vomitus; *tenderness* occurs over the epigastrium, and in half the cases *bleeding* from the stomach or blood in the stools is observed.

The *typical triad* of cardinal symptoms includes pain with localized tenderness, hematemesis and hyperacidity.

1. Pain.—The most important, early, characteristic and constant symptom (in 90 per cent.) is pain, which is paroxysmal and is localized and occurs during digestion. It is boring or burning, seldom lancinating. The *diet* affects the pain greatly; milk is well tolerated, usually, but solid food and very *hot* or very *cold foods* or beverages cause discomfort. This disappears quickly with rectal feeding. The seat of the pain varies in different patients, but is the same in a given case. Generally it is *epigastric* and near the ensiform or cramp-like in the right mammary line under the liver. Sometimes there is wide irradiation toward the sternum, ribs, shoulder, scapula, lower abdominal quadrants, and infrequently in the brachial plexus and pulmonary branches of the vagus, resembling angina pectoris or producing pain in the arms and legs. The pain may vary with change of position, depending on the localization of the ulcer.

Fig. 37.—Normal adult stomach. *x*-ray after bismuth meal. Note that the duodenal cap and pyloric part of the stomach are to the right of the spine. (*x*-ray by Dodd.)

In relation to eating, sometimes the pain occurs two to ten minutes after eating, the old "wound pain," supposedly due to contact of food with the eroded nerves in the ulcer. *Typically*, pain occurs at the height of digestion, *i. e., one and one-half or two hours after ingestion of food*, caused by the hyperacidity. Patients fear to eat because of the inevitable pain. Pain may increase at the time of the menses or cease during pregnancy. Pain from the scars or adhesions of an ulcer is infrequent and is usually drawing or vague. In old ulcers pain may be atypical or absent. If pain comes on at once it may suggest esophageal or cardiac ulcer; or, if it occurs three hours or more after eating it is suggestive of ulcer of the duodenum.

Moynihan describes the symptoms of *duodenal ulcer* as follows: "The patient tells you that he has certain definite attacks, and if you let the man tell his own story, he will give you the impression of having read something which has been written about duodenal ulcer, which he is recounting to the best of his recollection to please you. He says that his trouble comes on in attacks which are nearly always worse in winter than in summer and are very apt to be precipitated by a chill. He takes a

meal at eight in the morning and from two to two and one-half hours after it he is fairly comfortable; it is his best time. At the end of that time he has a feeling of discomfort in the epigastrium; he feels full and heavy and may get some relief from the belching of gas. Some of these patients develop a habit of belching. They may bring up a very sour fluid, which tastes very bitter and acid and makes the mouth dry and the teeth chalky. This pain gradually increases until the next meal-time comes. To this I applied the name of 'hungry pain.' At the next meal the patient almost instantly gets relief and that relief persists for two or three hours again. He probably eats a heavy dinner and will nearly always tell you he has something before he gets into bed, a glass of milk or a cup of cocoa and a biscuit. He sleeps comfortably, until he wakes about 2 A.M. He get relief from nibbling a biscuit, which he keeps at the bedside. The pain is found to be most relievable by something stodgy and indigestible. Taking an alkali relieves the pain; so will washing the stomach."

Tenderness is most often found under the ensiform. It is usually *circumscribed* and is more often and advantageously found on the lightest touch rather than on deep palpation; rigidity is a protective reflex. Tenderness in the back (Cruveilhier) suggests ulcer on the posterior wall of the stomach, often with adhesions to the pancreas, and is found in 30 per cent. of cases to the left of the tenth to twelfth dorsal or upper lumbar vertebræ. The abdomen, back and chest are often exquisitely hyperesthetic.

2. HEMATEMESIS.—The second cardinal symptom is hematemesis. Its average frequency is 50 per cent. Vomiting of blood in ulcer is most frequent in the pyloric ulcer (posterior wall and lesser curvature) or, less frequently, from ulceration reaching the liver, panrceas or spleen. Hemorrhage when small may be overlooked (occult hemorrhage); melena alone is found in 11 per cent. of cases. The hemorrhage must amount to a pint to turn the stools black. It may be large and lethal; no blood may be vomited and the stomach and bowels are found filled with one massive clot. The usual signs of internal hemorrhage exist, as collapse, pallor, moderate fever, hemic murmurs, amaurosis and rarely optic atrophy or hemiplegia. If profuse it is voided *bright red;* if slow and moderate the acid turns it dark, *"coffee-grounds" vomitus.* If red blood cells cannot be found because of complete disintegration of the cells, blood can be detected by treating it with acetic acid, extracting with ether and then testing with the guaiac and turpentine reaction. *Occult hemorrhage* occurs in 25 to 50 per cent. of cases but is not as constantly present as in gastric cancer (see HEMATEMESIS AND CANCER). The tests for occult hemorrhage are valuable to determine the length of the medical treatment of ulcer and to detect bleeding in apparently normal vomitus. Profuse hemorrhage usually indicates deep ulceration. Hemorrhage complicates acute more than chronic cases. The hemorrhage is usually fairly profuse in ulcer; it may be spontaneous during sleep or rest or may result from excitement, exertion or improper diet. One or two hemorrhages are the average but they may recur many times. In a case of "chronic dyspepsia" with vague symptoms the writer inflated the

stomach with carbon dioxide; the patient collapsed in the office and vomited nearly a quart of blood.

3. Hyperacidity.—This is the third cardinal finding. Leube finds it in 50 per cent. In old ulcers hyperchlorhydria is present in only 10 per cent. and they explain most of the cases of decreased acidity; in fresh ulcers hyperacidity occurs in 70 per cent. Hyperchlorhydria is probably due to irritation of the vagus (Pawlow), and like hypersecretion, develops notably in ulcers near the pylorus and especially with stenosis. Complicating cancer, hypersecretion or catarrh may cause subacidity.

Test meals should not be given when there is pain, tenderness and vomiting of blood, although some employ the stomach-tube for diagnosis in atypical cases, and also to relieve the vomiting and to introduce silver and bismuth. Organic acids, as lactic, are absent, the carbohydrates poorly digested and the albumins are rapidly converted into albumoses or peptones. The motility of the stomach is normal in one-quarter and slow in three-quarters of the cases.

4. Other Symptoms.—*Vomiting,* present in 75 per cent., is a less valuable diagnostic than pain. It occurs at the height of the pain, two or three hours after eating, whereas in dilatation or hypersecretion it occurs much later. Vomiting and pain depend directly on the diet and localization, vomiting and hypersecretion denoting chiefly pyloric ulceration; vomiting usually relieves the pain. The vomitus consists of fine particles, is sour, grits the teeth and shows much acid and no fermentation. The appetite is frequently good or exaggerated. Acid eructations may cause a burning sensation back of the sternum or between the shoulder blades; much esophageal burning indicates incontinence of the cardiac orifice. Diarrhea is rare. Constipation is due to anemia, diet, rest, vomiting, possibly reflex inhibition of intestinal motility or to impeded peristalsis by scars and adhesions. Dysmenorrhea and amenorrhea are not infrequent. Emaciation and anemia may become profound. The urinary findings are not characteristic; reduction of the chlorides does not differentiate from carcinoma; the urine is slightly acid or alkaline when the stomach is dilated; the phosphates are increased; with dilatation, indican and ethereal sulphates are increased. Acetonuria and diaceturia sometimes result from inanition.

Clinical Forms of Lebert.—(*a*) In the *hemorrhagic* form bleeding may be acute or chronic, the first or the last and fatal symptom. (*b*) In the *perforative* form there may have been no previous symptoms. (*c*) In the *dyspeptic* type there are atypical symptoms, such as pain which is not sharp, vomiting which is infrequent and excess of hydrochloric acid, but no increase in mucus. (*d*) The *cachectic* form, in old ulcers with dilatation and hypersecretion, may be confused with carcinoma. Other types are (*e*) *gastralgic* or neuralgic form; (*f*) *vomitive* form; (*g*) latent ulcer (scars at autopsy); (*h*) a form with *tumor;* (*i*) *recurrent* type; (*j*) *stenotic* type; (*k*) secondary *carcinomatous* type.

Clinical Course.—Ulceration is frequently long latent before diagnosis is possible and persists some time after all symptoms have disappeared. The course may be acute and rapid, especially under appropriate therapy, but averages three to five years (even twenty to thirty-five years, due to

pyloric obstruction, dilatation or adhesions.). Very large ulcers probably never heal. They frequently *remit* with appropriate therapy, but recur, especially in the spring and fall, or with the resumption of the old diet. Ulceration can be considered cured only when all symptoms have been absent for six months.

Localization.—Tenderness and tumor are indicative of ulcer on the anterior wall; hemorrhage and dorsal pain suggest posterior involvement. Pain in a lateral decubitus suggests localization in the fundus or pylorus; dilatation, in the pylorus or duodenum; pain under the ensiform during the act of deglutition suggests cardiac ulcer. Ulcers are palpable in only 0.1 per cent. of cases. The percentage of involvement of the cardia is 6; of the fundus 4; of the lesser curvature 36; of the posterior wall 30; of the pylorus 12; of the anterior wall 9. In over 80 per cent. of cases it occurs in a small area about the pylorus, lesser curvature and posterior wall.

Complications and Sequels.—(*a*) *Perforation* (3 per cent.) may occur into the peritoneum, usually with collapse, peritonitis and vomiting (in 33 to 66 per cent. of cases). Perforation may be prevented by omental, peritoneal, pericardial and pleural adhesions. The danger of perforation in ulcer of the anterior wall is seven times greater than of other locations. Perforation of a latent ulcer may be confused with rupturing appendicitis or salpingitis. Perforation may occur into the heart, colon, gall-bladder, portal vein, pericardium, thoracic or abdominal wall; or it may produce pneumothorax subphrenicus, which is suggested by pleurisy, epigastric and hypochondriac pain, stiffness in the back, painful eructations and swallowing, singultus or local edema. In 90 per cent. of all perforations there is a history of ulcer covering over three years (Brunner). (*b*) *Simple peritonitis* (perigastritis) may cause a friction murmur over the stomach. Perigastritis may invade all contiguous organs and cause the greatest gastric intolerance. (*c*) *Adhesions* develop in about 40 per cent. of ulcers; they are protective against perforation, but may impede cicatrization of the ulcer or lead to erosion of the pancreatic or hepatic bloodvessels; they may impede motility, deform the stomach (hour-glass stomach) or obstruct either orifice or even the bowels; 75 per cent. of adhesions are to the liver and pancreas. The dragging pain of adhesions is increased by exercise, standing and eating. Pain in the shoulder, back or chest, suggests adhesions to the liver, pancreas and diaphragm, respectively. Dilatation of the stomach indicates pyloric stenosis, or less often, pyloric spasm. Troublesome adhesions may finally so stretch as to relieve all symptoms. (*d*) According to Liebert (9 per cent.) or Zenger, the majority of cases of *cancer* develop from ulcer. The typical symptoms of ulcer are followed by loss of weight, occult bleeding, gradual lessening of hydrochloric acid and other evidences of carcinoma. Polyneuritis is a rare complication.

Diagnosis.—The diagnosis is determined by the cardinal symptoms and various clinical types enumerated. In general it is more easy in the young than in older individuals with chronic ulcer.

Pain is often the only symptom; if it is atypical, hyperacidity may indicate ulcer; according to Riegel pain in simple hyperacidity is less

regular than in ulcer and localized tenderness is absent. Where pain alone exists, one must remember the gastric crises of tabes, small hernias in the linea alba, and pain in the transverse colon, which usually ceases on evacuation of the bowels. Without careful examination movable kidney may be confused. The pain in *gall-stones* is paroxysmal, but more irregular and independent of eating. The gall-bladder is tender and may be distended and palpable. The paroxysm in gall-stones is longer, far more violent and is often attended by fever and a chill. The pain more often radiates to the right side and to the shoulder and the vomiting is not that of ulcer, though hyperacidity and gastric dilatation may follow cholelithiasis. As to *duodenal ulcer* Graham states "There remains a certain proportion of cases that will mislead. Those cases of gall-stones in which the stomach symptoms of gas, distress, sour belching and dilatation predominate, and pain is only of a dull character, will usually be diagnosticated as ulcer; but the duodenal case, of which the chief symptom is the sudden, sharp, intense pain of perforative peritonitis, and in which, with no obstruction or hyperacidity, the other stomach symptoms are in abeyance, will be diagnosticated cholelithiasis. Differentiation in many cases must be made on the operating table."

In differentiating ulcer, gall-stones and *appendicitis,* we consider the chronicity and periodicity of the symptoms, the "hunger" pain, vomiting and eructations of gas, which occur in all three; the time of the pain, its regularity and its relief by vomiting, eating or alkalies determine an ulcer. Even in "appendix dyspepsia" we may observe pain, vomiting and hematemesis. In gastric arteriosclerosis, pain may follow a full meal with or without exercise. Chronic poisoning, as by arsenic, may be suspected, as in the case of the Duchess of Orleans until Littré diagnosed ulcer.

Hemorrhagic erosions and fissures of the stomach present no constant clinical picture. After eating there is a sense of burning over the entire stomach. The chemism and tender points of ulcer are lacking, though Cohnheim finds hyperchlorhydria and believes that solid food relieves the pain. Shreds of tissue are always found, according to Pariser, which cease with cessation of pain.

Pyloric spasm may also occur in cardiac disease, appendicitis, tenia, ascaris, spastic constipation or nicotinism.

Differentiation from *carcinoma* is not often necessary. In Kraus's clinic the author saw 2 cases; 1 was an old man with cachexia, pyloric tumor and decreased hydrochloric acid, which was diagnosticated cancer; the other was a young girl with vomiting of blood, in whom the diagnosis of ulcer was made. The postmortem examination showed that the old man had a round ulcer and the girl had a cancer. The author has seen 15 cases of carcinoma under the age of thirty. (See table on page 517.)

X-*ray Findings.*—(*a*) In *gastric ulcer;* the finding of bismuth in the stomach after six hours indicates atony, tumor or spasm or stenosis of the pylorus. The pylorus may be displaced upward to the left, by ulcer on the lesser curvature, bringing the pylorus and cardia nearer together. The last part of the greater curvature may be drawn up vertically, instead

of curving upward to the right. Fluoroscopy may detect adhesions (causing displacement), antiperistalsis (indicating organic disease, as cancer or ulcer), puckerings, indentations, filling defects, protrusions, partial or imminent perforation or an organic or spastic hour-glass stomach. (*b*) In *duodenal ulcer*, we may observe gastric hyperperistalsis; residual bismuth in the duodenum or in the stomach if stenosis from scar contraction occurs; hypermotility with early emptying of the stomach; tenderness over the ulcer; irregular outline of the cap or first portion, where 95 per cent. of postpyloric ulcers are found; and dilatation of the duodenum or diverticulum of a perforating ulcer.

Prognosis.—The mortality averages 7 per cent., about equally divided between hemorrhage and perforation; in over one thousand cases, death occurred in 2 per cent. (Leube). Very deep, old ulcers rarely heal. The deeper the ulcer the greater the danger of hemorrhage and perforation. Ulcers with hypersecretion give a relatively poor prognosis. Carcinomatous degeneration must be considered when treatment is not successful.

Treatment.—1. Absolute Rest.—Rest in bed for a month or more was recommended by Cruveilhier, in addition to which Leube advises hot fomentations to the epigastrium, which relieve pain within a few days. Women should remain in bed during menstruation, even after recovery.

2. Absolute Rest of the Stomach.—The ingestion of even small quantities of food or water excites peristalsis and secretion and distends the ulcer. Rest permits the stomach to contract and the edges of the ulcer to come more completely in apposition. The more absolute the rest of the stomach the more rapidly the ulcer granulates and cicatrizes. Chronic ulcers show little tendency to cicatrize. In *rectal feeding* the usual rules should be followed; pancreatized milk, grape-sugar, baby foods, meat juice and eggs, should be given, in amounts not to exceed eight ounces, at eight-hour intervals; they should be injected slowly, with the patient on his left side and should be given warm, which precautions obviate excessive peristalsis. A fountain syringe with a soft tube is always used. Nutrient rectal enemata can be retained invariably if the technique is carefully carried out. Wine or spirits irritate the bowel. Rectal tenesmus is obviated by administering, a short time before the enema, an opium suppository of one-half grain of the extract. A patient in bed can manage with 1800 calories; in rectal alimentation by milk only 300 calories are absorbed; enemata of 10 per cent. dextrose, by the drop method, increase the nutrition and lessen the acidosis of starvation. Peptonized milk is far inferior to milk pancreatized for twenty-four hours, in which amino-acids abound. Among 1000 gastric ulcers, parotitis developed in 4.5 per cent. on the strictest diet, but only in 0.4 per cent. when water was given by mouth (Rolleston and Oliver). Water may be given by rectum or food by the duodenal tube.

3. Pain.—Rest of the stomach relieves pain in most cases; small doses of opium or atropine by rectum, may be given, but there is danger of the habit.

4. Superacidity.—Treatment should be initiated after one week of gastric rest. *Massive doses of sod. bicarbonate* ℥ss–j in a starch wafer,

PLATE XIV

Fig. 1

Fig. 2

Fig. 1.—Ulcer of Lesser Curvature, Retraction of Lesser Curvature, drawing Pylorus up and to the left. The so-called "snail-form," described by Haudek. (Radiologist, Dr. Leaming.)

Fig. 2.—Ulcer of Lesser Curvature and Pylorus. The typical bowl-shaped bismuth residue is not as marked as is usual, although pyloric stenosis exists. (Radiologist, Dr. Busby.)

Fig. 3

Fig. 4

Fig. 3.—Ulcer of Lesser Curvature. Spasmodic Incisure of Greater Curvature. (Radiologist, Dr. Busby.)

Fig. 4.—Ulcer of Pylorus with Adhesions and Distortion, Closely Resembling Carcinoma. (Radiologist, Dr. Busby.)

(From Lockwood's Diseases of the Stomach.)

	Cancer.	Ulcer.	Chronic gastritis.	Gastralgia.	Hyperchlorhydria.
ETIOLOGY	75 per cent. over 40 years.	75 per cent. under 40 years—2 to 4 times oftener in women.	Males.	Women.	Males.
TONGUE	Heavily coated.	Usually clean and red.	Grayish-white fur.	Normal.	Clean.
APPETITE	Usually anorexia.	Good; only fears to eat.	Poor.	Variable. Sometimes perverted.	Good, often increased.
PYROSIS	Often pyrosis.	Water brash.	Frequent.	None.	Present.
BELCHING	Usually fetid belching.	Usually absent.	Copious.	Variable.	Usual.
PAIN	The pain is less intense, but more constant; seldom free intermissions, during which no distress is felt in gastric region. Pain not increased at the height of digestion. Tenderness less frequent and intense.	Intense; appears shortly after meals; grows severer on pressure; disappears at the end of digestive period; perfectly free periods more frequent; paroxysmal.	May be present but not pronounced and rarely actual pain but tenderness of diffuse character. Lancinating pains in atrophic gastritis only.	The pain appears without regularity, and not dependent upon meals; may be relieved by pressure; intervals of several days free from pain. Tenderness rare; conforms to outlines of the stomach. Electrical test (Leube) during digestion; if pain decreases, it is gastralgia; if it does not, may be ulcer or gastralgia.	The pain appears about one or two hours after meals, disappears after eating, especially meat, milk or eggs, or after the administration of bicarbonate of sodium. Pain is less regular.
VOMITING	Usual. Hours after eating or once a day or alternate days, etc. Copious, coarse mucus, fermented.	Frequent—at height of digestion—smaller amount, finely homogeneous, no mucus, acid.	Frequent; mucus.	Rare; no mucus.	Much less frequent, then acid and no mucus.
HEMATEMESIS	Frequent—small, dark, like coffee grounds, decomposed, usually late in disease.	Frequent—copious, bright; may be early symptom.	None.	None.	None.
TEST MEAL.	Early reduction HCl and later, total absence; ferments decreased or absent; lactic acid present; impaired motility and absorption; abundant mucus, Oppler-Boas bacillus—yeast—possibly tumor shreds; coarse, undigested particles of food.	Hyperchlorhydria often; ferments normal or increased; no lactic acid or Oppler-Boas bacilli; food well digested. No mucus.	Decreased HCl (rarely increased, gastritis acida); later may be achylia. Much mucus.	Normal or variable. If HCl is decreased, the ferments are normal.	As in ulcer.
TUMOR	In 80 per cent.	In but one pro mille (from pyloric spasm, hypertrophy, cicatrix, etc.).	None.	None.	None (very rarely from pyloric spasm).
COLLATERAL SYMPTOMS	Cachexia—edema.	Absent.	Absent.	Absent. Nervous stigmata often.	Absent.
COURSE	Part of a year.	Longer; helped by treatment—aggravated by arsenic.	Chronic; helped by treatment—aggravated by arsenic.	Arsenic helps.	Like ulcer.

followed by a *swallow of water*, give the best results. Then Carlsbad water should be given, in small quantities and in sips; it consists of sodium sulphate 50, sodium bicarbonate 6 and sodium chloride 3 parts; it neutralizes the acid, aids intestinal digestion and cleanses the stomach (half an ounce of the salts in a pint of water, taken in quarters every ten minutes, the last dose one-half hour before meals). Alkalies operate also on the duodenum relaxing the pylorus. Roberts prescribed a lozenge

composed of magnesium carbonate grains $3\frac{1}{2}$, chalk grains $2\frac{1}{2}$, sodium chloride grain 1, which dissolved slowly in the mouth, increased the salivary flow and neutralized the excess of acid (*v. i.* 5 and 6).

5. Diet.—After a week of rectal feeding, eight to ten meals daily of predigested milk with soda should be given in small quantities. Cooked milk is more easily digested and curds less. The casein engages and neutralizes the acid. If peptonized milk is distasteful or the stomach is irritable it may be administered as a thin gruel. Potato purée, arrowroot, wheat-meal, baby foods, buttermilk, soups with meal and alkalies which precipitate the casein in finer form and ice-cream may also be given; but the milk diet, recommended by Cruveilhier is most efficacious. After the patient has been on the above-mentioned diet for ten days, soups, rice, sago, soft-boiled eggs, calves' brains and chicken may be administered. Slightly boiled or poached eggs are digested better than raw eggs and may be given with bread-crumbs from the inside of the loaf. Custards may be given. Tea and coffee should be forbidden. Strauss has found that sugar lessens the amount of acid. Cohnheim gives *olive oil* freely at all times to relieve spasm and inhibit hyperchlorhydria; it also protects the surface of the ulcer, promotes regurgitation of the alkaline duodenal contents into the stomach, increases nutrition and lessens constipation. After fifteen days Leube permits grated beef, and after about another week tender roast beef, chicken and pigeon, to which others would add lamb, macaroni, thymus gland and bread. After the fifth week roasts and soufflés may be given. The *Lenhartz diet*, which allows ice-cold milk and eggs, sugar and egg, raw minced beef and ham, rice, etc., is of higher caloric value; it is given even at once after hematemesis on the ground that increased nutrition promotes cicatrization. The practitioner will find the first method superior, but the writer withholds meat, meat extractives (in soup) and salt for months after the ulcer course.

6. Drugs.—*Bismuth* relieves pain and is taken most advantageously in olive oil, but it has no curative value. Fleiner, Kussmaul and others administer bismuth by means of the stomach-tube. Large doses may accumulate in the ulcer, the drug is expensive and the subnitrate may cause nitrite poisoning, methemoglobinemia, cyanosis, fever, salivation, vomiting, cramps, dyspnea, collapse and the black line on the gums, whence the subcarbonate is preferable. *Belladonna* relieves pain, relaxes the pylorus and lessens hyperacidity by its vagal action. *Silver* is used to wash the stomach before meals (Reichmann); it is readily precipitated and may produce nausea, diarrhea and even argyria.

7. Symptomatic Treatment.—*Pain* is best relieved by rectal alimentation, as is vomiting, which sometimes may necessitate the administration of bismuth, belladonna or minute doses of phenol. *Hemorrhage* is treated by absolute rest of the stomach, intestine, mind and body; morphine quiets peristalsis and mental excitement. Styptics, astringents, fomentations and ergot are contra-indicated. Syncope promotes spontaneous coagulation and stimulation should be avoided as long as possible. In severe hemorrhages adrenalin (gtt. x, 1 to 1000 solution) and horse serum are indicated; saline transfusion should be considered, but more

than one pint seldom should be given, for death has occurred from reaction and renewal of hemorrhage.

8. Surgical Treatment.—Most cases heal absolutely or improve greatly under proper medical treatment; this is especially true of fresh ulcers. Leube's last series (*v. s.*) of 627 ulcers, gave 2.5 per cent. mortality in the cases of hemorrhage; in the cases without hemorrhage, the mortality was 0.3 per cent., the complete cures 90 per cent., the improved cases 8.5 per cent. and the failures 1 per cent. Clairmont, with *gastro-enterostomy,* obtained complete cures in but 52 per cent. and improvement in 15 per cent.; a comparison with Leube's figures is suggested; the average surgical mortality is 6 per cent., though the figures of Robson, Mayo and Moynihan are close to 1 per cent.; 156 cases of sequential jejunal ulcer are recorded, and the gastric ulcer returned in 20 per cent. of gastro-enterostomy cases. Excision of callous ulcers entails a 5 per cent. death-rate, not a high figure in consideration of the frequency of carcinomatous degeneration. *Perigastric adhesions* or abscess and collections of pus beneath the diaphragm, necessitate operation. In cases of *perforation* if operation is undertaken within twelve hours, 71 per cent. recover.

Surgical interference is necessary in a limited number of cases. The writer's conclusions are that (i) fresh ulcers always and (ii) chronic ulcers in far over half the instances belong to internal medicine; (iii) half the "inveterate" cases respond to medical treatment; (iv) some of the cases with "chronic dyspepsia" and almost none of the cases with hemorrhage call for surgical interference; (v) the results of operation are disappointing; and (vi) no proof exists that ulcers heal better with gastro-enterostomy than with medical treatment.

CANCER OF THE STOMACH.

Cancer of the stomach constitutes 33 to 50 per cent. of all cancers and is found in 1 per cent. of autopsies.

Etiology.—This is as obscure as in other carcinomata. (*a*) *Age:* According to Lebert 1 per cent. occurs in persons under thirty years; 18 per cent. between thirty and forty; 61 per cent. between forty and sixty and 20 per cent. in those over sixty years of age. Rare congenital cases are recorded. Of late years cancer is becoming more frequent in relatively young persons. (*b*) *Heredity* is noted as an etiological factor in 6 to 17 per cent. and in Broca's series there were 15 instances among a family of 26. (*c*) Ulcer of the stomach is a frequent antecedent; C. Graham found ulcer in 60 per cent. of his cases—a higher figure than is found in general practice (4 per cent.). Trauma, external pressure, as in shoemakers, or internal ulceration, pulmonary tuberculosis and alcoholism are slightly predisposing factors.

Pathology.—(*a*) In *origin* it is an abnormal growth of the glandular epithelium (Waldeyer), which unlike mere adenomatous or papillomatous proliferations, extends beyond the mucosa into the other coats of the stomach and very often into other organs and tissues. Cancer may

develop in the normal or atrophied mucosa or in open or cicatrized ulcers. (*b*) It is almost *always primary* and is very rarely metastatic or extends to the stomach by contiguity; secondary growths are usually from mammary or pancreatic cancer; Welch collected 37 cases. It is almost invariably single.

Histology and Morphology.—A tumor, in the clinical sense, usually develops, although in some cases the cancer is infiltrative and flat (*carcinoma planum*). There are three main types: (*a*) *Carcinoma fibrosum* (scirrhus), the most frequent type, contains much poorly vascularized stroma and relatively little carcinomatous tissue; it is hard and, leading to much induration, is prone to stenose the pylorus, where it is especially frequent, or the cardia; if it is diffuse, it causes the stomach to shrink (carcinoma atrophicans); its onset is insidious; it grows slowly and ulcerates less than other types. (*b*) *Carcinoma medullare* is next in frequency; the cell elements preponderate; there is much soft, vascular tissue and the knife scrapes out much "cancer juice"; it may form cauliflower-like exuberances; early extensive degeneration, ulceration and metastases are common; this type includes the cylindrical-celled adenocarcinoma, epithelioma and the very vascular fungus forms. (*c*) *Carcinoma gelatinosum* (colloides) is least frequent; it produces nodular tumors or diffuse infiltrations, which contain colloid material; it inclines rather to extension by contiguity than to metastatic deposits.

The *gastric mucosa* shows focal wasting of the peptic- and acid-secreting epithelium and substitution by cylindrical epithelium, round cells and connective tissue. This *secondary atrophic gastritis* explains the chemism of gastric cancer, viz., diminution in the secretion of acid, rennet and pepsin; in some cases after surgical extirpation of the tumor, the secretion of acid and pepsin has returned.

Localization.—Cancer occurs (*a*) in the pylorus in about 60 per cent., (*b*) in the lesser curvature in 20 and (*c*) in the cardia in 10 per cent. of cases; *i. e.*, about 90 per cent. of cancers occur in these sites; this is explained by their greater exposure to mechanical insult, their relative fixation and the greater muscular activity of the pylorus and cardia. Many tumors, apparently pyloric in origin, begin in the lesser curvature, whence a palpable pyloric tumor often means one of considerable extension; this is an important surgical point.

Complications.—(*a*) *Ulceration* results from inadequate vascularization; it is usually central and does not depend on the size of the tumor. The tumor may ulcerate almost completely and Ziegler observed total disappearance, though metastases declared the nature of the scar. (*b*) *Hemorrhage* or slow seeping of blood is caused by ulceration, as is also (*c*) *perforation* (3 per cent.) into the peritoneum or by adhesions into adjacent abdominal organs or vessels, into the lung, pleura, pericardium or the abdominal wall, of which latter 25 cases are recorded (Taussig); perforation is often prevented by adhesions. (*d*) *Stenosis* of the cardia or pylorus is common, sometimes causing hypertrophy of the muscle fibers, which on section of the stomach appear as red lines. (*e*) *Dilatation* of the stomach follows pyloric obstruction; atrophy is observed in cardiac localization or in carcinoma atrophicans (diffuse cancer) and

hour-glass deformation may result when an annular scirrhus develops in the mid-stomach. (*f*) *Local extension* by contiguity occurs by the submucous lymphatics with early involvement along the lesser curvature; the peritoneum is often involved and less often there is extension to the portal vein, spleen, esophagus or colon. (*g*) *Metastases* occur in 50 per cent., in the liver, peritoneum, lymphatics, pancreas and lungs.

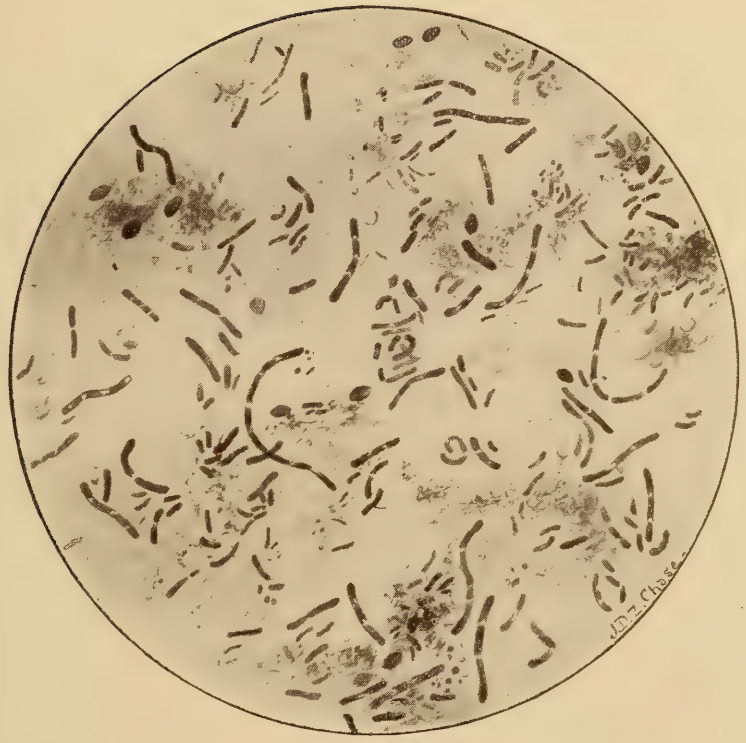
Symptoms.—General Clinical Picture.—Anorexia, distaste for meats, gastric oppression and eructations after eating, mark the usually insidious onset, and generally the dyspepsia develops in a patient previously free of gastric symptoms. Less frequently the disease begins acutely or follows closely on symptoms of gastric ulcer. Vomiting occurs, and then distress after eating solid food and mechanical or secretory insufficiency. There is gradual loss of weight, strength and endurance. Examination reveals anemia, cachexia, emaciation, lax skin, flabby muscles and often a tumor near the pylorus. A test meal shows imperfectly digested food and absence of free hydrochloric acid. The symptoms progress steadily or remissions occur, especially early in the disease. The ankles become puffy and the prostration is profound. Ulceration may relieve pyloric or cardiac stenosis, but gradually within a year after the first marked symptoms death results from the exhaustion due to pain or vomiting, local complications or intercurrent disease.

1. General Digestive Symptoms.—(*a*) The *tongue* is heavily coated, not being cleaned by mastication, and there is a bad taste in the mouth. The appetite is decreased or absent in 85 per cent., specially for meat and fats; the appetite may be normal when the motor power is good and cancer develops on an ulcer. (*b*) *Pain*, present in 90 per cent., is usually diffuse, dull, oppressive and less severe than in ulcer. Boas describes a grating sensation. It is increased several hours after eating or in the night, but is not wholly relieved by vomiting or dieting. Localized pain and tenderness are suggestive of circumscribed peritonitis. Pain may be reflected to the sternum, shoulders and acromion. (*c*) *Vomiting* (in 85 per cent.) is an obstinate and rather late symptom, resulting chiefly from pyloric stenosis. It appears late in the day or in the night, and may occur only on alternate days. There may be no vomiting in cancer of the curvatures, in diffuse cancerous infiltration involving the muscularis or in pyloric cancer when ulceration is marked. The frequently fetid vomitus contains much mucus, poorly digested food, little or no free hydrochloric acid, much lactic acid and often blood, which may be obvious or occult. Sudden vomiting may be the first symptom. Fecal vomiting usually indicates gastrocolic fistula (of which Voorhoeve in 1912 collected 104 cases); the stools in these cases may contain undigested meat (lientery)—"the patient vomiting into his own bowel." (*d*) *Hematemesis*, present in over 50 per cent. of cases, is due to parenchymatous oozing and is therefore moderate in amount; it is dark in color, as the blood stagnates in the stomach and is converted from oxyhemoglobin to hematin. Profuse bleeding occurs in 1 per cent. The author has seen 4 cases with fatal, profuse, bright hematemesis. (*e*) *Constipation* is present in 75 and diarrhea in 25 per cent.

of the cases; the writer has seen ichorous stools resulting from necrosis of the ileum.

2. Special Gastric Symptoms.—(*a*) *Tumor* is felt in 80 and seen in 40 per cent. of the cases, but is not an early symptom. It is most often missed in persons with wide thoraces and rigid abdominal muscles. It occupies the *right epigastric area,* as an ovoid, irregular mass, best determined when the knees are drawn up and the mouth is open, after the bowels have been flushed out and with the patient in several postures, as on the side, face, genupectoral position, etc.; in some cases the tumor may be felt lower or even on the left side. The tumor may be palpable one day and not the next. *Distention* of the stomach *by gas* brings forward tumors of the anterior wall and greater curvature, obliterates tumors of the posterior wall and lesser curvature and pushes downward and to the right tumors of the pylorus; motility on distention excludes adhesions. Distention must be moderate lest the stomach rupture. If the pylorus is incontinent from ulceration or infiltration, the gas escapes rapidly into the gut. A *visible peristaltic wave* or gastric rigidity (Cruveilhier, 1852) suggests pyloric obstruction. Percussion is subordinate to palpation and inspection, and auscultation occasionally elicits friction or a stenotic murmur over the abdominal aorta. *Respiratory excursion* is often present, though less than the excursion of the liver or spleen; if adherent to the omentum there is no excursion. If non-adherent tumors of the stomach, intestine and omentum are grasped at the height of inspiration their ascent during expiration can be prevented (Minkowski's expiratory fixation); on the other hand, tumors of the liver, spleen or stomach (if adherent to the liver) must ascend during expiration. (*b*) *Chemism:* (i) Bird (1842) and Van der Velden (1879) noted the *achlorhydria* of gastric cancer. Absence or decrease of hydrochloric acid is not pathognomonic, as it is also absent in nervous dyspepsia, fevers, cardiac, renal or hepatic disease, pernicious anemia, cachectic states, gastric atrophy, amyloid degeneration and toxic gastritis; hydrochloric acid is absent in 66 per cent. and decreased in 33 per cent. of carcinoma involving other organs than the stomach; the writer has seen many cases of rectal cancer in which digestive symptoms and achlorhydria led to an erroneous diagnosis of gastric cancer. In cancer it is due to the secondary *atrophic gastritis.* An Ewald test breakfast is withdrawn in an hour and tested as described under Chronic Gastritis. If there is no stagnation the total acidity ranges from 6 to 8; in stagnation it is higher from lactic and other organic acids. The absence of free acid is observed early, while the total acidity remains normal; later the total acidity is decreased. Free acid may persist, both in those cases which develop from peptic ulcer and in those without this antecedent. The gastric contents are often fetid. (ii) The *pepsin* and *rennet* are decreased in 66 per cent. of cases. (iii) *Lactic acid* is present in 75 per cent. of cases (Croner and Rütimeyer), and though also present in atony, gastritis and pyloric obstruction, 85 per cent. of cases in which it is found are gastric cancer (Schiff); it does not develop early. The factors involved in its formation depend on the reduced hydrochloric acid, motor insufficiency, stagnation, pyloric obstruction, slow absorption and probably poor digestion of

PLATE XV



Oppler-Boas Bacillus in Vomitus. (Musser.)

proteids; in the normal stomach it is absorbed as fast as formed (Sticker).[1] (*c*) *Microscopically* the thread-like, Gram-positive (Plate XV) *Oppler-Boas bacilli* appear in the test meal, feces, or in clumps, in the fenestræ of the stomach-tube. They produce lactic acid in the presence of proteids which act like ferments and originate from the tissues or the cancer; the bacilli point strongly to the existence of cancer, but may occur in ulcer and atrophic gastritis (with hypertrophy of the pyloric musculature). Small particles of tumor tissue or sarcinæ are rarely observed. *Occult hemorrhage, i. e.,* blood seen only with the microscope or detected chemically or spectroscopically, has the same significance as in gastric ulcer, but is more frequent (95 per cent.); pus in the fasting and washed stomach is very important. (*d*) *Motility:* Aspiration of the fasting stomach demonstrates stagnation in over three-fourths of the cases; disturbed motility may appear very early in the disease. The stomach is usually much dilated in patients whose appetites persist and little dilated in those with anorexia. If the stomach is empty after seven hours, good motility alone is proved, not good digestion. Every case of stagnation should arouse suspicion of carcinoma. The recent tryptophan reaction has no great or early clinical value; it depends upon the fact that, in gastric carcinoma, the ingested proteins are split into more elaborate products, amino-acids, than in normal digestion.

3. Other Symptoms and Complications.—(*a*) *Cachexia* is more than malnutrition; it is probably the result of toxins which break down the proteids. The *body weight* is usually greatly *reduced;* in exceptional cases it may temporarily increase, as in pyloric stenosis or from hope, as in a patient who gained 70 pounds after operation. The skin itches and is *anemic* or clay-colored, somewhat resembling the tint of pernicious anemia, though distinguished (i) by its later appearance, (ii) by its association with loss of weight, (iii) by the red cells being seldom lower than 2,000,000 (60 per cent. being between 2,000,000 and 4,000,000) and the hemoglobin seldom below 40 per cent.; and (iv) by the leukocytosis (see Pernicious Anemia). Schneyer described the *absence* of the *digestive leukocytosis;* it occurs in only half the cases. As in nephritis or chlorosis, the face may not show the real anemia; the wrinkling and wasting of the hands and face and the inelasticity of the skin may be far more significant. (*b*) The *urine* is decreased from poor absorption and vomiting, the physiological change in the acidity-curve after eating is absent, the urea is increased from increased toxemic catabolism, albuminuria is present in 35 per cent. of cases and indicanuria is common. (*c*) *Toxemic symptoms. Fever* of a low, irregular or intermittent type results in half the cases, from the cancer products, from septic absorption through the ulcer or from complications. *Coma* closely resembling coma diabeticum may result from the cancer toxins or the breaking down of the

[1] As a test meal Boas employs 1.5 per cent. oatmeal to 100 parts of water, given at night after lavage; the gastric contents are tested the next morning; the Uffelmann method is employed as modified by Strauss, for phosphates, alcohol and sugar also give the reaction; 5 c.c. of the gastric filtrate are added to 25 c.c. of ether and shaken in a burette; the lower 25 c.c. are drained away and 25 c.c. of distilled water are added to what remains in the burette (the etheral extract). Two drops of a 10 per cent. solution of ferric chloride and 20 c.c. of water are added; the blue solution turns to green if lactic acid is present.

proteids; acetonuria has been noted. Phlebitis and the less common multiple neuritis, tetany and amaurosis are toxemic. (*d*) *Metastases* or *extension by contiguity:* Enlargement of Virchow's supraclavicular gland may result from any abdominal carcinoma, but 50 per cent. are due to cancer of the stomach (Troisier); enlargements of the para- and peri-umbilical glands or of the umbilicus itself (Quénu and Longuet) are late symptoms. Tansini remarks that the abdomen, sunken in pyloric stenosis, becomes distended in peritoneal diffusion. Liver deposits may appear before any stomach symptoms. Icterus occurs in 5 per cent. Schnitzler remarked metastasis in Douglas's cul-de-sac, stenosing the rectum but without ulceration in it; Boas rates its frequency at 25 per cent. (*e*) *Ascites* results from the cachexia and, like the swelling of the ankles, appears late in the malady. Some cases are due to carcinomatous peritonitis, the fluid often showing blood and cancer cells, with mitoses (Dock), and others result from glands or tumor compressing the *porta hepatis*. (*f*) *Perforation* may occur (*v.* PATHOLOGY).

Cancer of the cardia presents symptoms like those of esophageal cancer (*q. v.*). Symptoms may be absent. The stomach is never distended. There may be tenderness under the xiphoid. The tumor when advanced may be palpated, as well as the perigastric lymph nodes.

Diagnosis.—Diagnosis is easy in the typical case. No single finding, as achlorhydria or even tumor, is pathognomonic; *only the grouping of symptoms and signs is final.* Single analyses of the stomach contents are often deceptive; they should be repeated frequently and considered in connection with other findings. Vomiting in middle or advanced life is suspicious when accompanied by emaciation, anemia, pruritus and insomnia. With absence of hydrochloric acid and the ferments, the findings of tumor, blood, lactic acid, Oppler-Boas bacilli and dilated stomach are positive. The diagnosis may be doubtful, even in the presence of pyloric tumor, emaciation and gastric dilatation, when hydrochloric acid is present; the tumor may then be due to peptic ulcer, hypertrophy of the pyloric musculature or pyloric spasm due to hyperchlorhydria; in the last instance the tumor is alternately hard and then soft. The diagnosis between beginning cancer, nervous dyspepsia and atrophic gastritis must be reserved for a time. *Atrophic gastritis* is more chronic, often lasting years without emaciation. Youth and adolescence do not exclude cancer; in the young, gastric cancer is more rapid in evolution, and hydrops and peritonitis are more common. Abdominal atheroma may closely simulate cancer. *Ulcer* (*q. v.*). Early diagnosis may be possible when, in an ulcer cure, the patient still vomits and occult blood persists. Achylia and stagnation, in the fasting stomach, are particularly suggestive. The meiostagmin, isohemolytic and complement-deviation reactions may prove helpful.

X-*ray* plates and fluoroscopy develop many decisive results—more often late than early in the disease. Holzknecht holds that residual bismuth after six hours with an achylia indicates cancer if the second bismuth meal shows a normally shaped stomach (thereby excluding atony) and if the first bismuth meal has reached the splenic flexure. Ragged edges may appear or indentations, like finger prints. Bismuth

PLATE XVI

Fig. 1

Fig. 2

Fig. 1.—Carcinoma of Body of Stomach. Rigid Patency of Pylorus. Increased Motility of the Colon, Head of Bismuth Column in Six Hours being in the Descending Colon. Fluoroscopic examination of this case shows duodenum filled with bismuth as far as the duodenojejunal angle within ten minutes after the ingestion of the bismuth meal. (Radiologist, Dr. Leaming.)

Fig. 2.—Carcinoma of the Lesser Curvature, not Involving the Pylorus. (Radiologist, Dr. Le Wald.)

Fig. 3

Carcinoma of Pyloric Half of the Stomach. (Radiologist, Dr. Leaming.)

Fig. 4

Carcinoma of the Pars Media.

(From Lockwood's Diseases of the Stomach.)

flowing early into the duodenum denotes pyloric insufficiency. The narrowing in ulcer is short, linear and sharply contoured, whereas in cancer it is longer, and its outlines more vague. The hook-shaped picture means an operable carcinoma, and the horn-shaped picture indicates inoperability. Gastrocolic fistula, adhesions, hour-glass deformity, perforation or metastases may be apparent.

The so-called latent, dyspeptic, gastralgic, anasarcous and ascitic forms of cancer, and those causing early metastases (for example, in the liver, lungs or bones) and intestinal obstruction, show in what direction error may occur. In some cases the general symptoms overshadow the local, as anemia, fever or inexplicable emaciation; in other instances, the local symptoms may mislead, as finding hypersecretion, or when the trouble begins acutely, as in one-quarter of cancer cases (Boas).

Differentiation.—(*a*) *Pancreatic* tumors (*q. v.*) are deep, fixed, immobile and disappear on gaseous inflation of the stomach; the normal pancreas is sometimes palpable in meagre subjects. (*b*) *Cancer of the gall-bladder* has respiratory excursion but neither expiratory fixation nor lateral movement; the digestive symptoms, chemism and ectasia of gastric cancer are lacking. (*c*) In *duodenal* carcinoma the gastric chemism is lacking; occult blood and icterus are suggestive. (*d*) *Cancer of the colon* is usually distinguished by the intestinal signs and absence of stomach symptoms. (*e*) Enlarged *aortic glands* are distinguished by lack of altered chemism, and abdominal *aneurysm* by presence of expansile pulsation. (*f*) *Omental* and peritoneal tumors are more diffuse. (*g*) Foreign bodies (hair tumors), subserous lipoma, fibromyoma (62 cases reported), myxoma and other benign gastric tumors, and palpable round ulcers, are much rarer sources of error. Of sarcoma 171 cases are reported (Grosset, 1912); perforation occurs in 10 per cent.; 52 were successfully operated on.

Prognosis.—The average duration is one year (three months to three years). Death results from exhaustion by pain or vomiting, hemorrhage, gangrene, perforation, portal pressure, intercurrent pneumonia, nephritis and tuberculosis.

Treatment.—1. Medical or Palliative.—(*a*) Condurango bark seems to increase the appetite. (*b*) *Vomiting* is treated as in gastritis, dilatation and ulcer, but with less success; *lavage* is indicated in pyloric stenosis; boric or salicylic acid may be added for fermentation; narcotics by rectum, rectal feeding, champagne and carbolic acid, as outlined before, are also useful. (*c*) Hemorrhage is rarely profuse and usually subsides on rectal feeding; iron, ergot and lead should be avoided. (*d*) Achylia is seldom helped by hydrochloric acid or pepsin. (*e*) *Pain* necessitates fomentations, lavage, chloroform (3 to 5 drops) with ice, and atropine or opium by rectum. (*f*) For *constipation*, cathartics are inferior to warm *colonic flushings* or salts, glycerin or olive oil by rectum. (*g*) *Diarrhea* is lessened by lavage; when due to pyloric insufficiency, salol, beta-naphtol (āā gr. x) and opiates are indicated. (*h*) Stenosis of the cardia may yield to dilatation. (*i*) Motor insufficiency necessitates lavage, which should be practised before supper. (*j*) The diet should be soft and small in bulk. Meats and fats are digested with difficulty. Vege-

tables, cereals, small quantities of milk, koumyss, honey, pastries, tea, coffee, diluted wine, peptones and baby foods may be tolerated.

2. Surgical Treatment.—Cases having good surgical prospects are few, but operation only can cure. When a tumor is found it usually has reached the pylorus from the lesser curvature, yet tumor does not contra-indicate operation (*v.* X-rays). For resection cases should reach the surgeon before the tumor is palpable, a condition possible only in private practice, for hospital cases are always advanced. Metastases, peritonitis, extreme anemia, nephritis and other visceral lesions and glandular invasion are contra-indications to operation. *Pylorectomy* results in an immediate mortality of under 30 per cent., half the death-rate in the time of Billroth, who did the first operation (1878). Recurrence has been noted after five years. Leriche reports one case well after 16, and Kocher, after 19 years. The Mayos report resections with a mortality of 10 per cent.; in one group of 25 cases only one case died; 38 per cent. of their cases were living three years and 25 per cent. were well five years after resection. *Gastrostomy* in cardiac cancer and *gastro-enterostomy* in pyloric obstruction seldom prolong life.

HEMATEMESIS.

Definition.—Hematemesis is not synonymous with gastric hemorrhage; it is the vomiting of blood, which may issue from the stomach or may reach it from the gums, nose, larynx, lungs, esophagus, or even the intestine. Gastrorrhagia designates profuse hemorrhage from the stomach. It may be arterial, venous or capillary.

Etiology.—(*a*) Various forms of *ulceration*, as simple ulcer, carcinomatous, tuberculous, diphtheritic, typhoid, syphilitic, uremic or phlegmonous ulcers, hemorrhagic erosions, and perforations into the stomach from without, as gall-stones, or aneurysm dissecting down from the arch of the aorta are etiological factors. Parrot described superficial ulcers in the newborn. Dieulafoy describes a pneumococcic hemorrhagic gastritis. Chiari, Murchison and Dieulafoy described small smple ulcers causing profuse, often repeated and fatal hemorrhage; they dievelop in perfect health, with no previous stomach symptoms; they are easily overlooked at operation or autopsy because they involve only the mucosa or perhaps some of the muscularis. (*b*) *Trauma*, such as external violence, or internal trauma, as from foreign bodies, thermal or chemical injury, and violent vomiting, as sea-sickness and hyperemesis, may be causal. (*c*) The cause may be *vascular*, such as amyloid degeneration which sometimes involves the stomach and bowels alone, aneurysm, embolism, vicarious congestion from suppressed menstruation, passive congestion of cardiac or hepatic origin; and esophageal varix (when due to cirrhosis of the liver, there is in 33 per cent. of cases no other evidence of cirrhosis); Welch and Powell found varices in the floor of peptic ulcers; malarial deposits in the liver; acute pylethrombosis, causing profuse hemorrhage; and parenchymatous oozing, where no postmortem changes are found (Hale White reports 36 cases of what he

terms gastrotaxis). (*d*) *Nervous* causes are injuries to the central nervous system, vasomotor influences, hysteria in which the blood is small in amount and occurs usually in the morning vomit, tabetic crises, etc. (*e*) *Acute infections* may be etiological factors, as malaria, hemorrhagic exanthemata, yellow fever, sepsis, appendicitis and genito-urinary sepsis; Busse (1905) collected 96 cases following operations; they result from direct embolism (of which 35 per cent. were in the mesentery or omentum) or retrograde embolism; the hemorrhage occurred in the stomach or bowels; 55 per cent. died. (*f*) *Blood diseases,* as pernicious anemia, leukemia, hemophilia, purpura, scurvy and splenic anemia, may be causal. (*g*) *Poisons,* which early erode and later ulcerate, as acids and caustics, those causing fatty degeneration of the vessels, as arsenic and phosphorus, and uremia or cholemia, may cause hematemesis.

Of all causes, round ulcer and liver cirrhosis are the first suggested.

Symptoms.—The symptoms are (*a*) those of the *antecedent disease.* (*b*) In some cases no blood may be vomited, and the incident remains undiscovered, unless the stools are examined. In *vomitus cruentus* gastric uneasiness is common. The amount vomited varies from microscopic quantities to those sufficiently large to cause death. "*Occult hemorrhage*" has been particularly emphasized by Boas; the diet for days previously should contain no meat, blood, iron or chlorophyl, but solely eggs, fruit, starch or fat. In profuse gastrorrhagia the blood is bright red, if it remains in the stomach only a short while, as in most cases of peptic ulcer. In smaller hemorrhages it is black or rust-colored, as in cancer, uremia, cholemia and various poisonings; rust-colored blood may become bright on the edges or surface when it has stood for some time. Blood may issue in the fluid form, or as clots, in which case it is suggestive of valvular disease, vascular disease or the dangerous hematemesis of cirrhosis; the author has seen large clots in typhoid and gastric carcinoma. (*c*) *Melena* may render the stools black, tarry and offensive. (*d*) *Systemic signs.* A gradual or sudden intense *anemia* may be noted, with the usual signs of secondary anemia (*q. v.*), vertigo, syncope, rapid pulse and some dyspnea, and later, amaurosis, slight fever, dicrotic pulse, hemic murmurs, moderate edema, albuminuria and sometimes hemiplegia or convulsions.

Diagnosis.—Differentiation from hemoptysis (*q. v.*), from the swallowing of blood from the nipples in nurslings or from the vagina in the newborn, fracture of the skull, esophageal or throat disease and from simulation of blood in the stools from ingestion of bismuth, coffee, tea, claret, iron or blueberries, does not need particular elaboration, as the microscopic, chemical and spectroscopic examination determines doubtful cases. *Treatment* (*v.* Ulcer).

NEUROSES OF THE STOMACH.

Neuroses of the stomach occur as a part of a neurosis, as isolated nervous symptoms, to be considered under neurasthenia or as reflex affections.

I. Secretory Neuroses.

These affections have been described especially by Reichmann, Rossbach, Sahli and Riegel.

1. Hyperchlorhydria.—A *qualitative* anomaly of gastric secretion in which there is an excessive secretion of hydrochloric acid during digestion; it is not solely a neurosis, though conveniently classified as such.

Etiology.—(*a*) *General* etiological factors: (i) It is most frequent in youth and middle age, the better situated classes, Polish Jews and brain-workers; (ii) a neurotic or psychopathic tendency is a predisposing factor, as is also (iii) chlorosis. (*b*) *Local* causes are (i) rapid eating, cold drinks and condiments; (ii) tobacco, alcohol and coffee; (iii) gall-stones, constipation and appendicitis.

Symptoms.—(*a*) *Pain*, the chief symptom, varies from a sense of discomfort in mild cases to extreme gastralgia in severe cases; it is most marked in ulcer, pyloric stenosis and dietetic vices. It develops at the height of gastric digestion—*i. e.*, one or two hours after eating—and is due to the excess of free hydrochloric acid; the more food, especially meat, ingested the later is the pain, for the food engages the free acid longer. Carbohydrates cause increased pain as they do not engage the acid and amylolysis is decreased. The patient often feels a cramping or *spasm of the pylorus*. Pain lasts one-half to several hours. It is *relieved by vomiting* and usually also by ingestion of more food or of alkalies. (*b*) *Belching and pyrosis* are frequent and the eructated hyperacid fluid causes a burning in the esophagus. (*c*) *Vomiting* of a burning sour fluid is not frequent, save in severe cases. (*d*) The appetite is usually good, and occasionally ravenous. (*e*) *Tenderness* over the stomach and *gastric hyperesthesia* are *usually diffuse*, moderate in degree, coincident with the pain and associated with some epigastric distention. (*f*) *Stomach tests* show a total acidity of 100, even 160; the proteids are well subdivided, amylolysis delayed, motility and absorption normal or increased in uncomplicated cases and no lactic acid, no fermentation and no gas are present. (*g*) *Constipation* and *headache* are common; the gums may retract; the *urine chlorides are decreased*, the acidity lessened during digestion and an alkaline reaction and phosphaturia are present.

Diagnosis.—The diagnosis is determined by hyperchlorhydria in the vomitus and test meal, by the relief of pain by vomiting or ingestion of more food or alkalies and by negative physical findings.

Differentiation.—(*a*) *Ulcer* is marked by localized tenderness, hematemesis or occult bleeding and more symptomatic regularity. (*b*) *Gastritis* is characterized by mucous formation and decreased secretion. (*c*) *Gastrosuccorrhea* or *continuous secretion* (*v. i.*). (*d*) *Gastrectasia* and *gastroptosis* are excluded by the *x*-rays. (*e*) In *gall-stones* pain has no relation to eating, occurs at longer, irregular intervals and is associated with tenderness of the gall-bladder.

Course and Prognosis.—The disease begins slowly and may disappear, to recur after days to months, from irregular living, nervous strain, excesses, diet or without obvious cause. The nutrition is fairly well

maintained. The outlook is good if early treatment is instituted; complications modify the prognosis.

Treatment.—1. PROPHYLAXIS.—Nervous factors, smoking, haste in eating, chlorosis, etc., should receive appropriate treatment.

2. DIET.—(*a*) *Proteids* are most beneficial, for they take up the largest amount of hydrochloric acid, without increasing the HCl; veal, beefsteak, mutton and ham absorb twice as much hydrochloric acid as other proteids; fish fat and salt are avoided; milk and some kinds of cheese may be given. (*b*) *Carbohydrates* are restricted, because the early amylolytic digestion by the ptyalin is arrested; a slice of stale bread, biscuits, toast, zwieback or purées of aleuronat meal may be tried; Strauss allows six ounces of 2 per cent. grape-sugar solution, unless there is motor insufficiency. Potatoes, salads and most uncooked vegetables should be interdicted. (*c*) *Fats* may be given in limited quantities, including small amounts of cream or unsalted butter (one ounce); two drams of olive oil before meals inhibit the secretion of acid. (*d*) *Condiments*, mustard, pepper, vinegar, spices, lemon, horseradish, coffee, alcohol, cold foods and drinks should be forbidden. Cocoa or milk may be given. (*e*) *Thorough mastication* and *small meals*, at frequent intervals, are most efficacious.

3. MEDICATION.—(*a*) Sodium bicarbonate ʒj, in a starch wafer with but little water, at the height of digestion neutralizes the hyperchlorhydria; alkalies not only act chemically but inhibit secretion by action on the glandular cells. (*b*) Extract of *belladonna*, $\frac{1}{4}$ to $\frac{1}{2}$ grain, given at meal-time, lessens secretion and mitigates pain; opiates should be avoided. Nux vomica is valuable. (*c*) *Lavage with silver nitrate* (1 to 1000) solution is useful in obstinate types.

2. Gastrosuccorrhea (Hypersecretion, Continuous Secretion).—This is a *quantitative* perversion of gastric secretion, in which secretion exceeds the needs of digestion; the lightest forms occur only when food is ingested (alimentary form); the severest occur without ingestion of food. Some gastric juice may be found in the normal stomach, due to secretion initiated by swallowed saliva or mucus, but over one ounce is rarely physiological. (*a*) The *intermittent form* occurs in attacks which last a day or two, is often called acute or nervous dyspepsia and has about the same etiology as hyperchlorhydria; it has been observed during tabetic crises. *Colic-like pain* develops, *usually at night*, followed by sour *eructations* and *repeated vomiting*, first of food, then of three ounces or more of yellowish-green fluid which contains hydrochloric acid and ferments; there is seldom blood. During the attack the pulse is small and rapid, the skin clammy, the urine scanty and alkaline, and headache is frequent. Thirst is great and drinking may give temporary relief, but is followed by increased vomiting. Between attacks the acidity is normal and the general health good; occasionally there is a constant sense of gastric discomfort. (*b*) The second form is *continuous gastrosuccorrhea*. Its etiology in most instances is pyloric obstruction or pyloric spasm from ulcer, gall-bladder disease or appendicitis.

Symptoms.—(*a*) *Pain is more marked* than in hyperchlorhydria, develops later after eating and very often from 11 P.M. to 2 A.M.;

it may occur before meals, for the gastric secretion is constant, or in severe cases, day and night; it is relieved by eating and vomiting. (*b*) *Vomiting* closely follows the pain and may occur at its height or during the night; the quantity of vomitus is large when there is dilatation (a frequent complication or a common cause of this type). It contains hydrochloric acid, ferments and little mucus; vomiting may be absent in light cases. (*c*) *Other digestive symptoms.* The *tongue* is often *red and clean,* sometimes coated. The *teeth* are often *carious.* The appetite is usually good, sometimes ravenous or in advanced cases poor. The *marked thirst* sometimes suggests diabetes. The bowels are sluggish. (*d*) The *test meal and lavage* show a total acidity up to 100 and free acid to 60. After a test dinner the fluid separates into three layers—the upper foamy, the middle consisting of yellowish fluid and the lower one of sediment of undigested carbohydrates; the proteids are found converted into peptones. When the stomach is washed out thoroughly at night, examination on the next morning of the fasting stomach reveals up to ten ounces (or even a pint) of hydrochloric acid and ferments, *i. e., secretion without the stimulus of food;* if the fasting stomach is washed out two or three hours later, gastric juice is again found. (*e*) The urine is the same as in hyperchlorhydria. Old cases show marked *emaciation,* even cachexia, lax muscles and dry, inelastic skin.

Course and Prognosis.—The onset is slow and the course protracted. Death from exhaustion may result in untreated cases. *Complications* are frequent, especially (*a*) dilatation, for the stomach is never empty; the hydrochloric acid does not inhibit the growth of sarcinæ or yeast and gases may generate. The dilatation is most often secondary. Dilatation may also result from (*b*) *pyloric spasm;* in health the pylorus relaxes intermittently and allows the acid chyme to reach the duodenum; the pylorus is reflexly contracted by the acid fluid in the duodenum until this is neutralized, when it again relaxes. When the secretion is continuous, the passage of acid into the duodenum constantly contracts the pylorus, which may be felt as a tumor. In this case cancer may be simulated, especially when there is emaciation or blood in the stomach. (*c*) Tetany is a rare complication.

Diagnosis.—The diagnostic features are pain at night, vomiting, and thirst, which are more common than in hyperchlorhydria; and continuous secretion after lavage and during fasting.

Treatment.—Treatment is (*a*) that of hyperchlorhydria as to prophylaxis, diet, mastication and stimulants. (*b*) *Rectal feeding* is a valuable accessory; water by rectum quenches the raging thirst. (*c*) *Oral feeding* is allowed at long intervals; overloading the stomach with fluids is carefully avoided. (*d*) Lavage with silver nitrate (1 to 1000) relieves pain and hypersecretion. (*e*) *Olive oil, alkalies* and belladonna are indicated as in hyperchlorhydria. (*f*) Gastro-enterostomy may be indicated by pyloric obstruction, never by hypersecretion *per se.*

3. Hyposecretion.—Hypacidity (hypochlorhydria) and anacidity (achlorhydria) refer respectively to reduction or absence of hydrochloric acid, which may occur from nervous or organic causes. When the

ferments and acid are absent, achylia gastrica nervosa is a more appropriate term; the condition may be transient or enduring. Suppressed secretion may occur in hysteria or tabetic crises.

II. Motor Neuroses of the Stomach.

1. Irritative Type.—(*a*) Hyperkinesis or *hypermotility* hurries the food into the intestine in three to five hours or less. It exists as a separate neurosis, or in the hasty digestion of hyperchlorhydria and in achylia, that the intestines may earlier digest the unaltered food. (*b*) *Peristaltic unrest* or *tormina ventriculi nervosa* occurs particularly in neurasthenia and organic pyloric stenosis. In some cases the unrest may be wholly subjective; in others, both neurotic and organic, the peristaltic movements from left to right may be seen, as well as antiperistalsis in the opposite direction. Treatment is that of the underlying neurosis. (*c*) *Pyloric spasm* occurs very seldom as an independent neurosis; it occurs usually in connection with ulcer, hyperacidity, hypersecretion, erosions, chronic stenosing gastritis, gall-stones, cancer of the stomach, poor mastication of food and drinking of cold fluids. (*d*) *Spasm of the cardia* occurs in organic disease or neuroses of the esophagus and stomach; it is the cause of idiopathic dilatation of the esophagus and the spasm in tetanus and hydrophobia; it makes vomiting difficult. The treatment is causal. (*e*) *Nervous eructation* was first described by Dejardin (1814). It occurs especially in sexual neuroses. The odorless *ructus* results from swallowing of air (aërophagia), which constitutes a most intractable habit. Bouveret thinks air is forced down by pharyngeal contractions, either unconsciously or during false eructations, in which the patient, in attempting to raise gas, swallows air; such large quantities could never generate in the stomach; Bardet estimated that one patient raised twenty quarts in an afternoon. Coincident cardiac and pyloric spasm causes distention (pneumatosis). Treatment lies in the use of the sound, suggestive therapy, the difficult task of breaking the habit and asafetida which renders the ructus offensive. (*f*) *Nervous vomiting* occurs (i) only in the neuroses, as neurasthenia, migraine and exophthalmic goitre; but under this caption we may bring for differential purposes (ii) cerebrospinal conditions, as compression of the brain, meningitis, tumor, tabes, etc., or toxic conditions, as uremia or cholemia. (iii) Most cases are *reflex* from the nose, as from disagreeable odors; the respiratory tract; pelvic or abdominal conditions, as pregnancy, menstruation, calculi, etc. The symptoms may present many incompatible elements; thus the patient may vomit without nausea, almost without effort like an eructation; it often occurs without pain or reference to the fulness or emptiness of the stomach; even though it is frequent, the nutrition may be remarkably good, the tongue clean and the appetite good. Periodic or *cyclic vomiting*, described by Gruère (1838), has some resemblance to tabetic crises; Edsall considers it an acidosis, for which ʒ ss–ij of sodium bicarbonate is indicated. In the 10 fatal cases on record there was fatty degeneration of the liver and sometimes appendicitis.

Robert's conclusions are as follows:

1. Vomiting of a chronic type, following a gradual epigastralgia, from one-half to three hours after eating, is attributable to ulcer.

2. Chronic vomiting, soon after food ingestion is stenosis of the cardia, nervous abnormality, cerebral lesion or acute gastritis.

3. Copious vomiting ten or more hours after eating indicates muscular insufficiency; frequent repetition indicates stenosis.

4. Vomiting in the night occurs in cholelithiasis, hypersecretion, muscular insufficiency and nervous abnormality.

5. Vomiting attempts when the stomach is empty indicate a cause other than gastric, *e. g.*, reflex cause, toxemic, cerebral or nervous.

6. Morning nausea and retching indicate pregnancy, alcoholism, pharyngitis, nephritis or a nervous abnormality.

7. Periodic vomiting of clear gastric juice in considerable amounts indicates a secretory neurosis or ulcer.

8. Vomiting as a sequel of headache, accompanied by severe nausea, but no gastric symptoms, characterizes migraine.

9. Sudden vomiting with tinnitus, deafness and vertigo is attributable to disturbances of pressure in the internal or middle ear.

10. Periodic sudden vomiting, with gastric pain and nausea, retraction of the abdomen, obstinate constipation during, but not preceding, the attack and freedom from abdominal tenderness are suggestive of the spinal crises, nervous vomiting and lead colic.

11. Periodic attacks of vomiting with colic, constipation and tympany suggest chronic intestinal stenosis.

2. Depressive Motor Neuroses (Lessened Motility).—There are three types: (*a*) *Atony*, which has been considered under dilatation; (*b*) *pyloric insufficiency*, which is seldom a neurotic condition, as compared with its frequency in cancer, ulcer and duodenal obstruction; inflating the stomach with carbon dioxide, it rapidly passes into the intestine with a gurgling gush. (*c*) *Insufficiency of the cardia* causes *regurgitation* of food and fluid; rumination (*merycismus*) is regurgitation in which the food is again chewed and reswallowed. These are generally involuntary processes, but some neurotic males, psychopaths or idiots can regurgitate at will. The affection is hereditary or imitative. Its mechanism is congenital weakness of the cardia, stimulation of the vagus or aspiration into the esophagus by the inspiratory act. It should be treated by giving small meals at frequent intervals; suggestive measures are indicated.

III. Sensory Neuroses of the Stomach.

1. Hyperesthesia.—A sensation of burning or weight usually occurs when the stomach is full, but at times also when it is empty. Carbohydrates, fats or spices may induce the condition. Silver nitrate, gr. $\frac{1}{4}$ to $\frac{1}{2}$, or phenol, gr. $\frac{1}{4}$ to j, given on the empty stomach, often affords relief.

2. Gastralgia.—Gastralgia (or cardialgia, a poor but widely used term) does not strictly include the pain of ulcer, hyperacidity or hypersecretion. An etiological classification is as follows: (i) Cerebrospinal

causes, including various brain, cord (tabes), peripheral and functional nervous affections. (ii) Constitutional causes, including chlorosis and debility. (iii) Toxic causes (gout, nicotine and constipation). (iv) Reflex causes, from the genito-urinary and alimentary tracts, splanchnoptosis, uterine or ovarian disease or masturbation. Fliess holds that dysmenorrhea and gastralgia may arise from disease of the lower nasal turbinates. Most cases occur in women between fifteen and forty-five years of age.

Symptoms.—Severe gastric pain begins suddenly, often at night, lasts for a few minutes to an hour or so and subsides, usually to recur after a variable interval. The pain may radiate widely. It bears no relation to eating. Diffuse superficial hyperesthesia over the stomach is not infrequent, but deeper pressure usually affords some relief. Vomiting, hiccough, hunger, headache, depression, polyuria, clammy sweats, rapid pulse or even collapse may attend the seizure.

Diagnosis.—A diagnosis is made only by exclusion and usually after the seizure. Rheumatic myalgia is more constant. A tabetic crisis is at once recognized by the Argyll-Robertson pupil and the absent knee-jerks. Intercostal neuralgia gives Valleix's three tender points. In ulcer or circumscribed peritonitis the pain is associated with localized tenderness. Hyperacidity and hypersecretion should be excluded. A sharp thrust over the gall-bladder during deep inspiration nearly always elicits well-localized tenderness in biliary colic.

Treatment.—Treatment is (*a*) etiological—neurasthenia, anemia or constipation. Fowler's solution ℿij before meals is excellent. (*b*) The attack. Morphine hypodermically affords the only certain immediate relief, but in these very cases may establish the habit. If given, it should be exhibited by mouth without the patient's knowledge. Heat and sinapisms may relieve or aggravate the attack. Silver, phenol, gelsemium, belladonna and Hoffmann's anodyne often give relief.

3. Disturbances in the Sense of Hunger and Appetite.—Loss of appetite (*anorexia*) may be nervous, though more commonly gastric, tuberculous or toxic. Anorexia nervosa may be absolute and cause profound emaciation. *Parorexia* is a perversion, in which the patient craves for unusual foods. In *polyphagia* the appetite is enormous, but is capable of satiation; in boulimia (*hyperorexia*) the polyphagia is paroxysmal, sometimes with vertigo, palpitation, etc.; they are most frequent in hyperchlorhydria, hypersecretion and hypermotility, but also occur in diabetes, brain disease, intestinal parasites or from quinine, orexin or even opium. *Acoria* is loss of the sense of satiation. These anomalies probably result from changes in the bulbar hunger centre.

IV. Mixed Neuroses of the Stomach.

Dyspepsia nervosa or pseudodyspepsia may occur as a species of local neurosis, as a reflex from other organs, but much oftener a part of the neurasthenic symptom-complex. Leube's contention that digestion itself is normal must be modified, as the hydrochloric acid may be increased, normal or decreased, but perhaps in the majority of cases the

gastric functions are nearly normal. Two main groups of symptoms prevail: (*a*) the *centric*, neurasthenic or hysteric apathy, irritability and vertigo; (*b*) the *gastric* eructations, variations in appetite, oppression and diffuse tenderness, which are probably also cerebral in origin. The symptoms vary greatly in degree, often change their form with rapidity and the general nutrition is good. Organic disease must be carefully excluded by repeated examinations of the secretions and motility of the stomach. The treatment is rest, correction of worry and strain, self-control and suggestion.

DISEASES OF THE INTESTINES.

ACUTE ENTERITIS.

It is difficult to draw a close distinction between acute catarrhal enteritis, intestinal dyspepsia and simple diarrhea. Catarrhal enteritis is the most common intestinal disease.

Etiology.—1. Primary Forms.—(*a*) Errors in *diet* and ingestion of spoiled food containing ptomaines, unripe fruit and impure water are common causes. (*b*) Disturbance in intestinal secretion possibly occasions acute enteritis. (*c*) Cold, trauma, foreign bodies, fecal retention and toxins eliminated from the blood may initiate inflammation. (*d*) Some cases are *infectious*. Bacteria are an important factor; one-third of the weight of the normal stool is caused by bacteria, of which 48 species are normally present; inflammation may arise from pathogenic organisms or virulence attained by some inhabitant of the intestine, *e. g.*, the Bac. coli and Gärtner's Bacillus enteritidis. In a milk diet the Bacterium lactis aërogenes and in a meat diet the Proteus vulgaris may be possible factors.

2. Symptomatic Forms.—(*a*) Enteritis is constant in some infections, as in typhoid; it is frequent in others, as in sepsis or influenza; and rare in others, as in measles, scarlatina, etc. (*b*) General diseases, as nephritis; burns; intestinal parasites, ulcers or tumors; and metallic or other poisons are etiological factors; (*f*) stasis is rarely by itself a potent cause.

Pathology.—Redness, swelling, increased mucus secretion, parenchymatous degeneration and interstitial infiltration, in the few cases coming to autopsy, may not be conspicuous, as they regress after death. Follicular swelling or even ulceration, ecchymoses, erosions or swelling of the mesenteric glands may be observed.

Symptoms.—The chief symptom is *diarrhea*, due to increased peristalsis, secretion or exudation. The *stools* number two to fifteen daily and contain mucus; they are brownish-yellow and moderately frequent, or colorless, odorless and alkaline or neutral, and very frequent; they contain triple phosphates, calcium phosphate and oxalate crystals, sometimes cholesterin, Charcot-Leyden crystals, and few leukocytes. Thirst, colic pains, borborygmus, visible peristalsis, tympany and gurgling are frequent. Febrile forms with acute splenic tumor are recorded, which may at first resemble typhoid. Albuminuria, cylindruria

or even acute nephritis may develop exceptionally; the urine is scanty in proportion to the amount of fluid lost by the bowel; indican, ethereal sulphates and acetone may be present. In some few cases, gastritis, herpes or muscle and joint pains occur. Collapse is rare in adults, though there may be a close resemblance to cholera. In the vast majority of cases convalescence is established in a few days.

Diagnosis.—(*a*) *Duodenitis* can seldom be diagnosticated, though suggested by pain, tenderness over the duodenum and catarrhal icterus. (*b*) *Jejunoileitis* seldom exists alone; diarrhea is absent if the colon is not affected; the stools contain intimately mixed mucus, as small hyaline globules under the microscope. (*c*) In *enterocolitis* the bile is decomposed below the small intestine; when it is present in the stools it signifies increased peristalsis which moves the bile along before it can be disintegrated and indicates acute enterocolitis; bile may be found only in the globules of mucus. Many *undigested food particles* (lientery) are rather significant if fever, biliary or pancreatic obstruction and gastric atrophy are excluded; they consist of undigested meat, starch granules (which are more significant than muscle fibers) and fat particles. (*d*) *Colitis* is characterized by the absence of bile and lientery, by thin stools and by more or less mucus, obtained especially on washing out the bowel immediately after a movement. (*e*) *Proctitis* is marked by tenesmus, dysuria, evacuation of mucus alone and bloody mucus covering but not mixed with the solid stools; it is also diagnosticated by digital or proctoscopic examination.

Treatment.—(*a*) In the early stage *evacuation* of the causal irritant indicates fractional doses of calomel, followed by castor oil; stronger evacuants irritate or inflame the intestine. Colonic flushings with pure water or injections of olive oil may be added. (*b*) *Absolute rest* in bed is always indicated. (*c*) The *diet* should be restricted to barley-water, or no food should be given for a day or two. (*d*) *Heat* is applied to the abdomen. (*e*) A dram each of *paregoric* and *bismuth* should be given after each bowel movement until the number is controlled.

If bismuth is given in large amounts, the subcarbonate is preferable (*v.* page 518).

℞—Phenylis salicylatis (salol)	gr. xx	ʒj
Sodii bicarbonatis	ʒss	ʒj
Cretæ preparatæ	ʒss	ʒiss
Bismuthi subnitratis	ʒj	ʒj
M. et divide in pulveres x.	For child;	adult.
S.—One powder in milk or thin arrow-root solution every three hours.		

Lack of tone is relieved by gambir (catechu), colic by belladonna and flatulency by ammonia and ginger:

℞—Tincturæ gambir compositæ	ʒj
Fluidextracti belladonnæ	gtt. xij
Spiritus ammoniæ aromatici	ʒij
Syrupi zingiberis	ʒss
Misturæ cretæ q. s. ad.	ʒiv
M. et S.—A tablespoonful every two or three hours.	

Betanaphtol gr. x p. c. in capsules, or tannigen gr. xv every three hours, are seldom indicated. (*f*) No laxatives are given for the resulting con-

stipation. (*g*) Opium suppositories relieve *tenesmus* (see DYSENTERY). (*h*) The diet may be increased in a few days to bouillon with raw egg, hashed beef or chicken, toast, zwieback, roast beef and milk; fruits and vegetables even when cooked should be given with caution, because recurrence predisposes to chronic enteritis. (*i*) Cramps, collapse, etc., are treated as in cholera (*q. v.*).

CHRONIC ENTERITIS.

Etiology and Pathology.—The etiology is that of acute enteritis, from repeated attacks of which the chronic type may develop.

Chronic enteritis suggests a causative chronic gastritis, achylia gastrica and other gastric conditions; or intestinal catarrh may cause anorexia, eructations or abdominal distention. Hyperchlorhydria may disturb the intestines by the escape into them of more acid than they can neutralize, and hypo- or achlorhydria initiates bowel trouble because hydrochloric acid bears an important relation to the intestinal and pancreatic secretions.

The pylorus remains closed until the food in the stomach is ready for the intestinal juices and then it relaxes; the hydrochloric acid, reaching the duodenum, provokes a flow of bile (which arrests the action of the pepsin), and of the pancreatic secretion (which neutralizes the hydrochloric acid) by acting upon the duodenal mucosa, which gives off into the blood "secretin"; secretin acting upon the pancreas causes it to secrete; then the pylorus again relaxes. When the pancreatic juice is poured out it contains no active proteolytic ferment, but in the succus entericus there is an activating ferment (enterokinase) which converts trypsinogen into trypsin. The bile activates the fat-splitting pancreatic ferment.

In chronic enteritis the mucous membrane is brownish-red or slate-gray and often thickened and the muscularis is sometimes hypertrophied. Polypoid hyperplasia, cystic degeneration, atrophic changes, catarrhal erosions and follicular ulcers may occur.

Symptoms.—*Constipation* is more frequent than diarrhea; in some cases a single, soft, morning evacuation is noted, or perhaps several; in others constipation is broken occasionally by short attacks of colic and diarrhea; continuous diarrhea indicates inflammation of both the large and small gut. Constipation is due to alteration of the nerve fibers or inflammation in the muscular coat. Abdominal tension or colic is distinguished from gastric discomfort and pyloric spasm in that it occurs independently of eating. Diarrhea results from decomposition and fecal irritation. There may be pain, flatulency, borborygmus or occasionally tenderness over the bowels, but the sole diagnostic criterion is the presence of *mucus;* its intimate mixture with the stool indicates inflammation in the small gut; coating of the passages with mucus indicates colitis, and evacuations of mucopus alone, proctitis. Mucus globules when bile-stained indicate inflammation of the small gut, as do shreds of bile-stained mucus. Pus cells are rare except in proctitis. *Mucus is always indicative of enteritis* and never occurs from ulceration or carcinoma

alone (a slight coating of mucus over hard feces is not significant.) *Blood always indicates a complication*, such as piles, ulceration or cancer. The stools may contain much *undigested meat, starch* or *fat.*

The *functional examination* of the feces does not give us as exact information as does the examination of the stomach contents. Schmidt's test diet includes 3 pints of milk, 3 ounces of zwieback, two eggs, 1.6 ounces of butter, one-quarter pound of rare, tender steak, 6 ounces of boiled potatoes, 2 ounces of oatmeal and two-thirds ounce of sugar, distributed among the three meals. With the first meal a capsule of five grains of charcoal or carmine is given and the first examination is not made until the stools become colored black or red. In this way (i) the "period of passage" is established, the normal time being ten to twenty hours. (ii) Mucus, microscopically, appears as translucent, sago-like and often bile-stained flakes, which must not be confused with starch particles. (iii) Much connective tissue, recognized by its yellowish-white color and its toughness, indicates reduction of the hydrochloric acid. (iv) Muscle fibers, appearing as reddish threads or lumps, may indicate achylia gastrica, lack of trypsin, lack of the activating enterokinase or very active peristalsis. (v) Free starch granules indicate intestinal catarrh; normally only starch enveloped in cellulose is present. (vi) The normal stools shows hydrobilirubin, detected by mixing fluid feces with an equal amount of a 5 per cent. solution of bichloride of mercury, which mixture turns yellowish-red after twenty-four hours; bilirubin, recognized by a green color in the above test, indicates catarrh of the small bowel (except in young children). (vii) The fermentation test is made by mixing a dram of the feces with sterile water in a bottle which is connected above with a tube containing water; almost no gas results in normal cases; an acid reaction with fermentation is due to carbohydrate fermentation; if it becomes alkaline and foul in odor it is due to proteid fermentation. (viii) With experience, it can be readily determined whether the fat is increased.

Nutrition is maintained in some cases; in others pallor, emaciation, slow pulse and cold extremities may be noted. Attacks of diarrhea and lientery suggest achylia gastrica.

Prognosis.—Save in either extreme of age it is good as to life, but is doubtful at any age as to complete recovery. Improvement is tardy and often transient. Intestinal atrophy may develop, though less frequently than in puerile forms.

Treatment.—(*a*) An *hygienic* conduct of life is most essential. A flannel band should be worn over the abdomen. (*b*) The *diet* must be carefully supervised; salads, alcohol, fruits and vegetables of every description, either raw, or cooked, sweets, pickles and acids are among the forbidden foods; eggs, fresh butter, lean meats, stale bread, rusks, thoroughly cooked rice or sago and potato purée are permissible; *i. e., simple foods,* in *small amounts* and at *frequent intervals,* controlled by frequent examination of the feces. (*c*) For constipation, often due to the careful diet, purgatives are avoided and the bowels moved by *enemata of olive oil.* For diarrhea, bismuth in dram doses and tannigen are most efficacious (see ACUTE ENTERITIS). Calcium carbonate and salicylate

are recommended (āā ℥ss to a quart of charged water; one-half glass q. i. d.). Schmidt employs 10 per cent. hydrogen peroxide in agar. (*d*) *Flatulency.* As flatus results from stagnation plus catarrh, the indications are restriction of foods which ferment (*v. s.*) and the administration of aromatic remedies (see page 535).

℞—Mentholis . . . . . ℥ss
Phenylis salicylatis . . . . . ℥iij
Extracti belladonnæ . . . . . gr. iv
M. et fac capsulas xx.
S.—One after meals.

(*e*) *Colitis* (see TREATMENT OF DYSENTERY).

ENTERITIS (COLITIS) MUCOSA OR MEMBRANACEA.

This disease was described by Mason Good (1825) and Da Costa (1871). The name implies inflammation, but most cases are neither enteritis nor colitis. Nothnagel named the non-inflammatory variety *colica mucosa,* which is a secretory neurosis.

Etiology.—(*a*) Eighty to 90 per cent. occur in young, *nervous women,* 40 per cent. of whom have uterine troubles; few cases are seen in nervous men or children. (*b*) Nearly all patients suffer from chronic *constipation.* Its pathology is unknown; in some autopsies no anatomical alteration is noted, in others that of enteritis.

Symptoms.—(*a*) *Mucous casts* of the bowel, shreds or lumps are voided periodically; the mucus may resemble a huge diphtheritic cast, is usually evacuated in large amounts, which often constitutes the entire bowel movement and contains very few leukocytes or other signs of inflammation. (*b*) There is usually in the attack *colic* or pain and tenderness over the splenic flexure of the colon, sometimes diffuse and occasionally radiating into the leg. If the mucus firmly adheres to the bowel wall the pain is particularly severe and obstruction of the bowels may be simulated. *Tenesmus* in voiding the mucus is usual, as are formation of gas and *increase* of the original *nervous symptoms,* as palpitation, tremor or vertigo. There may also be coincident diarrhea, achylia gastrica and a periodic sand formation. (*c*) *Constipation,* distention and anorexia exist between the paroxysms, which last a day or even a week and are separated by weeks or months. It is said that death may occur during an attack.

Treatment.—(*a*) The less the fundamental *neurotic symptoms* are in evidence the better are the results of therapy; education, the rest cure and other measures outlined under hysteria and neurasthenia are indicated. (*b*) *Constipation* (*v. i.*) is treated dietetically—Graham bread, leguminous vegetables, including the husks, vegetables with much cellulose (which seems to be the most important element)—fruits with small seeds and thick skins, as gooseberries, currants or grapes, large amounts of cream, butter and bacon, and olive oil. Von Noorden's results with this diet are: 79 per cent. partially or permanently cured, 16 per cent. unknown result and 5 per cent. failure. (*c*) Local measures include *colonic flushings* with normal salt solution, or half a pint of

olive oil left in the rectum over night (Kussmaul). (*d*) *Narcotics, purgatives,* irrigation with alum, tannin and silver solutions, and operations are distinctly *contra-indicated.* The morphine habit may be readily contracted. Belladonna suppositories are very efficient for the pain. The patient in an attack is kept in bed and heat is applied to the abdomen.

DIPHTHERITIC, CROUPOUS AND PHLEGMONOUS ENTERITIS.

Klebs-Loeffler diphtheria is very rarely encountered, and practically all membranous enteritis is *diphtheroid* (*v.* page 78).

Phlegmonous or *purulent enteritis* is extremely rare and chiefly of anatomical interest. Most cases develop in the duodenum. It may be primary, but more often is secondary to intestinal ulceration, carcinoma or intussusception. In a form due to the colon bacillus, multiple purulent foci are found in the intestinal wall, which frequently cause perforation.

INTESTINAL ULCERATION.

Ulceration in *acute infections,* as typhoid, dysentery, sepsis, diphtheria, etc., and *chronic ulcers,* as syphilitic, tuberculous and actinomycotic, are considered under General Infections. Rarely ulceration has been observed from favus and mucor corymbifer. *Inflammatory ulcers* as the catarrhal and follicular, usually occur in the colon; they may be simple or so-called dysenteric (*q. v.*); they are seen in enteritis, infantile diarrheas, etc. *Stercoral* (*decubital*) *ulcers* develop from the pressure of hard feces or foreign bodies, in the dilated pouches of the colon or above an intestinal stenosis. *Constitutional affections,* as leukemia, scurvy and amyloidosis, uremia and mercurial and arsenical poisoning are occasionally causes.

Symptoms.—The symptoms are rarely distinctive without suggestive etiological factors; most ulcerations occur without symptoms or with ambiguous symptoms. (*a*) *Diarrhea* is frequent, particularly in ulceration of the large gut; it is often absent with lesions of the small gut or upper colon; it results from decreased absorption and increased peristalsis, due to exposure of the intestinal nerves. (*b*) *Blood* in the stools is mixed with them in lesions of the small gut, sometimes covers them in lesions of the large gut and is voided independently of the feces in rectal lesions. It is more often dark and tarry, especially in tuberculosis, than bright, except in low-situated lesions and in some profuse typhoid, dysenteric or duodenal hemorrhages. Blood is common in other diseases than ulceration, as in cancer, piles, stasis and blood diseases. (*c*) *Pus* is seen more frequently in colonic than in ileac ulceration; it is common in cancer, proctitis and croupous enteritis. Pure pus is rather indicative of rupture of extra-intestinal pockets into the intestine, as appendicitic and salpingitic abscesses. (*d*) *Mucus* indicates enteritis only, which may complicate ulceration. (*e*) *Shreds of tissue* are very rare, save in dysentery. (*f*) *Local symptoms* are ambiguous, as tenderness, pain and tympanites. (*g*) *General symptoms* depend on the basic disease; fever is most frequent in cancerous, dysenteric or tuberculous ulcers. (*h*) *Rupture* causes

peritonitic adhesions, diffuse peritonitis, retroperitoneal cellulitis or subphrenic pyopneumothorax. (*i*) The *x*-ray may show total absence of a shadow where the ulcers lie or else a fine marbled appearance.

Treatment.—Treatment is that of enteritis or colitis. In chronic ulcerative colitis colostomy may result favorably.

INTESTINAL DISORDERS IN INFANTS.

Etiology.—Classification of the etiological factors is difficult because of the numerous species of bacteria in the intestine and the impossibility of drawing any absolute line between chemical and bacterial factors and between functional and organic changes. Most cases occur in children between *six and eighteen months old*, in the *summer months*, July especially, in the *poorer classes* and in infants *not fed at the breast*, for two main reasons: cow's milk is prone to disturb digestion and there is a greater chance for bacterial infection. Toxins may form in milk, when it is long in transit, particularly in hot weather. The *Bacterium lactis aërogenes* causes inflammation in the small and the *Bacterium coli* in the large intestine; the proteus, pyocyaneus, gas bacillus, various saprophytes, and Bacillus dysenteriæ are also factors, but their relative importance is not clearly defined; the Streptococcus enteritis can hardly be considered as a distinct type. The distinction between intoxication and infection is maintained with difficulty; *intoxication* causes symptoms as erythema, hemorrhagic diathesis, anemia, nervous disturbances and dyspnea; and *infection* produces complications, as lobular pneumonia, nephritis, phlegmon, arthritis, purulent pleurisy and necroses in the liver. In institutional epidemics infection may be carried from one individual to another by fecal contaminations. Insects may convey infection.

Symptoms.—1. The *acute dyspeptic or fermental form* cannot be sharply distinguished from other severer forms, though possibly caused by the same microörganisms. The pathological findings are often remarkably insignificant. The *temperature* usually rises suddenly to 103° or 104°, remains high for a few days and falls suddenly. The tongue is dry, the face red, the pulse rapid and the breathing sometimes dyspneic. There is restlessness—sometimes initial convulsions—anorexia and vomiting. The *stools* in infants are normally yellow from bilirubin, thick and slightly acid in reaction; they become green from bacterial action or reduction of the bilirubin into biliverdin, react alkaline from albuminous decomposition or acid from acid fermentation and become thin and offensive; the evacuations number six to ten daily. The microscopic findings are those of acute enteritis in adults, as mucus, leukocytes, triple phosphates in the alkaline stools or cholesterin in the acid stools; blood in any quantity is uncommon. Involvement of the small intestine is attended by gas, cramping, large movements, and tenderness. Marked nervous symptoms, extensive edema and emaciation may result. Albuminuria and cylindruria occur in 20 per cent. Recovery is usual and prompt after thorough intestinal evacuation.

2. *Cholera infantum* occurs in severe infections, usually in children already suffering from some intestinal disorder; it constitutes 2 per cent. of summer diarrheas. The *bowel movements are copious*, alkaline and flocculous; though at first they contain food and are perhaps offensive, they become watery and odorless. Both the stools and the general clinical aspect resemble the picture of *cholera Asiatica* and *cholera nostras* (*q. v.*). Though there is *fever*, the *extremities* are *algid and cyanotic*, the *pulse small*, the heart tones weak and all symptoms of *collapse* are present; there are the pinched pallid *facies cholerica*, the *thirst* and the incoercible *vomiting*. The skin is rigid, as though the fatty tissue had hardened (*scleredema* or *sclerema adiposum*), and nephritis is almost invariable. These symptoms result from loss of great quantities of fluid by purging and vomiting, and from intoxication which produces the convulsions, irregular and Cheyne-Stokes's breathing, retraction of the neck, stupor and other symptoms, termed the "hydrencephalic state." Oestreich holds that hypostasis, resulting from compression of the lungs by the tympany, causes more deaths than does the toxemia.

3. *Acute enterocolitis* (ileocolitis) may follow the dyspeptic form or the various specific infections in children, or may begin as an independent form. Pathologically it includes types which cannot be separated clinically, as simple enterocolitis, follicular ulceration and diphtheroid enteritis. In Rotch's cases of infantile diarrhea the Bacillus dysenteriæ was found in 16 per cent., sometimes in the dyspeptic type, but most often in the ileocolitic type (78 per cent.). In one collection of 412 cases Shiga's bacillus was found in 63 per cent.; Geo. Weaver never found it, though the Flexner-Harris type occurred in 25 per cent. (*v.* BACILLARY DYSENTERY).

The *symptoms* resemble those of dysentery in the adult. The stools are small, yellow, green or brown and very frequent. Rotch tabulates the differential features as follows:

Acute fermental diarrhea.	Acute ileocolitis.
(*a*) Small intestine, mostly.	Large intestine, mostly.
(*b*) 10 to 12 discharges; often large.	10, 15 to 50 discharges, usually small.
(*c*) No or little blood.	Blood and sometimes shreds of tissue or membrane.
(*d*) Mucus.	Mucus.
(*e*) No tenesmus.	Tenesmus.
(*f*) Not much abdominal tenderness and pain.	Abdominal tenderness and pain.
(*g*) No or slight lesions.	Lesions marked.
(*h*) Temperature high—104° F. for one or two days, and then falling sharply by crisis.	Temperature may be high at first (103° F. to 104° F.) but usually soon moderates (99° to 101° F.) and falls gradually by lysis to normal after some weeks.

The severity of the symptoms varies greatly; the catarrhal form may cause mild symptoms or result in death, with or without gastric symptoms. The ulcerative and diphtheroid types are less often attended by vomiting, but run a fatal course of days or weeks.

4. *Chronic dyspeptic disturbances* often occur with gastric dilatation. The onset is gradual or it may directly follow the acute forms. The stools are green or gray, contain mucus, gas, sometimes pus and tissue shreds. Food and fluid are poorly absorbed; undigested food particles and fatty stools are observed; mucus is not common in marked cases of intestinal atrophy. Vomiting is rather frequent. There is usually little general disturbance, no fever and little or no colic. In some cases the emaciation may become extreme (pediatrophy, athrepsia), the muscles becoming weak, the voice faint, the eyes lustreless, the face of the "Voltaire type," and the abdomen distended. Death results from marasmus or intercurrent infections.

Preventive Treatment.—Children should, if possible, be taken to the country in the hot months. Excessive swaddling is to be avoided.

DIET.—(*a*) If the mother cannot nurse her child, a *wet-nurse* should be procured, as few stomach and bowel diseases occur in breast-fed infants. The wet-nurse must be carefully examined for syphilis and tuberculosis. The milk from the breast should spurt freely in several streams. A weak child may become dyspeptic even at the breast; calomel irritates but magnesium carbonate gives the most rapid relief; other children thrive, even when the stools are green. Sometimes colic and dyspepsia in the child are due to menstruation, beginning pregnancy, excitement, lack of exercise or alcoholic indulgence in the nurse or mother. If a wet-nurse cannot be obtained, (*b*) *cow's milk* or some *prepared food* must be used, but most of the latter are objectionable because their carbohydrates are not dextrinized. Cow's milk differs from human milk in many details; its contamination is far more likely, whence the importance of pure milk and sterilized nipples and bottles. Human milk contains about 1 per cent. of proteids, 4 per cent. fats and 7 per cent. of milk-sugar, but in cow's milk the percentages are respectively about 3.5, 3.5 and 5 per cent.; in cow's milk the casein constitutes 80 per cent. of the proteids, which are physically and chemically different and contain more whey proteid and more of certain relatively uninvestigated substances. In cow's milk the ferments and fats are different and the mineral salts, especially phosphates, are increased, which impede digestion. The first objection is the high percentage of proteid (casein), which must be predigested or the milk diluted. Simple dilution, however, necessitates giving too much water or reducing the fat and milk-sugar. In *substitute feeding, the proteid of cow's milk must be reduced, fat added in the form of cream,* and *milk-sugar added* to attain the normal percentage. The milk may be diluted with thin oatmeal-water, as a diastase ferment is present earlier and in larger amounts than previously admitted, though nature doubtless has her designs in withholding starches from mother's milk. Low dilutions should be given at first and gradually increased, regarding digestion and *nutrition,* as determined by regular weighing. Sterilization of milk destroys ferments and protective substances.

Treatment of Acute Dyspepsia.—(*a*) The *stomach* is *washed out,* if toxic symptoms are marked. with plain water or a 1 to 3000 resorcinol solution. (*b*) *Calomel* gr. $\frac{1}{4}$ and magnesium carbonate gr. j every three

hours for three doses, or one dose of castor oil (℥ss–j) are usually administered, but cathartics congest or even erode the mucosa. (*c*) The *bowel should be flushed* with one pint of warm water. (*d*) *Food should be withheld* for two days, and plenty of boiled water should be given. Albumen-water (the whites of two eggs beaten in a pint of water) may then be given, and thin arrow-root-water, beef-juice and peptonized milk, may gradually be administered until the green color of the stools changes to a normal yellowish-brown. In one type, fat is not assimilated; the fatty acids, uniting with alkali, form soapy stools and withdrawing alkali, lead to alkali-penia and acidosis. In another type, starch is the injurious factor and in another, there is sugar-injury, as in gas-bacillus infections. The dysentery bacillus works least injury with lactose administration, which minimizes the nitrogenous waste (Kendall). Milk proteid seems the least injurious, particularly if the whey is removed from skimmed milk. (*e*) *Other medication* is usually superfluous. Lesage recommends lactic acid for alkaline green stools, ℥j of a 2 per cent. solution (p. c.). Resorcinol ⅓ grain and dilute hydrochloric acid ♏v may be given after meals.

Treatment of Enteritis.—If the case is seen early the *stomach* is *washed* out and *feeding suspended* as outlined above. The child should have plenty of boiled water and cool applications to the skin are indicated unless the extremities are blue and cool. *Tannigen* may be given, 10 grains, several times daily, even in the very young. As in enteritis of adults, bismuth subnitrate, ℥ss, bismuth salicylate, ½ to 1 grain and if these are ineffective, opium may be administered. (See page 269 for dosage.)

Treatment of Cholera Infantum.—Lavage is ineffective, unless it is done in the earliest stages. There are *three main indications:* (*a*) *fluid must be restored* to the tissues, by subcutaneous saline transfusions which support the heart, fill the vessels, stimulate the kidneys and eliminate the toxins; 2.5 per cent. dextrose infusions are valuable; (*b*) *cardiac stimulation,* by hypodermics of strychnine and camphor and local heat; champagne quiets vomiting and supports the heart; brandy and whisky are given as in bronchopneumonia (*q. v.*), though perhaps alcohol further injures the intestinal epithelium; and (*c*) *secretion must be checked* by paregoric every four hours, small enemas of starch and laudanum or, which is more effectual, morphine. (See page 269.) In the fortunate cases which rally, nourishment is resumed only with the greatest caution.

The Treatment of Colitis.—This is like that of dysentery and includes the measures above indicated, flushings with salt solution, 1 per cent. tannin or 1 to 1000 salicylic acid.

The Treatment of Chronic Dyspepsia.—Treatment in institutional cases begins with nursing; one wet-nurse can nurse several children. Gastric lavage with two ounces of plain water, normal salt or 1 per cent. Carlsbad salts solution, increases functional activity. The extract of nux vomica stimulates secretion. Food is given in the smallest amounts compatible with maintenance of weight and energy.

APPENDICITIS.

Definition.—Appendicitis, the chief abdominal malady, is an inflammation of the vermiform appendix.

History.—Fitz in 1886 drew attention to the subject and coined the name, and Willard Parker first (1867) advocated operation. Appendicitis was previously confused with typhlitis, inflammation of the cecum; the term "perityphlitis" should be discarded. Our knowledge is largely due to the work of American surgeons.

Etiology.—1. Predisposing Factors.—(*a*) The appendix, a functionless vestige of the cecum, is inherently *less resistant.* (*b*) Its *lumen* is *smallest at its cecal end,* which favors retention of foreign bodies, promotes microbic growth and decreases or suspends drainage into the bowel. (*c*) The *meso-appendix,* which conveys the blood to the appendix, is *shorter than the appendix,* which therefore curls; the blood supply is easily altered by appendiceal edema and infiltration. (*d*) *Foreign bodies* and fecal concretions may produce erosions which apparently promote microbic access and therefore inflammation; Aschoff combats this view. J. F. Mitchell found foreign bodies in 7 per cent. of 1400 cases of appendicitis and Louis J. Mitchell in 1600 autopsies, 1 per cent.; they include seeds, capsules, gall-stones, pins, nails, shot, fish-bones, etc. (*e*) *Age.*—Over 50 per cent. occur under twenty years of age and 30 per cent. between twenty and thirty. It is rare in infancy and with advancing years obliteration and atrophy lessen its frequency. (*f*) *Sex.* There is a slight preponderance in males.

2. Bacteriology.—The colon bacillus is found in 80 per cent. of cases, though probably in some of them it has overgrown another causal organism. Next is the staphylococcus, the virulent streptococcus, then the pneumococcus, proteus and pyocyaneus. In symbiosis with these organisms, or possibly operating without them as in gangrene, the anaërobes (gas bacillus, fusiform bacilli and anaërobe streptococcus) are factors in the necrosis, peritonitis and toxemia. Less frequently the typhoid, tubercle, influenza or actinomycosis organism is found; of the last 150 cases exist; Fenwick, in 1120 tuberculous ulcerations in the bowel, found but 0.8 per cent. in the appendix.

Symptomatology and Pathology.—1. The Early Clinical Course.—The *onset is precipitate* and characterized by *four main symptoms.* (*a*) *Sudden, severe abdominal pain,* the intensity of which ranges between colic and agony, is due to acute retention within the appendix caused by the occlusion of the cecal end. It is felt over the *right lower abdominal quadrant* or over the pit of the stomach, and may even radiate along the genitocrural nerve to the testis, which is sometimes retracted. Reaching its greatest severity in a few hours, the pain persists until the pressure of the pus is relieved by drainage into the bowel, perforation or gangrene. Sudden relief from pain often indicates gangrene. (*b*) *Nausea and vomiting* result reflexly from occlusion and follow within a few hours after the pain. (*c*) *Abdominal tenderness* is *general at first* and is often associated with a cutaneous hyperesthesia, which covers like half a girdle the right lower abdomen from the spine to the mid-line. The

right rectus muscle resists palpation, which explains most cases of early apparent tumor. A little *later* the general tenderness disappears, leaving *well-localized tenderness* over the appendix; *McBurney's point* is in the right lower quadrant of the abdomen (about two inches from the anterior superior iliac spine in a line drawn between it and the navel) or a little

Fig. 38

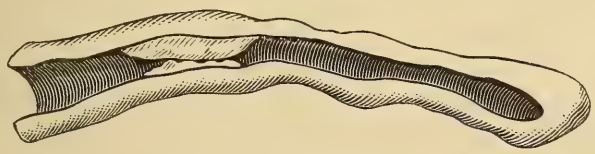


Fig. 39

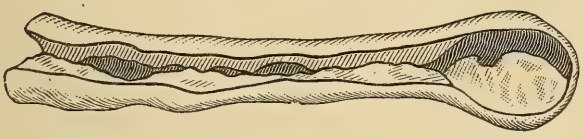


Fig. 42

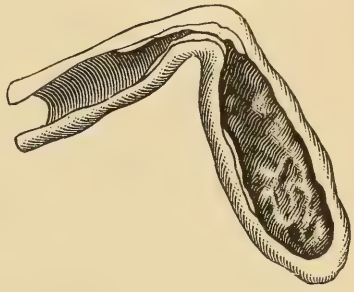


Fig. 40

Fig. 43

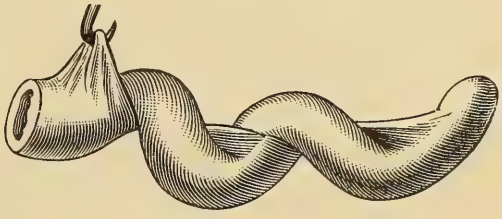


Fig. 41

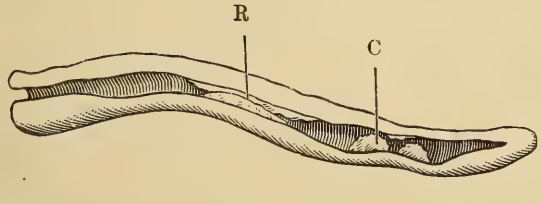


Fig. 44

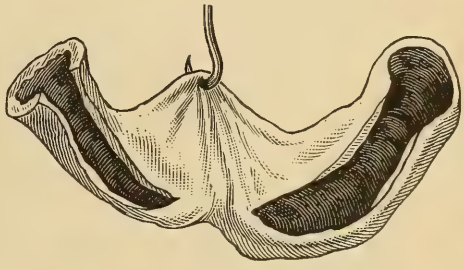
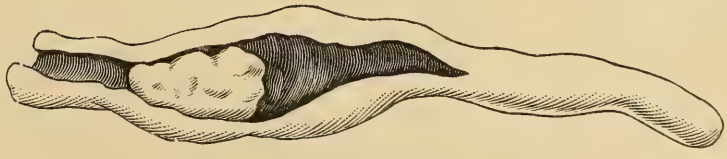


Fig. 45



Figs. 38 and 39.—Obliterating appendicitis. Fig. 40.—Obliterating appendicitis, with distal enlargement. Fig. 41.—Obliterating appendicitis (at *R*), with calculi (*C*). Fig. 42.—Appendicitis with bending, obliteration and distal distention. Fig. 43.—Appendicitis with curling around its mesentery. Fig. 44.—Strangulation by a band. Fig. 45.—Calculous obstruction. (Dieulafoy.)

below it in adults or above it in children; less frequently the tender point may be found near the loin, pelvis (5 per cent.), bladder, rectum or uterus on rectal or vaginal examination, gall-bladder or very exceptionally on the left side of the abdomen or in hernial sacs, all of which correspond to possible variations in appendicular location. Pressure with the hand on the left half of the abdomen causes pain in the right side when the

pressure is relieved. Palpation must be made gently, lest rupture occur. (*d*) *Fever* generally develops in a few hours to a day, according to the severity of the infection, rarely with a chill, reaching 101°–102°, or higher in some virulent types or in children. It signifies septic absorption and may cease suddenly in gangrene or gradually in rupture and may slowly recur from extension; abscess or peritonitis may coexist with low temperature. The *pulse* usually follows the temperature curve, though the author has seen it remain normal in cases of early gangrene. The *leukocytosis* parallels the fever, and often measures the sepsis; no reliance can be placed on its absence as an assuring sign nor on its presence as an indication for operation; extremely high counts (over 100,000) are ominous; counts of 900 or 600 are reported. The urine is often febrile, scanty and usually contains indican and sometimes albumin or even blood; dysuria may result from appendicitis in the pelvis; renal colic may be closely simulated. Profuse bowel hemorrhage is recorded. Constipation is usual. In Dieulafoy's toxemic type toxemia may obscure the local symptoms.

2. The Later Clinical Course.—(*a*) The *inflammation subsides* gradually by drainage into the cecum. Though the inflammation may be circumscribed, the appendix rarely returns to normal; it suffers necrosis and ulceration which leave the appendix infiltrated, thickened, and more or less constricted at some point or the lumen is obliterated. The mucous membrane may be wholly substituted by granulation tissue. Occlusion at its intestinal end causes a cyst-like swelling as large as the thumb or even as a sausage. Adhesions to the abdominal wall and gut are common. (*b*) After a few days a *localized* peritonitic *abscess* frequently forms, usually over the psoas muscle at the angle between the ileum and cecum, but occasionally in some unusual localization of the appendix (*v. s.*), from perforation, generally near its mesenteric attachment, from necrosis or gangrene; when the abscess is well demarked, the pain, fever and pulse-rate decrease. A *well-defined tumor* may develop, over which no peristalsis occurs; dulness or tympany is elicited according to the depth of the tumor or its fluid or gaseous contents; fluctuation is present only in large abscesses; in infections due to the colon bacillus the tumor is more largely infiltration than pus. In the incised abscess the green or black and totally gangrenous appendix usually may be seen. The abscess may form without any symptoms other than those of the onset. The *result of the abscess* varies greatly: (i) it may rupture into the intestine which forms its wall (2 per cent.), and possibly carry the appendix with it; (ii) it may rupture into the urinary tract, of which cases nearly one-half are fatal; (iii) it may cause purulent thrombophlebitis, and any manifestations of septicopyemia, as secondary fever, suppurative pylephlebitis, pulmonary embolism, liver abscess, or subphrenic abscess; (iv) it may rupture into the retroperitoneal tissue, followed by high temperature; and all possible extensions may occur, as into the groin, scrotum, pelvis, pararenal tissue, pleura (84 per cent. are right-sided), lung, gluteal region, back, hip-joint, etc.; and (v) it may rupture into the free peritoneal cavity. (*c*) *The third cardinal issue is diffuse septic peritonitis;* its unfortunate and unmistakable symptoms are a sudden

fall of temperature followed by a secondary fever, diffuse replacing localized tenderness and rigidity, leukocytosis, persistent projectile vomiting, rapid pulse, Hippocratic facies and collapse from profound intoxication. It most often follows the localized abscess, but may occur directly after a gangrene which is so sudden as to allow of no protective adhesions, or after perforation from a foreign body in the appendix. Variations, due to the type of infecting organism are as follows: (i) The staphylococcic peritonitis generally produces but little pus, or, if any, a seropurulent fluid; plastic lymph prevents absorption for a short time, but, as the peritoneum becomes abraded, leads to intoxication, collapse and diarrhea. (ii) The streptococcic form produces no pus, but causes a granular, blistered, dry peritonitis, rapid pulse, delirium and tympany. (iii) Colon infection produces an abundant, creamy, thick, offensive pus and slowly causes death after great depression, delirium and low arterial pressure.

3. Remote Sequences.—Intestinal strangulation by adhesions and from chronic changes in the appendix reflex disturbances may result.

In *children* the onset is very abrupt, the vomiting repeated, pain greater, the appendix more palpable and diarrhea and diffuse peritonitis more common than in adults, because the omentum is less able to envelop the appendix. In *aged persons* gangrene is more frequent.

Diagnosis.—The diagnosis depends on the four cardinal symptoms of onset, followed by leukocytosis and other secondary developments. If all these symptoms are considered separately an analysis is necessary of every abdominal lesion causing pain alone, vomiting alone, temperature, sensitiveness, abscess, tumor, disease in the right lower abdominal quadrant, perforation or peritonitis.

Pain occurs in gall-stones, renal calculi, tabetic crises, internal incarcerated hernias, epigastric hernia, intussusception, gastric or duodenal ulcer, tuberculous peritonitis, stone or torsion of the ureter, Dietl's crisis in floating kidney, uremia, colica mucosa, acute pancreatitis, the crises of angioneurotic edema, herpes zoster, pelvic peritonitis, ruptured extra-uterine pregnancy, twisted pedicles, pyosalpinx, dysmenorrhea and the reflected pain of vertebral disease, pleurisy and pneumonia. *Fever* in typhoid precedes the local symptoms. These affections, as well as gravitating abscesses, infected urachus, lesions of the abdominal wall, cancer, hysteria and hypochondriacal fear of appendicitis, are almost invariably excluded by the sequence of symptoms and careful general examination. *Appendicitis, chronic ab initio,* causes flatulency, constipation alternating with diarrhea and a dragging discomfort in the right lower abdomen; appendicitis may cause hypersecretion, hyperchlorhydria, gastralgia, epigastric tenderness or pain (also experienced there when pressure is made upon the appendix) and pylorospasm (*v.* page 515). Confusion is possible with a mobile cecum.

Prognosis.—The prognosis is most uncertain, because (*a*) there is no safe criterion for early diagnosis of the type; (*b*) the symptoms and signs are not proportionate to the actual lesions, *e. g.*, light cases clinically may prove to be the most severe anatomically; (*c*) apparent improvement is often to the experienced observer the onset of alarming

complications; Dieulafoy well calls them "treacherous lulls" (*accalmies traitresses*); (*d*) recovery is seldom complete anatomically, whence chronic, relapsing or residual appendicitis occurs in a more severe form in a full third of the cases.

Treatment.—Cases may recover, temporarily (70 to 80 per cent.), but it is impossible to predict which ones will do so. After testing and seeing tested the so-called medical treatment, consisting of rest, starvation, laxatives and other placebos, and seeing gangrene, peritonitis with low temperature and death occur in "mild" cases, the conviction was inevitable that *appendicitis is always surgical, regardless of type, time or tendencies.* A surgeon at once should be associated with the practitioner. Recovery under medical treatment is due to good fortune alone. The so-called indications for operation are too often signs of irreparable damage. Early operation *within one or two days* of onset is attended by the least danger. Many operators dislike to operate between the *second* and *eighth days* or other arbitrary limits, but this position implies that the clinician can diagnosticate the anatomical changes which are present; gangrene, thrombophlebitis, imminent rupture, etc., cannot be excluded; in this period simple drainage is indicated—possibly, too, appendicectomy, if the appendix obtrudes in the field of incision; lavage, exploration and protracted operation are dangerous. *In later cases,* when there is apparent regression, treatment may be more conservative; complete demarkation may be waited for, but even in such cases thrombophlebitis or rupture into the general peritoneum may intervene. In *diffuse peritonitis* incision and simple drainage are indicated. The patient is placed in the semisitting posture (Fowler) and given constant normal salt enemas (Murphy). Many cases recover (see PERITONITIS). (*e*) *After the attack.* After one attack of appendicitis the patient is prone to recurrence. In cases in which there was only incision and drainage of the abscess the attacks recur. Adhesions, flexions and stenoses favor a recurrence and digestive disturbances.

INTESTINAL OBSTRUCTION.

Etiology.—Obstruction arises from:

I. Intussusception (Invagination).—In this most frequent form of acute obstruction (40 per cent.), the external part (the intussuscipiens or vagina) receives into it the intestine just above (the intussusceptum or invaginatum). It develops in perfect health, in various acute and chronic affections, tumors, strictures of the gut, diarrhea and contusion. *Two theories* are advanced: (*a*) that the gut is spastically contracted and enters the gut below, and (*b*) that the gut below is paretic. The author saw, with Dr. Walls, a case in lead colic. In acute intussusception Wiggin found 50 per cent. in the fourth, fifth and sixth months of life, when the long mesentery of fetal life persists. In chronic intussusception 50 per cent. occur between twenty and forty years of age and 66 per cent. in males. *Pathologically* only the vital form is of clinical importance, in which the mesentery and its vessels are also invaginated; the "agonal" form occurs during the death agony, chiefly in children, in

the small gut, is often multiple and does not involve the mesentery. The vital form is usually single, descending (in 99 per cent.) and complete; incomplete forms may result from polyps or other tumors, usually benign, which drag down the gut. There are three main forms: (*a*) the enteric, the small gut invaginating into the small gut, 30 per cent.; (*b*) the ileocecal, the small gut entering the large gut, 52 per cent.; and (*c*) the colonic, the large gut invaginating into the large gut (18 per cent.) or rectum; in rare cases Meckel's diverticulum may invaginate into the small gut.

Symptoms.—(*a*) There is sudden, severe, *reflex pain*, which is usually umbilical in children but sometimes occurs elsewhere in adults, is continuous or in chronic forms of intussusception, paroxysmal. (*b*) Early *reflex vomiting* may occur in children, though in adults it is less frequent than in other forms of obstruction; the later vomiting is fecal in 13 per cent.; vomiting is more marked in high than in low invagination and depends considerably on the degree of mesenteric invagination (vascular disturbance). (*c*) The *stools are characteristic*, showing blood and mucus in 97 per cent. of acute and in 50 per cent. of chronic cases; the movements may not stop at once but only after several evacuations, and severe tenesmus (50 per cent.). (*d*) A *sausage- or egg-like movable firm tumor* (55 per cent.) may be palpated, most often over the ascending or transverse colon, sometimes by the rectum or vagina, and is occasionally visible at the anus, where it has been mistaken for prolapse or polyp, and extirpated. It is most common in children, in the ileocecal form, and nearly always to be felt during paroxysms of pain. (*e*) Meteorism and a depression in the right flank are infrequent.

Clubbe's description is excellent: "The very sudden onset in a previously healthy baby is a peculiarity of intussusception. The child is seized suddenly, screams, turns pale, vomits and in a short time seems to recover itself, only to cry again at intervals as if in pain. Soon after the first scream it may pass a normal motion. In from two to ten hours, in 97 per cent. of cases, blood will be passed by rectum. The mass, which varies much in size and position, can nearly always be made out by careful palpation when the child is under an anesthetic. The child may not look ill, its pulse-rate may be hardly raised and its temperature may be normal."

Issues.—Peritonitis results in 50 per cent. of acute and in nearly 100 per cent. of chronic cases; perforation occurs in chronic (15 per cent.) more often than in acute cases; sloughing off of the gangrenous bowel occurs in 42 per cent. of adult cases, but seldom in children; in Cruveilhier's case three yards of dead gut were discharged. Spontaneous reposition and fistula are very uncommon.

II. Strangulation.—This causes 35 per cent. of intestinal obstructions. Its *mechanisms* are innumerable; it is generally due to *bands* or isolated peritonitic adhesions; Meckel's diverticulum, the remains of the omphalomesenteric duct, which lies within a yard of the ileocecal valve and is sometimes continuous as a cord to the navel, caused strangulation in 183 cases (Hilgenreiner, 1902); omental strands under which the gut is caught; holding of the gut in abnormal positions; and binding of coils

to each other. Incarceration may occur in congenital or acquired *orifices and clefts* in the omentum or mesentery or under the suspensory ligament of the liver, lig. teres or lig. latum uteri. *Hernias* include strangulations in the femoral, inguinal, umbilical locations or in the less frequent types, as the obturator, sciatic, lumbar, intersigmoid, retrocecal, foramen of Winslow, duodenojejunal (Treitz's retroperitoneal form) or diaphragmatic. Seventy per cent. of cases occur in males, 40 per cent. between fifteen and thirty years of age, 90 per cent. in the small intestine, 67 per cent. in the right iliac fossa and 83 per cent. in the lower abdomen (Fitz).

Symptoms.—The symptoms are urgent. (*a*) Early reflex *vomiting*, from shock, voiding in succession gastric contents, bile and fecal matter which usually consists of yellow, brown or black fluid, or very rarely solid feces, is strongly indicative of organic strangulation and lasts from the beginning to the end of the disease. (*b*) There is early severe reflex *pain*, which is greatest at the navel and lasts until collapse, peritonitis or paralysis of the gut develops. (*c*) *Collapse*, with Hippocratic expression, weak pulse and hurried breathing, results reflexly and may occur early from the disturbed local circulation in the gut or later from peritonitis. (*d*) *Complete, sudden stoppage of the bowels* may result, in which no feces or flatus can pass; some feces from the lower gut may pass with enemata, but no gas. Meteorism of two kinds occurs: (i) the static variety in which gas accumulates above the obstruction and (ii) the local variety, in which the strangulated loop becomes distended, because its circulation is impaired; it may be seen or felt as an *immovable, tense mass*, showing no peristalsis and corresponding to the thick, edematous, hemorrhagically infiltrated and finally gangrenous gut. (*e*) The *urine* is decreased because of the vomiting and collapse; it contains indican if the obstruction lies in the small gut. Sometimes casts, albumin or nephritis are observed. (*f*) Strangulation sometimes may be found in the usual hernia locations.

Diaphragmatic hernia is left-sided in 88 per cent., is congenital or acquired (from trauma especially) and is recognized in but 3 per cent. of the reported cases; it closely resembles pneumothorax (*q. v.*). The *x*-ray differentiates.

In obstruction the mucosa in the occluded area may become ulcerated, easily penetrated by bacteria and hemorrhagically infarcted, leading to necrosis, perforation and peritonitis.

III. Volvulus and Knots.—Volvulus is a twisting of the intestine around its mesenteric axis; twisting of the gut on itself is rare; volvulus occurs in 14 per cent. of obstructions (Fitz); 68 per cent. of the cases are males, usually between forty and sixty years old; it occurs in the sigmoid flexure in 66 per cent., less frequently in the cecum, colon ascendens, small gut and stomach. Predisposing causes are a vegetable diet, constipation, a long sigmoid flexure with a long, narrow mesentery, and mesenteritis. The twist may occur through 180 to 360 degrees or more.

Symptoms.—The symptoms closely resemble those of the forms described, especially when knots form between coils of the small intestine. In volvulus of the sigmoid there is pain and vomiting, which are less marked than in strangulation or invagination and emesis is seldom fecal; the *local*

meteorism is *more pronounced* than in any other type of obstruction, for the huge coil of obstructed gut passes upward, in extreme cases pushes up the diaphragm to the third or fourth rib and seldom exhibits peristalsis, since its circulation is disturbed. But little water can be injected into the rectum. Hemorrhagic peritoneal exudate is common.

IV. Strictures.—Enterostenosis more often causes chronic than acute obstruction. (*a*) *Congenital* stricture is even rarer than congenital atresia, of which Helmholz collected 199 cases in the small bowel. (*b*) *Cicatricial* stricture results from healing of tuberculous, syphilitic, dysenteric, duodenal and very rarely typhoid ulcers. Arbuson collated 129 cases of tuberculous stricture. (*c*) Annular *cancer*, (*d*) compression by *tumors* (80 per cent. of which are pelvic) and (*e*) *traction* are other causes. (*f*) Inflammatory hypertrophy of the mucosa and muscularis is exceedingly uncommon.

Symptoms.—The symptoms of chronic obstruction vary with the location of the stenosis. (1) *Stenosis of the small intestine* may occur (*a*) above the duodenal papilla, as a result of gall-stones or duodenal ulcer and closely resembles pyloric stenosis; (*b*) stenosis in the duodenum below the papilla results from gall-bladder adhesions, ulcer, tumors or pancreatic disease; icterus is frequent, as well as vomiting of bile and pancreatic juice which digests starch and fibrin in an alkaline medium; other symptoms are dilatation of the stomach, pale stools, very slight abdominal meteorism, except over the epigastrium, and the absence of indicanuria. (*c*) Jejuno-ileac stricture may result from healed ulcers, inflammation in replaced hernias and adhesions to the internal genitalia; the ulcer stricture may be multiple, twelve being recorded in one case; extreme stenosis may cause no symptoms. There is constipation, which sometimes alternates with diarrhea, colic, meteorism and visible palpable peristalsis, causing "intestinal stiffness" until the obstruction is finally overcome by the hypertrophied muscle above the stricture, when a gurgling sound is sometimes heard. The colon is collapsed. (2) *Stricture of the large gut* is marked by *constipation*, which is significant in a person not previously constipated or when cathartics have no effect in chronic constipation; by *colicky pain*, even more than in stricture of the small gut, because the stools are harder; eventually by nausea and vomiting, which are successively gastric, bilious and stercoraceous; by *meteorism* along the colon and in the lumbar region behind, which is normally dull; by increased, even tetanic, *peristalsis* above the stricture, which possesses a localizing value; and by the *ribbon-like* or *sheep-stools*, which also occur in tumors, colica mucosa and constipation. Fluoroscopic results are often invaluable.

V. Tumors.—(See page 553.)

VI. Foreign Bodies.—Foreign bodies caused obstruction in 12 per cent. of Fitz's series. (*a*) *Gall-stones*—of which 250 obstruction cases are on record—lodge in the duodenum or lower ileum, ulcerating through the gall-bladder into the duodenum or less often the colon, or compressing the duodenum through a dilated pouch of the cystic duct. In but 50 per cent. is there a history of colic or of icterus. Collapse is usually not marked, a tumor is seldom felt and the constipation is often only rela-

tive or alternates with diarrhea. (*b*) *Fecal masses* constitute the chief obstruction of the large gut, especially in the sigmoid. The writer saw absolute obstruction develop after a Thanksgiving dinner; there was fecal vomiting, collapse and a mass of hard food, measuring two inches in diameter. (*c*) *Enteroliths* form slowly of phosphates, sometimes of bismuth, chalk, iron or magnesia, usually with an organic nucleus. They measure one to nine inches in diameter and one enterolith weighed four pounds. Most common in the large intestine, they may develop in diverticula of the small intestine. (*d*) Hair tumors, twisted masses of ascarides, fruit seeds, oatmeal husks, stones and foreign bodies (in insane subjects) are possible causes.

VII. Dynamic Ileus.—Under dynamic ileus (paralytic or spastic obstruction) may be included obstruction following mesenteric embolism, trauma, abdominal operations or inflammation in an undescended testicle; it is largely associated with acute peritonitis. Spasmodic obstruction may develop in gall-stone occlusion, ulceration, hysteria and intestinal crises of tabes; it rarely produces fecal vomiting.

Diagnosis of Intestinal Obstruction.—1. Of the Fact of Obstruction.—The symptoms are generally unmistakable and consist of constipation, cessation of flatus, local distention, compensatory peristalsis and frequently collapse. The hernial sites, rectum and vagina must be carefully examined.

2. Of the Location.—The uncertainties are manifold:

Obstruction of the Small.	*vs.*	Of the Large Intestine.
General Condition.—Earlier and more profoundly impressed.		Later and less impressed.
Symptoms more marked, more stormy onset, more vomiting, more pain. Fluoroscopic tests.		*Signs more marked*, as meteorism, less water can be injected, more peristaltic effort and palpable rigid coils, more results by rectal or vaginal examination, more tenesmus.
Indicanuria.—Earlier and marked.		Develops later or not at all.
Urine.—Much decreased.		Little decreased.

3. Of its Nature.—(*a*) It is practically impossible to recognize obstruction by *bands*, *adhesions*, Meckel's diverticulum, clefts and internal hernias (except diaphragmatic). (*b*) *Invagination* is not only the most frequent but also most characteristic in its clinical picture. (*c*) *Strangulation* is differentiated by its greater and earlier collapse, pain and vomiting, local meteorism and hemorrhagic peritoneal effusion. (*d*) *Volvulus* is suggested by advanced age, constipation, slower course, infrequent fecal vomiting and meteorism, which is greater than in any other form and is accompanied by little peristalsis. The small amount of fluid which can be injected, the hemorrhagic peritoneal fluid, tenesmus and slightness, lateness or absence of indicanuria are also suggestive. (*e*) In some instances of *gall-stones*, the lack of shock, of increased pulse and of meteorism, and the paradoxical intermittent release of flatus and feces, fecal vomiting, combined with the passage of flatus and feces, may be suggestive.

4. Differentiation.—Flatulent colic, gall- or kidney-stones and incarceration of the kidney, uterus or testis are excluded by rapid stoppage

of the bowels. Acute pancreatitis must be considered. Peritonitis is characterized by early fever, tenderness, meteorism, cessation of peristalsis, abdominal rigidity, fluid in the peritoneum and late collapse, fecal vomiting, later and less complete obstruction.

Treatment.—In *stenosis*, typified by ulcer cicatrices, a concentrated fluid or semifluid *diet* is given and thorough mastication of solid food and avoidance of food chaff, skins, stone or gristle should be insisted on. *Cathartics* are inferior to enemata of oil.

Thiosinamin, recommended to dissolve cicatricial stenosis, is given in 10-drop doses, every other day, hypodermically (1 to 6 of alcohol).

Operative measures may be instituted for foreign bodies or malignancy; the outlook is best in lesions of the colon and ileum.

1. Medical Treatment of Obstruction.—In every case the physician at once should divide responsibility with a surgeon. In acute cases food causes aggravation. Early gastric lavage affords transitory relief. Chronic *fecal obstruction* should be eliminated by colonic flushings, given with the body inverted; hard, desiccated feces must be dug out with the finger, with enemata to bring away fragments. High enemata, colonic inflation with air, laxatives, massage and puncture of the gut with an exploring needle, are always contra-indicated. *Opium* is useful only in the early stage to relieve shock. *Stimulants* are frequently indicated. *Atropine*, eserine and physostigmine sometimes help.

2. Operative Treatment.—Medical treatment of strangulation, intussusception or volvulus is of little value. A most convincing statistical argument for *early operation* is Naunyn's series of 288 cases, in which 75 per cent. of recoveries were obtained by operation in the first two days, while each successive day lowered the patient's chances to 40 per cent., 30 per cent., etc. In 1074 recent cases, Wiltner reports recovery in 53 per cent. In 100 cases of intussusception in children 63 per cent. of operated recoveries are reported by Clubbe; 40 per cent. of adults and 80 per cent. of children die under medical treatment. Fifty-two per cent. of obstruction by gall-stones recover under expectant and 33 per cent. under surgical treatment.

INTESTINAL TUMORS.

Carcinoma, the only neoplasm of clinical importance, constitutes 22 per cent. of all cancers. Previous ulceration is predisposing. It is most frequent in males over forty years of age; 17 per cent. occur between thirty and forty, and 14 per cent. under thirty years of age.

Pathology.—According to Leube, 80 per cent. occur in the rectum, 15 per cent. in the cecum and colon and 5 per cent. in the small intestine. Hausmann, in 268 cancers of the bowel (excluding the rectum), found 91 per cent. in the large and 9 per cent. in the small bowel; if figured more in detail, 37 per cent. occur in the sigmoid, 18 per cent. in the cecum, 11 in the descending colon, 9 in the transverse and 8 per cent. in the ascending colon, 8 per cent. in the ileum, 4 in the splenic and 3 in the hepatic flexure and 2 per cent. in the jejunum. The sigmoid, splenic and hepatic flexures are especially predisposed, probably because more

fixed and exposed to irritation. It is usually primary, sometimes secondary by contiguity, but very rarely metastatic. Originating in the intestinal glands, it is usually annular in form and causes stenosis and ultimate occlusion of the bowel; it is less often nodular and very rarely diffuse and flat. Histologically it may be an adenocarcinoma (especially in the duodenum), scirrhus (in the sigmoid), medullary, cylindrical-celled (in the cecum and sigmoid), colloid or flat-celled (in the rectum). Beginning in the mucosa, early involvement of the other coats and lymph vessels is followed by later involvement of the peritoneum and metastasis, especially in the liver. McWilliams finds in the literature 105 appendiceal cancers, but many reports are indecisive.

Symptoms.—1. Disturbance of the Intestinal Function.—(*a*) There is usually *constipation*, resulting from narrowing of the intestinal lumen; it is most marked in tumors of the large intestine. Ulceration frequently causes diarrhea. (*b*) The *stools* are irregular and band-like or *sheep-like*, to which, however, undue importance has been attached. (*c*) *Blood*, *mucus* and *pus* are common in the feces, though equally frequent in other ulcerations and in enteritis. The evacuations may be stinking. (*d*) Fragments of tissue are uncommon and are diagnostic only when showing carcinomatous arrangement of the cells. (*e*) *Pain*, due to enterostenosis, is usual; it may be sacral when due to sigmoid cancer. (*f*) Sudden complete *occlusion* of the bowel may follow previous gradual narrowing of the lumen. (*g*) The lower the tumor in the intestinal tract the greater is the *tympany* and *fecal retention* and the purer the pus and blood (*i. e.*, the less mixed with the feces). Above the stricture the bowel is dilated and its muscle is hypertrophied (*visible peristalsis*). (*h*) Bismuth, given by mouth and by rectum may localize the digitated markings of the stenosis in *x*-ray plates.

2. Cachexia and marasmus are most suggestive.

3. Tumor.—A tumor is best felt if the bowel is empty; it may be confused with a normal contracted colon, the edge of the ileopsoas or with fecal masses; it is irregularly oval in clear cases; annular forms escape detection, for they merely constrict the bowel. It is usually tender and is movable or immovable accordingly as the bowel involved is naturally free or fixed, or is adherent to other structures by adhesions or extension by contiguity. Whether free or fixed, the tumor is generally below the navel. Gas and fluid are sometimes felt or heard to escape through the stenotic point and distention of the large bowel by introduction of air or fluid *per rectum* may aid in localization. If the tumor grows from movable gut it may disappear and reappear from time to time. A rectal and vaginal examination always should be made.

4. Complications.—The malignancy of primarily ambiguous symptoms may be declared by (*a*) icterus, especially in duodenal cancer; (*b*) hemorrhage from the bowels; (*c*) rupture into the bladder or vagina or into the stomach, with fecal vomiting and diarrhea from partially digested food entering the colon.

Diagnosis.—Diagnosis is often difficult, except in rectal localization, for many symptoms indicate stenosis without declaring its nature. Differentiation is required from:

1. Movable Tumors.—(*a*) These may be pyloric, requiring differentiation from duodenal cancers. (*b*) The corset liver, movable spleen and kidney. (*c*) Omental and mesenteric tumors produce less enterostenosis. (*d*) Scybala may coexist with malignancy.

2. Immovable Tumors.—(*a*) Renal and retroperitoneal tumors lie beyond the colon, on its inflation, and rarely cause the same degree of obstruction. (*b*) Encapsulated peritoneal exudate; the author has seen chronic appendicitic abscesses in individuals over sixty years of age diagnosticated as tumor. (*c*) Ovarian or uterine tumors are less obstructive. (*d*) Localized inflammatory infiltration of the intestinal wall and circumscribed inflammation of the sigmoid flexure and colon.

Rectal cancer demands special consideration. It is usually annular and when developed is rigid, frequently resembles an inverted funnel, becoming narrow upward. The colloid form extends upward and downward, is often tubular and causes much regional invasion. Digital or proctoscopic examination—unaccountably neglected more often than any other simple method of physical examination—saves many lamentable errors. The early stricture may yield when ulceration develops, with discharge of blood, often ichorous pus and sometimes malignant tissue. Tenesmus, piles, morning diarrhea, sacral pain, and abdominal pain before and during the movement are common. Cachexia usually develops with great rapidity; death may result from acute obstruction with preservation of color, nutrition and energy. Intoxication symptoms, as stupor, are frequent.

Syphilitic strictures are slower and subject to greater variations; tuberculous ulcers are flatter, softer and more undermined.

Sarcoma, of which Corner and Fairbanks collected 175 cases involving the small bowel, is more rapid and seldom stenoses the lumen. Hellström found 45 lipomata and Stetten 74 myomata recorded.

Prognosis.—Marasmus, coma, anasarca, thrombosis, secondary infections, stenosis or perforation causes death.

Treatment.—Treatment is that of enterostenosis, in regard to diet. Opium must be used toward the end. Operation, either palliative enterostomy, colostomy or curative enterectomy, may be indicated. Hochenegg (1902) in 194 radical operations, reports a mortality of but 12 per cent. and estimates his recoveries at 16 per cent.

ENTEROPTOSIS.

Enteroptosis is not a disease, but only a condition in which the abdominal viscera prolapse in the abdomen, as the stomach (gastroptosis), intestine (enteroptosis), colon (coloptosis) or solid viscera (splanchnoptosis), as the liver, kidney or spleen. The condition, first described by Morgagni, is known as Glénard's disease. Whatever causes relaxation of the abdominal wall or of the intra-abdominal suspensory structures may induce prolapse, as trauma, coughing, loss of weight, pregnancy, tumors of the liver, spleen or kidney, spinal deformity and corsets. Seventy-five per cent. of the cases occur in women. Stiller maintains that splanchnoptosis is a congenital asthenic condition whose most

frequent stigmata are a floating tenth rib (*costa fluctuans decima*), found in 80 per cent. of cases, and a *habitus enteroptoticus*, somewhat resembling the *habitus phthisicus;* in the normal chest the vertical line drawn from the ensiform to the navel is short, compared with the distance from the median line, horizontally, to the tips of the floating ribs; in the *habitus enteroptoticus* the vertical line is much longer and the horizontal line is shorter than normal and shorter compared with the vertical line.

Symptoms.—1. Stomach Findings.—Gastroptosis is characterized by falling of the organ to a lower point in the abdomen, whereby both its upper and lower limits are abnormally low, the epigastrium is flattened and the hypogastrium is distended. Eichhorst finds it in 35 per cent. of women and in 5 per cent. of men. Dilatation of the stomach is excluded by finding the lesser curvature at a lower point than normal. Gastric symptoms may be noted, as pain in the left hypochondrium from tension and increased by heavy meals, eructations, nausea and gastric or intestinal hyperesthesia, or as complications, vomiting, hyperacidity or motor insufficiency. In many cases gastric and other symptoms are entirely lacking. Radiographs demonstrate the ptosis, showing sagging of the middle portion of the stomach and pyloroptosis.

2. Intestinal Findings.—Enteroptosis or coloptosis is frequently found with gastroptosis. The transverse colon sinks in its central segment so as to assume the form of the letter U or V, as shown by radiography. Constipation is frequent.

3. Floating kidney, liver and spleen will be described separately.

4. Nervous symptoms occur in 50 per cent. of cases. Many subjects are nervous, slight and delicate women, who exhibit depression, cephalic pressure, backache, throbbing of the abdominal aorta, emaciation, constipation and anemia.

Treatment.—(*a*) The treatment outlined under *Neurasthenia* (*q. v.*) is indicated. The diagnosis should not be disclosed to the patient, for suggestion initiates a long train of additional symptoms. (*b*) *Increase in weight* is effected by full feeding and the rest cure. (*c*) *Supports* to press upward and inward and maintain the viscera in their normal location often give considerable relief. *Operations* shortening the mesentery, resecting parts of the recti, suture of the liver, stomach and other organs to fixed tissues can as yet receive no general commendation.

DILATATION OF THE COLON.

Colectasia may be classified as follows: (*a*) *Gaseous distention,* which may occur in acute toxemias; death resulted from heart paralysis (*v.* Typhoid and Enteritis). (*b*) Cases which are due to *obstruction* by feces, foreign bodies, volvulus. (*c*) Hirschsprung's disease or *idiopathic colectasia,* which occurs in children (in 80 per cent. of cases) as a congenital lesion or in male adults over fifty years. It is due to obstruction, spasm, abnormal valves or long colon; in Formad's case there was great hypertrophy of the muscularis; the circumference of the colon was 30 inches and its weight, with the contained feces, was 47 pounds. The leading symptoms are obstinate constipation and meteorism. Of

223 reported cases about 25 per cent. recovered under medical treatment and 57 per cent. after resection. Death results from marasmus, auto-intoxication, obstruction, perforation or peritonitis.

INTESTINAL HEMORRHAGE.

Etiology.—There are five groups: (*a*) *Abnormal intestinal contents,* as hard feces, foreign bodies, gall-stones, caustic poisons or parasites. (*b*) *Intestinal lesions,* as the various ulcerations, inflammation, invagination, etc. (*c*) *General affections*; stasis, amyloidosis, mesenteric infarction, aneurysmal rupture, scurvy, uremia and cholemia. (*d*) *Infections,* including typhoid, dysentery, hemorrhagic exanthemata and sepsis (see HEMORRHAGIC DISEASES OF THE NEWBORN). (*e*) *Blood from the stomach.*

Symptoms.—(*a*) *Blood in the feces* may be *manifest,* or "*occult,*" *i. e.,* found only microscopically, chemically or spectroscopically (generally present hours before it is seen by the naked eye). It is mixed with feces and dark, tarry and offensive when it comes from the small gut; it may coat the movements and be bright in color when it comes from the lower intestine. (*b*) *Acute anemia* follows profuse hemorrhage, evidenced by pallor, syncope and slight fever.

Diagnosis.—The diagnosis of enterorrhagia concerns (*a*) the detection of *blood,* which may be simulated by bismuth or blueberries; (*b*) the question whether the blood comes from the bowel, mouth or stomach; (*c*) whether it comes from the large or small gut (*v. s.*); and (*d*) its cause, most often hemorrhoids, typhoid, dysentery or cancer of the lower bowel. No extended examination of the abdomen is permissible at the time of the hemorrhage.

Prognosis and Treatment.—The prognosis and treatment are involved in the basic lesion (*v.* page 55).

PILES.

Etiology.—Hemorrhoids result from (*a*) *stasis;* portal stasis operates through the superior hemorrhoidal and inferior mesenteric veins and is more potent than cardiac stasis, which congests the middle hemorrhoidal and internal iliac veins; (*b*) from *constipation,* pregnancy and obstructive lesions, as cancer of the rectum. Vigorous contraction of the abdominal muscles is a more potent cause than constipation (Duret). Usually spoken of as varices, piles are cavernous angiomata.

Symptoms.—*External piles* appear as tender, painful, purple swellings outside the sphincter ani, singly or in a circle around the anus; they may be soft or hard. The pain is throbbing and often severe. Itching is very common. They may bleed, atrophy, ulcerate or suppurate. Piles which bleed are called "open" and those which do not, "blind." *Internal piles* occur above the sphincter, below which they may prolapse and strangulate. Tenesmus, mucus secretion and dull pain sometimes referred to the back and the sacro-iliac articulation, may be noted.

Diagnosis.—Inspection excludes prolapse, condylomata, papillomata, etc. Causal carcinoma should be borne in mind.

Treatment.—(*a*) *General* factors, sedentary habits, and constipation are considered. (*b*) *Local* treatment: a small *injection of cold water* before each movement softens the irritating hard passage, constricts the piles and favors their reduction. *Cleanliness* relieves itching, for which also a 1 per cent. phenol salve is of value. The following formula is astringent and anodyne, but must be given with reserve. (*c*) *Surgical* treatment is indicated when local measures fail. Boas lauds Bier's stasis, till edema appears; atrophy ensues.

℞—Extracti opii	gr. ij
Extracti belladonnæ	gr. ij
Cocainæ (alkaloid)	gr. iij
Acidi tannici	ʒss
Olei theobromatis	q. s.

M. et ft. in suppositoria no. x.
S.—One at bedtime.

DIARRHEA.

Definition.—Increased frequency of the stools with decreased consistence. Diarrhea is not synonymous with intestinal catarrh. In some individuals two or three movements a day are physiological.

Etiology.—(*a*) *Irritation;* cathartics (some, as the aromatics, increase peristalsis and others, as salts, cause increased transudation); diarrhea dyspeptica; diarrhea stercoralis; diarrhea entozoica. In general there are *two mechanisms:* (i) the stools are hurried through the bowel by increased peristalsis, in which case bile is found in them; and (ii) there is increased serum transudation into the intestine when little or no bile is found. (*b*) *Nervous diarrhea* causes from two to fifteen thin movements, containing no increased mucus; it is attended by peristaltic unrest and is chiefly observed after excitement or in neurotics; some cases are reflex, as from uterine irritation. (*c*) *Toxic substances* in the blood may irritate the ganglia or brain, *e. g.*, in uremia, typhoid or sepsis.

Treatment.—Opium is the most valuable remedy, except in stercoral and dyspeptic types; it acts less by checking secretion than by suspending peristalsis and mitigating spasm; the crude drug is superior to its alkaloids, as its absorption from the bowel is slower and its effect more marked; ¾ of a grain of the extract or a dram of the camphorated tincture should be given with bismuth in massive doses as in enteritis. Important adjuncts are *local heat, rest* and a *spare diet,* as in enteritis. In nervous diarrhea two drops of Fowler's solution before meals is advantageous.

CONSTIPATION.

Constipation is insufficient or unsatisfactory defecation.

Etiology.—(1) "*Physiological*" constipation results from irregularity in going to stool, as in travel, from obesity, insufficient exercise or water, loss of fluid by sweating, a dietary rich in proteids, as milk, and from overmedication. (2) Constipation may be *symptomatic of other affec-*

tions, as *gastric* ulcer, cancer, dilatation or hyperacidity; *intestinal* obstruction, cancer, adhesions, catarrh, passive congestion or enteroptosis; *pelvic* conditions, as pregnancy, ovarian or uterine disease or enlarged prostate; *infections* which stimulate the inhibitory nerves; *nervous* disease, as meningitis, tumors, myelitis, the neuroses; diabetes and old age. (3) *Habitual constipation* may be familial. It may result from insufficient nervous energy in the large intestine; atony of the intestinal muscle may result from general debility, neurasthenia, etc., but seldom from atrophy or degeneration. Nervous constipation may be due to a spastic condition of the bowels.

Symptoms.—Symptoms other than constipation are often absent, and when present are less often general than *local* (anorexia, coated tongue, bad taste in the mouth or abdominal uneasiness).

Two or three movements in one day or a movement every two or three days come within physiological limits. The stools normally accumulate in the sigmoid and pass each day into the rectum, causing a desire to defecate, usually at the same hour. The constipated stool accumulates in the haustra of the colon or ampullæ of the rectum. It is difficult to state the quantity of the normal passage, which does not depend wholly on the amount of food ingested; feces consist largely of mucus and bacteria and form even during starvation. The stools may resemble sheep's dung, probably due to intestinal muscular spasm. Retained feces are usually voided spontaneously in a few days with a serous diarrhea or as scybala covered with mucus which results from irritation. *Fecal colic* is due to peristalsis above the obstruction; there may be *stercoral fever* and *meteorism.* Marked obstruction rarely simulates enterostenosis, being marked by collapse, vomiting and great vesical or rectal *tenesmus,* which should always excite suspicion. Neglected cases may result fatally, especially in the old, debilitated or insane or when defecation causes great pain, as from fistula or piles.

Fecal concrements (coproliths) occur in the large intestine, even simulating tumors of the intestine, omentum, kidney or spleen, from which differentiation may be possible only after thorough purging and flushing. They may be soft and easily indented with the finger, or hard, nodular and tender from the bowel ulceration they incite. Fecal tumors may develop with daily, though inadequate, movements. On pressure the bowel may at first seem to adhere to the fecal masses and then become free (Gersuny's sign).

General symptoms are uncommon. Nervous symptoms are causal rather than coprostatic, as in the neurasthenic who believes he would be well if his bowels would move and who has most unpleasant sensations when they do. Vertigo, pressure in the head and mild dyspnea are always neurotic. Copropsychiatry is grossly exaggerated. *Intoxication* is rare, but has been considered causal of neuralgia, marasmus, chlorosis and numerous other concomitant conditions. The aromatic sulphates of the urine may be increased, due to formation of indol, skatol and pyrocatechin.

Lane's Kink. Lane places great stress upon the development of accessory peritoneal bands, which produce kinks at the pylorus, lower

ileum, hepatic and splenic flexures and sigmoid. It is claimed that profound intestinal toxemia and constipation result, which induce dilatation of the duodenum, vomiting and even gall-stones, gastric ulcers, changes in the breasts, arthritic changes and tuberculosis. To avert these disasters, he recommends ileosigmoidostomy or excision of the colon.

The *x*-rays determine the rate of passage of bismuth; leaving the stomach, it should reach the cecum in four hours, the hepatic flexure in six and the splenic flexure in nine hours; visceroptosis and kinks are apparent; intestinal constipation and dyschesia (difficult evacuation of the rectum) are differentiated (*v. i.*).

Treatment.—Treatment of causal factors (*v. s.*) is important.

1. Diet.—(*a*) *Coarse foods,* as whole wheat, Graham, rye, cornmeal and bran breads, act less mechanically than chemically by the production of lactic or oleic acid. (*b*) *Fruits* relax the bowels, because of the malic, tartaric and citric acids they contain, as plums, raisins, apples, peaches, pears and prunes; grapes sometimes and bananas and persimmons usually cause constipation. Fruits operate best when eaten at bedtime; it may be best to remove the excess of seeds or skins. (*c*) *Vegetables* act by the acids and gases they form; melons, sprouts, cabbage, cauliflower, cucumbers, turnips, carrots, spinach, tomatoes, asparagus, onions, cress, celery and squash should be given freely; excessive amounts may cause fatigue of the bowel, fermentation and constipation. Agar-agar, ʒij daily, may be given in mashed potatoes or apple sauce. (*d*) *Sweets* are laxative; if they do not cause indigestion, sugars, syrup, candy, sauces, molasses, jellies, jams, marmalades and honey are valuable laxatives. (*e*) *Fats.*—Moderate amounts of butter and olive oil are superior to animal fats, which tend to derange digestion. (*f*) *Cold* or *charged water,* taken on rising and through the day, excites peristalsis. Hard water constipates. (*g*) The following are *to be avoided:* red wines (containing tannic acid), excess of meat, eggs or rice, buckwheat, macaroni and other carbohydrates containing little water, tea, milk (sometimes laxative when not taken in sips but swallowed in larger quantities), chocolate and cocoa. The diet recommended affords material for bacterial action, which follows the idea advanced by Schmidt, Strassburger and Lohrisch, that constipation results from too complete utilization of the average diet by the intestinal bacteria. Some individuals digest all kinds of vegetables; others pass unchanged even cooked vegetables. Hydrochloric acid partly digests and loosens the middle sheath of the cellulose, so that the duodenal and pancreatic juices operate more readily on it. This explains the constipation of hyperchlorhydria. *Diet and copious drinking of water* cure most cases.

2. Punctuality at Stool.—The best time is immediately after breakfast; piles or prolapse may come down during the day, whence in these cases the bowels should be moved at bedtime.

3. Posture at Stool.—Squatting, the natural posture, may be approximated by leaning forward or by placing the feet on a foot-stool; the thighs support and compress the abdomen, a matter of importance when it is relaxed.

4. Massage.—Gentle massage along the course of the colon for a quarter of an hour, before breakfast, may give relief if continued for months. Sahli rolled over the abdomen a 7-pound cannon-ball. Massage is contra-indicated in spastic constipation.

5. Mild Faradization.—Weak applications arouse short contractions in the colon and are given with a moist sponge. The cold compress to the abdomen is a good adjunct.

6. Exercise.—Exercise to develop and contract the abdominal muscles, as in tennis or rowing or by lifting the legs while lying on the back or lifting the rigid body from the lying to the erect posture solely by the abdominal muscles, is superior to walking, bicycle and horse-back riding. In ovarian and uterine disease exercise increases constipation, due to reflex inhibition of peristalsis, as shown by the laxative effect of narcotics. It is claimed that constipation is *spastic* (not atonic) in 25 per cent. of female cases, as evidenced by colic, palpable or visible knots in the intestine and sometimes by ribbon-like passages; in these cases an irritating, coarse diet, massage, cold and electricity are to be avoided. The radiograph may show the spastic sigmoid or colonic flexures, etc. Hot baths or hot compresses to the abdomen for colic are indicated; an enema of warm oil is given each evening; extract of belladonna gr $\frac{1}{3}$ in a suppository relieves spasm and tenesmus; the diet should consist of fine carbohydrates, butter and cooked fruit.

7. Enemata.—Enemata excite peristalsis, lessen intestinal hyperemia and apparently benefit the hepatic circulation. They do not derange digestion as do cathartics, but may lose their effect by dilating the colon, whence the indication for their intermittent use. Water and olive or linseed oil may be used; oil enemata (five to eight ounces) are especially indicated in spastic constipation and colica mucosa, in which the oil may be left in the bowel over night. Antiperistaltic movements in the colon carry the oil to the cecum, in the low as well as in the "high" enema. In ordinary constipation, the passage of feces through the intestines is delayed, while defecation is normal; in *dyschesia*, there is no delay in the arrival of feces in the pelvic colon, though their final expulsion is not adequately performed; diet, abdominal massage and aperients, appropriate for intestinal constipation, are useless in dyschesia, attention to the hygiene of the bowels and reëducation of the defecation reflex by means of graduated enemata being the correct treatment. Dyschesia leads to secondary retention of feces in the pelvic colon, and in severe cases in still higher parts of the large intestine; unless enemata are given, the rectum is never empty, and in spite of its dilated condition there is insufficient room for all the retained feces. Glycerin suppositories are valuable, if used alternately with other methods. Fecal impaction indicates enemata, cathartics and digital evacuation of the hardened masses.

8. Cathartics.—(*a*) Cathartics should be administered only after failure of the measures enumerated above; (*b*) their most successful employment is intermittent and alternating; (*c*) drastics, hydragogues and cathartics producing secondary constipation are avoided, save in extreme cases. Every new remedy is the best for a short while.

Varieties.—Aloes, found in nearly all proprietary and official pills, should be avoided in piles and uterine hemorrhage, because it congests the pelvic vessels; it is given in combination, *e. g.*—

℞—Extr. aloes	gr. ss	Acting in 15 to 20 hours, on the large bowel chiefly, increasing tonus.
Extr. rhei	gr. ij–v	Actively purgative in 4 to 8 hours; stomachic and tonic.
Extr. nucis vomicæ	gr. $\frac{1}{4}$	Acting on peristalsis and combating atony.
Resinæ podophylli	gr. $\frac{1}{10}$	Acting in 10 hours, producing "bilious stools."
Extr. belladonnæ (or extr. hyoscyami)	gr. $\frac{1}{4}$	Relieving spasm (griping) and operating on the intestinal nerves.
Extr. taraxaci	gr. j	
M. et ft. pil. i.		

Rhubarb gripes and produces secondary constipation, caused by its tannic acid, whence it should never be given alone:

℞—Pulveris rhei compositi	ʒiv
Sodii sulphatis	ʒiv
Sodii bicarbonatis	ʒj
M. et S.—One teaspoonful at bedtime.	

Cascara sagrada improves digestion and produces little constipation afterward; the fluidextractum rhamni purshianæ, ♏xv, is very bitter; the extractum is given in doses of from two to eight grains. *Licorice* is given as pulvis glycyrrhizæ compositus, ʒss–j. *Castor oil* is a soothing laxative, relieving irritation and spasm, in nervous or lead constipation, in the latter combined with opium gr. $\frac{1}{4}$; its offensive taste may be partly overcome as follows:

℞—Olei ricini	ʒij
Olei gaultheriæ	gtt. xl
Glycerini	ʒij
M. et S.—One teaspoonful to one tablespoonful.	

In anemic patients the following formula is recommended:

℞—Ferri sulphatis	gr. x
Extracti aloes	gr. v
Extracti rhamni purshianæ	gr. xx
Extracti belladonnæ	gr. iij
Extracti nucis vomicæ	gr. iij
M. et ft. pilulæ no. x.	
S.—One after meals.	

Calomel should be given in the evening in fractional doses, combined with podophyllin, which also acts in eight hours, and followed the next morning by half an ounce of magnesium sulphate; calomel increases the peristalsis of the large and small intestines. *Senna* easily deranges digestion; it acts upon the large bowel; a small amount may be cooked with prunes; *confectio sennæ* is given in dram doses. Cathartics often cause blood in the stools. *Tobacco smoking and coffee* in the morning are beneficial, but tea promotes flatulency and constipation. Consideration of the salines and drastics (*v.* Nephritis) is omitted, as their use is contra-

indicated except for emergencies; table salt, ℥ss, well diluted and taken on rising frequently moves the bowels. *Hormonal*, a peristalsis hormone discovered by Zuelzer in the intestines, spleen, etc., promotes peristalsis, given intramuscularly or intravenously; it is prone to lower blood-pressure, even to the point of collapse.

INTESTINAL DIVERTICULA.

Telling tabulated 105 cases (1908). They may be congenital or acquired, false or true, in type. They may be the site of inflammation or malignancy. Occurring in the colon or sigmoid, the symptoms may resemble those of left-sided appendicitis. Infection, ulceration or gangrene may lead to peritoneal disasters. Pain, leukocytosis, fever, vomiting, diarrhea or constipation, blood in the stools and a low, perhaps pelvic, tumor which appears and disappears, are suggestive symptoms.

NERVOUS AFFECTIONS OF THE BOWEL.

1. Neuroses of Motility.—(*a*) *Nervous diarrhea* has been considered. It may occur in the neuroses, reflex irritation during dentition or in the crises of tabes; it is usually transient and is marked by absence of inflammatory symptoms, as mucus, pus or blood. Nervous diarrhea may be confused with organic disease. (*b*) *Enterospasm* is caused by simultaneous contraction of the circular and longitudinal muscles, which normally contract alternately. (See Dynamic Ileus, Spastic Constipation and Colica Mucosa.) (*c*) *Sphincter spasm*, aside from local rectal or anal lesions, results from sensory hyperirritability or decreased cerebral inhibition; it is most common in hysteria and tabes. (*d*) *Peristaltic unrest* may be associated with diarrhea if the colon is involved, but more commonly affects the small bowel only, when constipation or normal movements are usual. (*e*) *Nervous constipation* (atony) may result from the neuroses, intoxication from carbon dioxide and organic cerebrospinal diseases, in which conditions (*f*) *sphincter paralysis* may also result.

2. Neuroses of Sensation.—Neuralgia mesenterica (*enteralgia*, enterodynia) is most important; strictly speaking, it is always nervous, *i. e.*, not due to organic causes; practically, other forms of abdominal pain are frequently included under enteralgia. The chief symptom is the abdominal pain, which is usually umbilical, sometimes relieved by pressure and sometimes not; the abdominal wall may be hyperesthetic. Reflex disturbances in other organs may aid in establishing its functional character, as hiccough, vomiting, dyspnea, palpitation, rectal or vesical tenesmus, muscular twitchings, etc. The *diagnosis* necessitates exclusion of innumerable affections, chiefly abdominal but also general: (*a*) rheumatism of the abdominal muscles which is superficial; (*b*) lumbo-abdominal neuralgia, in which Valleix's three tender points are found (see Neuralgia); (*c*) hysteria, distinguishable by its stigmata; (*d*) tabetic crises; (*e*) intestinal affections, as appendicitis, peritonitis, coprostasis, para-

sites, flatulent colic, excessive catharsis, lead colic or colica mucosa; (*f*) *gout;* malaria and typhoid; renal and bilary calculi.

Its *treatment* and that of the motor disturbances is of the fundamental nervous state. Symptomatically, hypodermics of narcotics produce the quickest but most dangerous relief; atropine is much safer.

℞—Spiritus chloroformi . . . . . ʒij
Tincturæ opii camphoratæ . . . . . ʒiv
Tincturæ asafœtidæ . . . . . ʒiv
M. et S.—One tablespoonful in hot water every fifteen minutes for three or four doses.

3. Neuroses of Secretion.—The secretion neuroses are important. The chief types are serous nervous diarrhea and colica mucosa (*v. s.*).

AFFECTIONS OF THE MESENTERY.

I. Inflammation.—Mesenteritis is chiefly important in its relation to ascites, tuberculous peritonitis and other peritonitides (*q. v.*).

II. Hemorrhage.—Primary hemorrhage is most rare. It is generally secondary to acute hemorrhagic pancreatitis, retroperitoneal hematoma, aneurysmal extravasation or the hemorrhagic fevers.

III. Diseases of the Mesenteric Vessels.—(*a*) *Infarction* by embolism or thrombosis involves chiefly the arteria mesenterica superior, essentially a terminal vessel. Most cases develop in men past middle life; 360 cases are reported. Its onset is sudden, with intense abdominal pain, collapse, vomiting (usually bloody and perhaps fecal, due to intestinal obstruction), diarrhea (40 per cent.) and bloody stools (40 per cent.). Unless there is an apparent cause for embolism, as valvular disease, the diagnosis is rarely made, for peritonitis or obstruction is usually suspected. The usual outcome is early death from collapse, but if the patient lives long enough acute peritonitis sets in, with distended abdomen and diffuse tenderness. In very rare cases collateral circulation allows of recovery; 94 per cent. are fatal. The treatment is purely symptomatic and supportive, but operation was successful in 46 per cent. of Boinet's series. (*b*) *Periarteritis nodosa* (*v.* page 410). (*c*) *Dilatation* of the mesenteric veins and phlebosclerosis occur, especially in liver cirrhosis. (*d*) *Suppuration* (thrombophlebitis suppurativa) may mark umbilical pyemia of the newborn or may carry infection from the appendix or rectum. (*e*) Of simple thrombosis of the mesenteric veins, Kraft collected 16 cases; in over half of the cases syphilis or liver cirrhosis was the cause.

IV. Affections of the Chyle Vessels.—These vessels may become varicose or even hyperplastic (chylangioma). Cysts containing chyle may cause tumors and, by rupture, may result in chylous extravasation in the mesentery or chylous ascites (*q. v.*). Twenty-four chylous cysts are described by Broca.

V. Mesenteric Tumors.—These may be dermoid, hydatid, serous, sanguineous, chylous or malignant, and may fill the abdomen. Their symptoms are those of an abdominal tumor, which is centrally located, movable and covered with a zone of resonant intestine.

DISEASES OF THE LIVER.

ACUTE YELLOW ATROPHY.

Definition.—An acute degeneration of the liver cells, accompanied by shrinking of the liver, icterus and fatal nervous toxemia. Ballonius (1600) reported the first case. Best collected 500 cases.

Etiology.—(*a*) Sixty-six per cent. occur in *women* and 50 per cent. between the years of *twenty and thirty;* but 22 cases occurred under 10 years of age. (*b*) Thirty-three per cent. of women with the disease were in the *last half of pregnancy;* it is infrequent in the first three months and the puerperium. (*c*) Ten soldiers were affected in Arnould's epidemic series. (*d*) *Infections,* as osteomyelitis, diphtheria, sepsis, erysipelas, typhoid, recurrent fever and secondary syphilis (in about 50 cases); or (*e*) *poisoning,* as from phosphorus, alcohol and chloroform narcosis, may antedate the disease, which may occur primarily in a sound or secondarily in a diseased liver. Worry is a predisposing factor in pregnancy or syphilis.

Pathology.—The liver is small, the minimum record being 400 grams; it is often so lax that it can be rolled up; the capsule is wrinkled, the color yellow (*atrophia hepatis fusca*) and the lobular markings obscure. The liver cells are granular, fatty, or tinged with bile; some are necrotic and others show regeneration. Foci of red tissue represent an advanced stage in which the fatty necrotic cells have been absorbed; the left lobe is often red, homogeneous and composed of detritus, showing the most advanced process, and the right lobe is yellow, showing more recent changes. Crystals of tyrosin are found. The apparent increase of the connective tissue is relative only. Icteric staining, small hemorrhages and fatty degeneration are found in the heart, kidneys, muscles, lungs and digestive tract.

The disease is rather a necrotic process than an acute parenchymatous hepatitis. It is not determined whether the disease is an *infection,* to which it has some resemblance, or an *intoxication,* which initiates autolysis of the liver.

General Clinical Picture.—Acute yellow atrophy begins with a *prodromal stage* in which gastric disturbance predominates; icterus develops in a few days when the *second stage* suddenly begins with cerebral symptoms, delirium, vomiting and convulsions; shrinking of the liver, hepatic tenderness, enlarged spleen, hemorrhages, and abortion, if pregnancy exist; urinary symptoms, as bile, albumin, leucin, tyrosin and other unusual products in the urine; and subnormal or normal temperature. The issue is fatal.

Individual Symptoms.—The *prodromal stage* lasts a few days (to three weeks or more) and is not characteristic. In the latter half of gestation, icterus and hepatic tenderness or enlargement are suspicious symptoms. Gastric irritability prevails. Icterus, absent only in extremely rapid cases, is obstructive from intrahepatic changes and increases with the second stage; the stools are acholic.

In the second stage (*a*) the *hepatic dulness shrinks* first in the left lobe, to one-half or one-quarter of its former dimensions, or the liver falls back toward the spine so that there is no liver dulness. The liver dulness may remain normal if death is rapid, as from hematemesis, or if the liver is cirrhotic or adherent to the abdominal wall. The liver is *tender and painful*, and may feel flabby or pit to the finger. The liver region may appear sunken. (*b*) The *spleen is enlarged* in 66 per cent.; it is not enlarged when profuse hemorrhage or diarrhea is present. (*c*) *Vomiting* is *almost invariable*, the vomitus consisting of mucus, bile and finally blood; it is accompanied by hiccough, dry tongue, sordes and constipated, uncolored stools. (*d*) *Nervous symptoms* come on gradually with headache, or abruptly with *delirium*, anxiety, *meningeal symptoms*, trismus, wide pupils, amaurosis, cerebral vomiting and convulsions (in 33 per cent. of adults and almost constantly in children). The nervous symptoms are due to hepatic insufficiency (hepatargia), the degenerated liver being unable to protect the nervous system, as it does in health, against various toxic products. (*e*) The *urine is decreased* or even suppressed. *Bile pigment, albumin*, casts and epithelial cells are found. Unusual products are found in the urine, due to the *autolysis of the liver cells; leucin and tyrosin* occur most frequently, though neither constant nor pathognomonic; sarcolactic acid, oxyamygdalic acid, peptone and albumose are also found. The urea is decreased, perhaps absent. The ammonium compounds, normally 2 to 5 per cent. of the nitrogen excretion, are increased, even to 17 to 20 per cent., to neutralize the acidosis. The uric acid and xanthin are increased. Indicanuria may be noted. Glycosuria is rare, though the glycogenic function of the liver can hardly be normal. (*f*) The *temperature* is usually *normal* or subnormal at the height of the process. The *pulse* at first may be slow, but later becomes *very rapid and dyspnea* develops. (*g*) *Hemorrhages* occur in the majority of cases, largely from the stomach and into the skin. Hemorrhages from the bowel, nose, mouth and genitals are less frequent. Uterine hemorrhages occur if pregnancy exist. Hemorrhages in the retina are frequent, together with white flecks due to tyrosin deposit and fatty change. Erythematous eruptions, muscular pain, articular swellings and herpes may occur.

Course.—Fifty per cent. of cases die between the fifth and fourteenth day and 30 per cent. between the third and fifth week. The more abrupt and severe the cerebral symptoms the more rapid is the course. Pregnancy hastens the issue. Remissions may be observed. The pulse becomes rapid, the breathing difficult and irregular and nervous excitation is followed by paralysis of the brain centres.

Diagnosis.—Diagnosis is based upon the *icterus, nervous symptoms* and *shrinkage of the liver*. Diminution of the liver may be simulated by tympanites or by the colon lying over the liver. The urinary findings are valuable with the above symptoms. Confusion may occur in *icterus gravis* or in the so-called bilious typhoid, pneumonia, recurrent fever, puerperal fever, yellow fever, or Weil's disease, most of which affections are febrile, while yellow atrophy is afebrile; the intensity of the icterus, shrinking of the liver, severe cerebral symptoms and the urinary findings are diagnostic. The greatest difficulty in diagnosis is *acute phosphorus poisoning*, for which the etiology alone is distinctive.

Recovery usually indicates error in diagnosis. Ascites in acute yellow atrophy occurred in but eight instances (Tileston) (*v.* page 587).

Treatment.—Treatment is directed to the heart depression, vomiting and nervous excitement (*v.* Typhoid). Two recoveries under salvarsan are alleged.

PORTAL CIRRHOSIS.

Synonyms.—Laennec's cirrhosis, alcoholic or atrophic cirrhosis.

Definition.—Portal cirrhosis is a fibrosis of the liver, characterized (*a*) etiologically by alcoholism; (*b*) anatomically by induration around the portal vein radicles enclosing numbers of liver lobules, whose cells degenerate; and (*c*) clinically by portal obstruction (dyspepsia, hematemesis, enlarged spleen and ascites) and often by signs of hepatic insufficiency.

Classification.—Cirrhosis may be classified as follows:

1. Capsular or Glissonian.	Portal vein syphilis. Chronic perihepatitis.
2. Vascular.	*Portal vein* cirrhosis, first in clinical importance; its leading type is the alcoholic, atrophic type of Laennec. (A subtype is a hypertrophic cirrhosis, like the atrophic save that the liver remains large.) *Hepatic vein* cirrhosis, occurring in stasis (cyanotic induration), and, according to French authors, with portal vein cirrhosis.
3. Biliary Cirrhosis.	Hanot's disease.
4. Mixed Cirrhosis.	(Portal vein and biliary cirrhoses combined.)

Of these, two main forms are conspicuous—the portal and biliary.

Etiology.—(*a*) Sixty to 75 per cent. of cases are found in *men* between *forty and fifty years* of age. Musser collected 685 cases in children. (*b*) *Alcoholism* is the chief cause; cirrhosis may be due to whisky, wine, beer or absinthe, especially in sedentary individuals; alcohol is absorbed by the gastro-intestinal veins and conveyed to the portal vein radicles, where its toxic effects are expended on the liver cells or interstitial tissue, but in what sequence it is not known. (*c*) *Syphilis* is a cause, especially in the young, in whom the spirochetes are conveyed by the umbilical vein to the liver. (*d*) *Other infections*, as malaria, typhoid, tuberculosis, etc., are possible causes; experimentally cirrhosis has been induced by the Bacillus pyocyaneus, B. coli and staphylococcus; and lactic, acetic and butyric acids; local mycotic necrosis is thought to be followed by fibrosis, the toxins probably entering by the hepatic artery. (*e*) *Unknown toxemias.* Spices, drastics, lead, phosphorus, arsenic, hemochromatosis, anthracosis, silicosis, repeated protein intoxication and possibly gastro-enteric auto-intoxication are exceptional etiological factors. It may be associated with gout, diabetes or other constitutional affections. There may be *multiple causes, e. g.*, syphilis plus malaria or alcoholism.

Pathology.—The essential change is fibrosis around the branches of the portal vein; either the connective-tissue growth is primary, causing, as it contracts, atrophic or fatty alteration of the liver cells, sometimes

with pigmentation; or the cells are first injured and are replaced by fibrous tissue. The connective tissue surrounds several lobules at a time (*multilobular cirrhosis*). Histologically the cirrhosis is *atrophic*, and this point is emphasized first, because some clinicians maintain that the liver is first large (the so-called hypertrophic stage) and later is shrunken; and a normal-sized or enlarged cirrhotic liver is histologically atrophic. An atrophic liver may be large from fat deposit, congestion or compensatory parenchymatous hyperplasia. The *weight* is *as often increased as decreased*, ranging from 2000 gm. to 1000 gm. or less. Its *edge is rough, i. e.*, granulations can be felt which represent the remaining relatively normal liver tissue. Regeneration of the parenchymatous cells is active but somewhat disorderly, irregular and unstable, the cells again wasting. The *surface* is sometimes smooth, and cirrhosis may appear only on microscopic examination. Granulations also occur in fatty liver, pylephlebitis and passive congestion. Its *consistence is increased.* The liver is *gritty* to the knife; its color is yellow (cirrhosis), perhaps green or brown. The *connective tissue is increased* and prominent, encroaching on the parenchyma. Injections of the organ through the portal vein are not successful, *i. e.*, there is portal obstruction; there is a narrowing of the capillary vessels; these and the increased arterial pressure in the organ account for many of the clinical symptoms (*v.* page 574).

General Clinical Picture.—The disease often exists for years without symptoms; this latency is marked by enlargement of the liver, and is due to compensatory cellular hyperplasia. In the minority of cases, transient swelling, pain or active hyperemia of the liver, dyspepsia and fever or icterus are observed; they are not characteristic. With marked shrinking there are symptoms (*a*) of *disturbance of nutrition*, as emaciation, which is often not noticed because the abdomen remains obese and a sallow skin, in which dilatation of the small arterioles (toxemia or alcoholism), dryness and hemorrhages are also observed; (*b*) of *portal congestion*, as early hematemesis, epigastric pressure, ascites, large spleen and disturbed breathing, circulation and digestion; (*c*) of *intoxication*, resulting from functional failure of the heart, liver and kidneys; and (*d*) of *complications*, as hemorrhage, diarrhea or cholemia.

Symptoms.—1. The Liver.—The liver is *enlarged as often as it is decreased in size;* the left lobe suffers greater and earlier contraction. Its *edge is hard*, but often cannot be felt, even after tapping, on account of its shrinkage or because the bowels cover it. The *edge is uneven;* its granulations may be confused with the small islets of fatty tissue in the abdominal wall, due to unequal wasting of the adipose tissue. *Palpation* of the liver is *the only certain way* of determining its lower limit, the results of percussion being unreliable. In infantile forms of portal cirrhosis the liver and spleen are larger than in adult types (*v. i.* for symptoms of impaired hepatic function).

2. Portal Stasis.—Obstruction to the portal circulation causes: (*a*) The peritoneum is thickened, edematous and the seat of transudation, *i. e.*, *ascites*, which occurs in 85 per cent. when the patient dies of the cirrhosis itself and in 50 per cent. when he dies from other causes.

It is more common in small than in large livers. *Ascites appears first without general edema,* though later the legs may become swollen as the ascites compresses the inferior cava; pre-ascitic edema of the legs or abdominal wall may develop from cardiac or renal complications, involvement of the cava by perihepatitis or from thrombosis of the iliac veins. Under ascites its physical signs will be more closely considered. The amount of fluid may be enormous (nine gallons.)

The fluid is usually amber-colored, seldom red from admixture of blood or very rarely turbid from fat (adipose ascites) or lecithin (chyliform ascites). The *specific gravity* is 1.008 to 1.015, with 0.6 to 1 per cent. albumin. The patient may die before ascites develops. A slowly developing *collateral circulation* may permanently relieve an earlier ascites. It may disappear after hemorrhage or diarrhea. (*b*) *The spleen* is *enlarged* two- to sixfold, due to the early toxemia and the later stasis, in 75 per cent. of cases; it is a fair measure of the degree of liver cirrhosis. Its capsule and trabeculæ are hard and thickened (sometimes preventing splenic enlargement), and a systolic murmur is sometimes heard. (*c*) *Gastro-intestinal catarrh* is due to stasis and alcoholism. Pain after eating, a coated tongue, vomiting, achylia, constipation, piles, diarrhea toward the end of the course and meteorism also occur. (*d*) *A collateral circulation* may be evidenced by enlargement of the veins about the navel (*cirsomphalos* or *caput Medusæ*). Prominence of the epigastric veins results from compression of the inferior cava. *Anastomoses* occur (i) between the middle and inferior hemorrhoidal veins (passing by the hypogastric to the cava) on the one hand, and the superior hemorrhoidal (passing by the superior mesenteric to the portal vein) on the other hand; (ii) between the coronary veins of the stomach on the one hand and the esophageal and phrenic veins on the other, which empty into the vena azygos; (iii) between the veins in the ligaments of the liver and new adhesions and the phrenic veins; between the veins of the *ligamentum teres* and the veins of the abdominal wall and epigastric veins, which may sometimes be felt and may produce murmurs; (iv) the mesenteric, peritoneal and parumbilical veins also communicate with those of the abdominal wall. A well-developed collateral circulation is both a favorable and an unfavorable symptom; it *relieves* the *portal stagnation* and helps the overtaxed liver to perform its functions; on the other hand, the dilated collateral vessels are likely to *rupture* and they convey directly into the general circulation *toxic substances* which the liver normally destroys or alters; this failure of detoxication causes the frequent arteriosclerosis, granular kidneys and more acute toxemia, which resembles uremia. (*e*) *The kidneys are congested,* degenerated and often show hypertrophy. The urine is decreased in amount, darker and of higher specific gravity, because the arterial tension is low in cirrhosis. More urine is often secreted during fasting than during digestion (opsiuria), which is the converse of normal conditions. The *urobilin may be increased* twofold, indicating failure of hepatic function, if there is lessened urobilin in the stools (and hemolysis is excluded). The chlorides are decreased, as they accumulate in the ascitic transudate. The urea is often decreased, but the *ammonium compounds* are often increased, less from interference

with the liver functions than from increase of acid products in the blood (acidosis), which the ammonium salts seek to neutralize. The amino-acids are regularly increased. Glycosuria is uncommon save in the pigmentary cirrhosis (see DIAGNOSIS). Alimentary glycosuria may be produced by administration of 100 gm. of grape-sugar or of levulose and galactose (in 50–86 per cent. of cases). In the writer's experience, physical signs are far more weighty than functional tests. *Albuminuria* may result from stasis or nephritis (*v. i.*). Blood may be found in the urine as a result of stasis in the bladder.

3. GENERAL SYMPTOMS.—*Loss in weight, anemia* and an *earth-like color* are explained (*a*) by the inability of the liver to handle the carbohydrates, proteids, etc., (*b*) by the diversion of toxins through the collateral circulation into the general circulation, and (*c*) stasis catarrh. In 4 cases with well-developed collateral circulation, scurvy developed which the writer interpreted as toxemic. It was once thought that bile salts entered the blood and produced "*cholemia*," or that substances were retained which the bile normally excreted, producing "*acholia*;" now *liver insufficiency* (*hepatargia*) is considered responsible for toxemic symptoms (adynamia, headache, itching, delirium, stupor, convulsions, paralysis, contractures or coma); the writer twice observed Kussmaul's breathing. Much depends upon the integrity of the kidneys, which may for a time maintain adequate excretion.

Complications.—1. HEMORRHAGE.—(*a*) *Cachectic* capillary hemorrhage from the nose, mouth, lungs or stomach is less frequent than in biliary cirrhosis. Epistaxis occurs from dilatation of the vessels on the anterior part of the septum. (*b*) Of *mechanical* congestive hemorrhages from the esophagus, stomach, bowels, kidneys, bladder or uterus, those from the *esophagus* are most important. Their rupture causes *hematemesis* (which occurs in 23 per cent. of cirrhosis cases), chiefly in adults; if very profuse, blood may come up without vomiting; one of Osler's cases bled 10 pounds; it often causes blood in the stools. It may form dark clots; it is usually attended by less collapse than in hemorrhage from gastric ulcer. In 33 per cent. the first hemorrhage is fatal; in 66 per cent. hemorrhages occur over a period of months to even eleven years. Varices are present in 80 per cent. and in over half, macroscopic rupture is detected; fatal hemorrhage may occur solely from capillary rupture. Hemorrhage may be the first symptom of cirrhosis.

2. PERITONITIS.—(*a*) *Chronic* peritonitis (15 per cent.) may occur with retraction of the mesentery and intestines; it prevents absorption of the ascites. (*b*) *Tuberculous* peritonitis (20 per cent.), often with an hemorrhagic exudate, follows the ascites of cirrhosis and hastens the fatal outcome. One would think that stasis would prevent the development of tuberculosis.

3. NEPHRITIS.—Nephritis occurs in 33 per cent. of cases.

4. HEART.—The heart is "arteriosclerotic" in 60 per cent.; chronic myocarditis, endocarditis, fatty degeneration and dilatation of the right ventricle often occur. The arterial tension is low in cirrhosis.

5. LUNGS.—Bronchitis, emphysema and in 28 per cent. pulmonary tuberculosis occur. Right-sided hydrothorax occurs from stasis in the

vena azygos and right intercostal veins; right-sided pleurisy, due to extension of perihepatitis through the diaphragm, occurs in 10 per cent. of cases.

6. Brain.—Pachymeningitis, or progressive degeneration of the corpus striatum (see diagnosis of cerebral thrombosis) occasionally occurs.

7. Fever.—"*Intermittent hepatic fever*" may result from hepatargia or perihepatitis. The temperature is usually subnormal in advanced cases, though fever and leukocytosis are observed in terminal infections.

8. Icterus.—In atrophic cirrhosis jaundice is a genuine complication, due to duodenal catarrh or a radicular cholangitis; few pass through the disease without a muddy, yellow areola under the eyes. If present it is incomplete. Its frequency is 15 per cent.

9. Anasarca.—Anasarca results from pressure on the cava by an extensive ascites, from perihepatitis with cicatricial involvement of the cava and from complicating cardiac or renal disease and marasmus.

10. Other Complications.—These are rarer, pylethrombosis, clubbed fingers, multiple neuritis or delirium tremens.

Course and Prognosis.—The clinical course seldom exceeds two or three years, but more often months than years; the maximum is fifteen years. There is no acute cirrhosis. *Compensatory factors* include (*a*) the collateral circulation, though this may throw toxins directly on the nervous centres and produce symptoms like uremia; (*b*) hyperplasia of the liver cells, which may produce veritable adenomata. As Hanot puts it, the diagnosis is based on the condition of the connective tissue, but the prognosis depends on that of the liver cells. (*c*) The kidneys may compensate to a small degree. Hale White holds that ascites is ominous, the patient rarely surviving the second tapping; though ascites is a late symptom, the author has seen numerous cases which survived frequent tappings, excluding perihepatitis, which White considers explanatory of these cases. The prognosis is relatively favorable in subjects under forty years with large livers and small spleens. Recovery is possible. In advanced stages death is the usual outcome.

Death results from hepatargia (cholemia), marasmus, hemorrhage, ascites or intercurrent terminal infections; exhausting diarrhea or vomiting; cardiac, renal or other complications (*v. s.*).

Differential Diagnosis.—Two classes of conditions require differentiation: those in which the liver is large and those in which it is small.

1. When the Liver is Large.—(*a*) The *fatty, amyloid, congested, cancerous* and *luetic livers* are differentiated on page 587. (*b*) *Leukemic* deposits cause hepatic intumescence, but the blood examination is decisive. (*c*) *Pericarditic pseudocirrhosis,* described by Pick (1896), occurs in young, rheumatic and cardiac subjects and is characterized by symptoms which on the one hand resemble disproportionately extreme passive congestion of the liver and on the other a capsular cirrhosis; the liver is large and firm, there is recurrent ascites without edema of the extremities and little or no icterus; contracting pericarditic adhesions may perhaps cause dilatation of the right auricle and vena cava inferior, thereby distending the hepatic vein system; or possibly the inflammation extends down the cava inferior to the liver. (*d*) *In Banti's disease* the spleen is

enlarged first and there is early icterus and splenic anemia, while the cirrhosis and hematemesis occur later.

2. When the Liver is Small.—The following possibilities should be considered: (*a*) *Syphilis*, characterized by stellate scars, nodes, pain and a positive Wassermann. (*b*) *Passive congestion;* the nutmeg liver may atrophy, but anasarca is observed first and there are primary characteristic heart findings. (*c*) In simple *marantic atrophy* the liver is small, there is no portal stasis and arteriosclerosis is found. (*d*) *Pylethrombosis* is characterized by a rapid onset with swelling of the spleen, severe or repeated hematemesis, dilated veins and rapid recurrence of the ascites after tapping; it is due to tumor, nearby ulcers, suppuration or gallstones. (*e*) Perihepatitis has a very much slower course than has cirrhosis and often results from pericarditis. (*f*) In *chronic peritonitis* the fluid is turbid, often hemorrhagic, with high specific gravity and many leukocytes; for differentiation see pages 615 and 616. (*g*) The peculiar *pigmentary cirrhosis* of hemochromatosis (diabète bronzé) was described by Troisier (1871), named by Recklinghausen and elaborated by Hanot, Chauffard and Letulle. Some toxic substance destroys the red blood cells (hemolysis), and so operates on the cells of the liver, pancreas and other organs that they transform the soluble blood pigment into insoluble hemosiderin and iron-free hemofuscin. These pigments are found in the muscles, intestine, heart, liver and pancreas, whose cells become pigmented, degenerated and necrotic; the pigment passes into the connective tissue of the liver, skin and pancreas, with these sequels—cutaneous pigmentation like Addison's disease; cirrhosis of the liver and of the islands of Langerhans in the pancreas, causing the diabetes, which occurred in all but five of the French cases, but less commonly in this country; 94 per cent. are males.

Treatment.—1. Causal Therapy and Prophylaxis.—These have but limited application, save in preventing further injury by syphilis and liquors or medicinal tinctures. Antisyphilitic treatment may help all forms of cirrhosis.

2. Medicinal Treatment.—Cirrhosis cases come under observation only after contracting connective tissue has formed. Naunyn and Rolleston advocate potassium iodide and ammonium chloride.

3. Diet.—Meat is tolerated not as well as skimmed milk. Rich or highly seasoned foods are to be avoided.

4. Symptomatic Treatment.—The treatment instituted in chronic gastritis or alcoholism (*q. v.*) may be indicated. Flatulence is met by calomel in fractional doses, and salol 10 grains p. c., though it may cause renal irritation. Diarrhea should not be checked until the bowels have been cleansed, for it is eliminative. Unusual splenic intumescence may be treated by purging, for it is often a forerunner of hematemesis. Hematemesis indicates absolute rest, horse serum and rectal feeding, as in gastric ulcer. Pain is controlled by local heat, calomel and salines. Nervous symptoms necessitate the same treatment as uremia, with sodium bicarbonate for the acidosis.

5. Ascites.—Ascites is seldom benefited by the cardiants, sweats, diuretics or purges. Early repeated punctures may induce adhesions and consequent collateral circulation (see page 616).

6. Operation.—Talma and Van der Meulen (1889) recommended operative measures, which were later amplified in various ways, to induce a collateral circulation; these consist of sewing the omentum to the abdominal wall, abrading the surface of the liver to promote adhesions and Widal reports a successful Eck's fistula. Bircher collected (1908) 343 operations; the immediate mortality was 6 per cent.; of the surviving cases, 13 per cent. were improved, 33 per cent. recovered and 48 per cent. were not improved. The ascites and collateral circulation involve vital as well as mechanical considerations; perhaps lessening of venous tension in the portal circuit helps the liver function.

BILIARY CIRRHOSIS.

Definition.—A cirrhosis characterized by fibrosis of an intralobular type, enlargement of the liver without contraction, splenic enlargement, chronic icterus without gross obstruction of the bile vessels, paroxysmal fever and absence of portal stasis. It is known as *Hanot's disease* (1876). The term hypertrophic cirrhosis is unfortunately ambiguous.

Etiology.—Its etiology is obscure. Alcoholism is not a factor. Sometimes it apparently results from (*a*) *infection,* either hematogenous, or an ascending *cholangitis.* (*b*) *Age.* Most cases develop between the twentieth and thirtieth years; it is rare after forty; a considerable percentage occurs in the young, especially in India. (*c*) *Sex.* Over 80 per cent. of adult cases are *males.*

Symptoms.—The onset is characterized by early icterus, less often by asthenia, fever, dyspepsia, diarrhea, splenic or abdominal pain.

Cardinal Symptoms.—1. The *liver* is *uniformly enlarged* and may weigh 2000 to 4000 gm. Its surface is smooth and its edge even, though somewhat blunt. Its pathology is considered under diagnosis. Its dulness extends from the fourth rib in the nipple line to the umbilicus or even to the iliac crest; it visibly bows outward the right costal arch and its weight drags down the right shoulder. The gall-bladder is not enlarged. The liver progressively increases, often paroxysmally with fever, pain and increased icterus. It never shrinks, except when combined with portal cirrhosis.

2. *Icterus develops early* and is *constant, permanent and characteristic.* It is usually intense. It is due to obstruction of the smaller bile vessels; perhaps also to oversecretion of bile (polycholia) or bile pigments (polychromia). The skin may become green or brown, frequently with itching, xanthelasma and eczema.

3. The *spleen is hypertrophied,* usually relatively more than the liver, averaging 400 to 1000 gm. in weight, but sometimes actually outweighs it (2600 gm.), especially in infantile forms. It is hard, smooth and evenly increased in all its elements and dimensions. It is distinctly palpable and often clearly visible.

Other Symptoms.— *Digestive.*—Nausea, vomiting, dyspepsia and tympanites are uncommon. Diarrhea is frequent. The normal or diarrheal movements contain bile in over 90 per cent. of cases.

Nutritive.—Nutrition is maintained for years, but wasting subsequently appears. Infantilism may be as marked as in cretinism or congenital syphilis. Gilbert and Lereboullet collected 40 cases of clubbed fingers from hyperplasia of the soft tissues.

Urinary.—The urine is highly colored, bile-stained, increased in amount, and may contain urobilin, indican or albumin.

Circulatory.—The pulse is not slow; the often dilated heart may reveal a systolic murmur; dyspnea is frequent from pressure on the diaphragm by the swollen liver and spleen; the blood shows secondary anemia, decreased coagulability and moderate leukocytosis. Arthritis and neuritis are rare complications.

Diagnosis.—The following table shows the essential pathological and clinical points of contrast with the portal type:

Portal (Atrophic) Cirrhosis.	Biliary (Hypertrophic) Cirrhosis.
1. *Etiology;* alcoholism; over forty years of age.	Infection; under forty years.
2. *Liver*, large or small (possibly both in succession); often shrinks.	Always large and to greater degree. Never shrinks in pure forms.
Granulations; larger.	Small; very fine.
Yellow.	Dark, icteric, green.
3. *Connective tissue* ensnaring several lobules (multilobular or annular), and around portal radicles.	Begins in lobule (monolobular or insular) around smaller bile radicles (cholangitis), in early stages at least.
Distinction between connective tissue and lobules sharp.	Not sharp.
Connective tissue firmer, more fibrous; more elastic tissue.	More delicate, reticular, richer in cells, embryonal.
Contracts.	No contraction (elephantiasis hepatis.)
4. *Liver cells*, early fatty, degenerate, disappear.	Seldom fatty, normal for long time, regenerate, pigmented.
5. *Portal system* cannot be injected	Easily injected.
6. *Bile vessels.* (Proliferating bile vessels in biliary form are not characteristic, and are probably merely compressed rows of liver cells.)	
7. *Icterus* infrequent, a complication; when present, usually catarrhal.	Constant throughout course, though the stools contain bile.
8. *Ascites* rarely absent in advanced cases.	Absent in all (except mixed forms).
9. *Collateral* circulation—usual.	Never.
10. *Hematemesis*, frequent, early, mechanical.	Very rare, late, cachectic or toxemic.
11. Constipation usual.	Diarrhea frequent.
12. Duration of two or three years.	Five to ten years.
13. Death results from hematemesis, nephritis, tuberculosis, peritonitis.	Rare; death from hepatargia (cholemia) with fever, delirium, typhoid state.

It is a rare affection and a diagnosis of biliary cirrhosis is too often made. If the cardinal features under its definition are borne in mind it is differentiated with ease from *hepatic cancer, echinococcus* (*q. v.*), *fatty and amyloid* liver and *Banti's disease* (*see diagnosis of portal cirrhosis*). *Obstruction* of *the bile ducts* may produce an enlarged gall-bladder (*v.* Icterus); the stools are usually but not necessarily acholic (*e. g.*, in stone of the common duct). The spleen is not large. (It may be stated that some recognize a biliary cirrhosis due to chronic obstruction of the duct; simple mechanical obstruction never causes fibrosis, though it may follow ascending cholangitis. The liver is far less large in this obstructive type and is seldom hard.) In hemochromatosis and pigmentary cirrhosis icterus is absent and glycosuria is present in about 85 per cent. of cases. (See Differentiation, page 587).

Treatment.—The treatment is that of portal cirrhosis; no known means stops the formation of connective tissue. A generous diet may be given. Cold and dampness are especially to be avoided. Calomel in small doses (grain $\frac{1}{60}$—$\frac{1}{40}$ t. i. d.) may be given for weeks at a time and the salicylates and simple salines are sometimes beneficial. Operative relief by drainage of the gall-bladder is unwarranted and in the reported cases the true biliary cirrhosis is often confused with obstructive icterus.

ABSCESS OF THE LIVER.

Suppurative hepatitis occurs in 1.5 per cent. of autopsies; 97 per cent. occur in males.

Varieties.—1. In the *septic type* (55 per cent. of cases) the hepatic artery is the usual carrier of infection; the primary focus is usually in the external parts, especially the skull (Hippocrates), osteomyelitis, pulmonary abscess or gangrene, putrid bronchitis and, less often, from ulcerative endocarditis. Liver abscess occurs in 15 per cent. of surgical septicopyemias. Infected thrombi in the peripheral veins may become detached, infarct the lungs and thence reach the arterial circulation. In rare cases infective material may fall back from the cava into the hepatic vein, known as retrograde embolism. The bacteria are those enumerated under sepsis—the pyogenic organisms, pneumococcus, colon and typhoid or paratyphoid bacilli.

The *symptoms* are obscured by the general toxemia and multiple abscesses scattered through the liver usually produce few decisive local symptoms. The liver enlargement is uniform and generally indistinguishable from the cloudy swelling of sepsis; fluctuation is rare; the portal circulation is rarely obstructed; pain and tenderness are usually present; jaundice is inconstant; the spleen is swollen from sepsis; the course is rapid.

2. The *second variety*, from infection through the *portal vein* (31 per cent.), has been known since Morgagni's time and is clinically the most important type. Any ulcerative or suppurative affection in parts containing the portal branches may be causal, for instance, amebic dysentery (the most frequent cause), appendicitis (in 6 per cent. of its fatal cases), neglected hemorrhoids, gastric ulcer or cancer and disease of the bile vessels. Pyogenic organisms, the Ameba dysenteriæ, foreign bodies (as fish-bones, pins and parasites) may enter by the portal circulation. Pus organisms may infect tuberculous, echinococcic or actinomycotic foci, of which latter 40 cases are recorded. The lesion is (*a*) the *solitary* or tropical *liver abscess* (*v. i.*) or (*b*) there may be suppurative pylephlebitis, which is less a suppuration of the liver substance than an inflammation in the portal vein itself, following in a dendritic fashion through all of its intrahepatic ramifications; its branches are distended and present small accumulations of pus, which may appear as yellowish foci through the capsule, but show more clearly on section as portal vein suppuration; the whole liver may be involved or certain areas may be exempted by total thrombotic occlusion of the larger branches. In some cases the purulent thrombophlebitis may be traced back to the original lesion in

the stomach or intestine; the liver is enlarged but the diagnosis is rarely more than a probable one.

3. The *third variety* is infection by the *bile vessels* (10 per cent.), in which an ascending cholangitis (*q. v.*), often calculous in origin, produces multiple small abscesses throughout the liver substance; this is rather similar in appearance to the pylephlebitic form.

4. Infection in infants through the *navel* is infrequent.

5. Direct *involvement* of the liver *by contiguity*, may occur in suppuration of the gall-bladder or gastric ulcer.

Solitary or Tropical Abscess.—The abscess is solitary in 75 per cent. of cases; in 11 per cent. there are two and in 14 per cent. more than two foci. Twenty to 30 per cent. of cases of amebic dysentery, by far the most common cause, are complicated by liver abscess. Amebic abscess may even occur without intestinal ulceration, though amebæ are found in the stools; in 5 out of 27 of Futcher's cases there were no intestinal symptoms. Bacillary dysentery is rarely a cause, and trauma, pyemia, typhoid or hydatid cysts occasionally. Though seen in the Southern States and sometimes in the North, it occurs chiefly in the tropics, and among Europeans; alcoholism and a rich diet are predisposing factors; 97 per cent. occur in males and 50 per cent. in those between twenty and thirty years of age.

Pathology.—Amebæ reach the liver by the portal vein and cause necrosis by their toxins, followed by suppuration. The smaller foci contain glairy, gelatinous, translucent fluid; the larger foci contain white, yellow, green, reddish (like anchovy sauce) or serous, bloody or viscid, chocolate-colored fluid. Absence of leukocytes in the walls and contents is pathognomonic. Red disks and necrotic liver cells are frequent and the pus is usually sterile. The amebæ are more abundant in the abscess wall than in its contents and may be found outside of the abscess, necrosing the liver cells. In contrast to other abscesses, the walls of the amebic type are soft, ragged and show little granulation or fibrous tissue.

Symptoms.—Latency occurs in 13 per cent. of cases.

1. Septic Symptoms.—(*a*) *Fever* is remittent or intermittent, less often continuous and in chronic cases there may be a normal or subnormal register. (*b*) *Rigors* and sweats are frequent, even independent of fever (Lafleur). (*c*) *Leukocytosis* occurs in 50 per cent. only. In 43 uncomplicated dysentery cases the leukocyte count was above the average count of the abscess cases in 23.4 per cent. (Futcher). (*d*) The *pulse* is small and frequent. (*e*) Progressive *emaciation* is usual, and the facies is a combination of hectic, subicterus and cachexia. (*f*) The urine is febrile and may contain albumin or albumose.

2. Hepatic Symptoms.—(*a*) The *liver* is enlarged from the abscess and from sepsis. The *abscess* in 70 per cent. is located in the *right lobe;* venous blood from the pancreas and intestine is conveyed to the right lobe (whence its involvement in intestinal ulceration or cancer), while the blood from the stomach and spleen finds its way to the left lobe. The *enlargement* is more often *upward* than downward, wherein abscess differs from cancer and resembles echinococcus. The swelling is often localized, occasioning a *dome-like dulness on the upper border* of the liver (Frerichs),

which may be corroborated by the *x*-rays, and may also give Grocco's dulness to the left of the vertebræ; in other cases there is bulging of the lower interspaces or of the hypochondrium. The entire organ may be greatly swollen, as in one abscess which contained 2⅓ gallons of fluid. When it occurs below the ribs, fluctuation in the liver and, less often, edema of the abdominal wall may be noted. The liver consistence may be increased at first. (*b*) *Pain* is usually present and is due to increased weight or to capsular tension and perihepatitis or pleurisy, causing a friction rub, or adhesions in more chronic cases. The patient usually lies on the back or right side (to avoid pain caused by sagging of the liver in the left lateral decubitus). Pain is referred to the right shoulder in 17 per cent., being reflected from the phrenic filaments on the liver convexity along the phrenic nerve to the acromial branch of the fourth cervical nerve; in rare cases the deltoid may waste. (*c*) *Tenderness* over the liver is usually circumscribed. The right rectus is frequently tense.

3. Other Symptoms; Complications.—(*a*) *Icterus* occurs in 16 per cent. of cases; it may be slight from sepsis or compression of some bile ducts within the liver, or it may be complete from catarrh of the common duct. (*b*) The *spleen* is *seldom enlarged*, except from a causal septicopyemia. (*c*) A dry spasmodic *reflex cough* may be noted (*tussis hepatica*). (*d*) *Rupture* may be the first sign of abscess of the liver. In Waring's 300 cases, 56 per cent. remained intact, 16 per cent. were operated on and 28 per cent. ruptured. (i) It occurs *into the lungs* in 43 per cent.; lung abscess results more often from direct rupture than from indirect metastasis. The patient presents signs of lung abscess (*q. v.*) and expectorates chocolate-, anchovy- or cream-colored pus containing amebæ, often liver and lung tissue, hematoidin crystals and perhaps bile. Death may be caused by profuse hemoptysis. (ii) Rupture into the *pleura* causes empyema (15 per cent.). Pleurisy, both fibrinous and serous, may also result from infection passing through the lymphatics of the diaphragm; a right-sided friction rub, serothorax or empyema always suggests the possibility of an hepatic etiology. (iii) Rupture into the peritoneum causes localized or diffuse peritonitis (15 per cent.). A subacute serofibrinous peritonitis or an encapsulated serous effusion over the liver rarely results. (iv) Rupture into the *bowels* (colon 2 per cent. and duodenum 1 per cent.) gives pus, sometimes in large amounts in the stools. (v) Less frequent are ruptures into the stomach, pericardium, pelvis of the kidney, portal or hepatic vein, inferior cava, bile vessels or abdominal wall. (*e*) Less frequent complications are pyloric obstruction (from abscess of the left lobe), pseudorheumatism or clubbed fingers (from toxemia), cerebral abscess and intestinal obstruction.

Diagnosis.—Diagnosis is based on (*a*) an *etiological factor;* (*b*) symptoms of *sepsis;* (*c*) *hepatic signs, e. g.,* localized enlargement, pain and tenderness; the *x*-rays may be useful when the bulging of the convexity is covered by the lung; (*d*) exploration with the *aspirating needle,* which the author has seen result in two deaths from peritonitis and one from an enormous hemorrhage into the peritoneum; puncture should be

performed only when an operation can be done immediately after pus is found; pus is far less often withdrawn in multiple small abscesses than in the large solitary form.

DIFFERENTIATION.—*Five types* of liver abscess are often described: (*a*) The latent, (*b*) typhoid (page 46), (*c*) malarial, intermittent, (*d*) phthisic, and (*e*) a type declared by rupture.

Subphrenic abscess simulates pneumothorax (*q. v.*) rather than liver abscess, because of the presence of tympany; in its etiology and treatment it resembles liver abscess (see page 611). In *gall-stones*, with or without suppuration, the fever usually occurs in paroxysms separated by long intervals, the history is different and marasmus is rare. Differentiation from *pleurisy*, *echinococcus* and *cancer* is considered under these topics and on page 587.

Prognosis and Treatment.—Ninety-three per cent. of unoperated cases die from rupture (peritoneal, pleural, pulmonary), sepsis and amyloid disease. It is possible for an abscess to desiccate or to heal after breaking into the lung or bowel, but this cannot be depended upon. The treatment is solely operative; Solonoff estimates the mortality of 1094 cases at 30 per cent.

TUMORS OF THE LIVER.

Tumors of the liver are chiefly *carcinomata*. *Benign tumors* possess little clinical interest, as fibroma, lipoma, teratoma, adenoma or angioma; single adenoma may develop from the liver cells or bile ducts; multiple adenomata occur chiefly in cirrhosis. Three cases are reported of adrenal "rests," and scant 20 of *sarcoma;* it may develop as a single primary tumor, multiple primary tumors or diffuse infiltration; a melanotic growth is almost always secondary to melanotic sarcoma of the eye or skin.

Cancer of the liver occurred in 2.7 per cent. of Virchow's autopsies, and ranks fourth among internal cancers (in order of frequency, uterus, stomach, mamma and liver). (*a*) *Primary cancer* is 20 to 48 times as infrequent as secondary cancer; 66 per cent. occur in males. It originates from the liver cells (88 per cent.) or bile vessels (12 per cent.). The main forms are observed (i) as a sharply outlined massive local swelling (in 23 per cent. of Eggel's 163 cases); (ii) as sharply demarked multiple nodes of moderate size (in 65 per cent.) of which one of the tumors is primary and the rest secondary; (iii) as diffuse infiltration, fusing with the parenchyma (in 12 per cent.); (iv) the last form develops in hepatic cirrhosis (or hepatic cyst). Metastases occur in 66 per cent. (*b*) *Secondary cancer* results from primary cancer in the stomach (33 per cent.), colon (12 per cent.), esophagus, pancreas, gall-bladder, uterus, ovaries, mamma, etc., by metastasis or contiguity; cancer cells may enter by the portal vein and hepatic artery, much less frequently by retrograde metastasis through the hepatic vein and never by the lymph vessels, as their current flows from within to without the liver.

Symptoms.—1. HEPATIC SYMPTOMS.—(*a*) The *liver enlarges rapidly*, even to eight times its normal size and may weigh 30 to 40 pounds. Its *edge is uneven;* on its *surface are lumps* which may present a central

umbilication. *Great unevenness* is more characteristic of secondary than of primary tumor, in which the liver is more uniformly swollen. The *enlargement* is chiefly *downward,* though sometimes upward from ascites, tympanites or adhesions. Nodules in the falciform ligament may be felt near the navel or linea alba. Sudden increase in size occasionally results from hemorrhage due to icterus. There is no enlargement when the nodules are few and small, and the liver is smaller than normal in 10 per cent. of cases (liver cirrhosis plus malignant adenomata). (*b*) *Hepatic pain* is *more conspicuous than in any other liver lesion,* from capsular tension, perihepatitis, pleuritis or, less frequently, from rupture of tumor particles into the bile passages, simulating gall-stones. (*c*) The *liver is* often *tender.* (*d*) Exceptionally there may be pulsation transmitted from the aorta or a venous hum over the liver.

2. Cachectic Symptoms.—These are adynamia, marasmus, anorexia, secondary anemia, leukocytosis (in any carcinoma, but especially in hepatic), stupor and illusions, probably from the katabolic toxins elaborated by the tumor, and hepatargia. Fever may result from the tumor alone, or terminal infection.

3. Compression Symptoms.—(*a*) *Jaundice* occurs in over 50 per cent., more frequently in secondary than in primary cancer and usually from mechanical pressure on the larger bile ducts by the tumor or carcinomatous glands. Pressure on the ducts within the liver often produces moderate jaundice and some bile then appears in the stools; complete icterus may result from concomitant catarrhal or calculous obstruction, especially significant when there is no ascites. Exceptionally the tumor grows into and along the bile ducts, thereby totally obstructing them. (*b*) *Ascites* is present in 50 per cent. of cases. It is usually moderate in degree and results from pressure on the portal vein, peritonitis or pylethrombosis; occasionally it is due to the cirrhotic form of cancer, growing of the tumor into the hepatic or portal vein, rupture—perhaps with syncope and great hemorrhage—of a cancer nodule into the peritoneum, or rarely to plugging of the liver capillaries by the pigment of a melanosarcoma. A collateral circulation or perihepatic friction may be noted. The fluid is usually serous or hemorrhagic, rarely adipose, chylous, opaque or melanotic. (*c*) *Other compression symptoms* include splenic tumor from marantic pylephlebitis (14 per cent.), hydronephrosis, vomiting, dyspnea and edema.

4. Other Symptoms.—The cervical glands are seldom swollen unless there is coincident gastric cancer. Carcinomatous nodes in the navel or skin over the liver are due to regional extension. In liver cancer combined with cirrhosis any cirrhotic symptom may occur. The urine shows increased destruction of the tissue nitrogen; acetone and diacetic and beta-oxybutyric acids, indican, leucin and tyrosin are not infrequent. The urine is decreased, high-colored and often icteric. Albuminuria is due to stasis and toxemia. Melanuria (sarcoma) is sometimes detected by adding to the urine a solution of ferric chloride; the pigment exists in the blood as melanin or melanogen; melanuria nearly always indicates a melanotic tumor of the liver and is often confounded with indicanuria and alkaptonuria.

Diagnosis.—The chief features are rapid enlargement of the liver, irregular tumors, pain, icterus, ascites, cachexia and in 50 per cent. of cases the recognition of the primary tumor. (See page 587.)

Differentiation.—*Cirrhosis* (see both forms); *syphilis* (*q. v.*); the *amyloid liver* is equally large and causes confusion only when complicated by gummata; amyloidosis of the spleen, kidneys and intestines usually coexists with amyloid liver. *Hydatid cysts* develop more slowly and *earlier in life* and cachexia appears late, if at all. The rare multilocular echinococcus produces enlarged liver, ascites and icterus, but the spleen is also enlarged; puncture may give characteristic findings. Puncture often excludes those doubtful cases of cancer in which fever, leukocytosis and semifluctuation of the necrotic neoplastic nodes simulate *liver abscess*. *Congested liver;* the writer saw 4 cases of nutmeg liver in which stasis caused more local than general hepatic enlargement. The *corset liver* can be easily differentiated if the general symptoms, such as cachexia and the compression symptoms of cancer, are duly considered. *Cancer of the stomach* (*q. v.*) is readily differentiated by its chemism and stagnation; gastric cancer may cause hepatic involvement. *Renal or adrenal tumors* are covered by the inflated colon, encroach less upon the thorax, evert the ribs less, and often are separable from the liver by an intervening strip of tympany, which occurs in but 13 per cent. of hepatic tumors.

Differentiation between primary and secondary tumors is somewhat irrelevant, as the causal growth leading to hepatic metastasis is latent in 50 per cent.; the following characteristics are subject to many exceptions: the solitary tumor, its rapid growth, less emaciation, more rapid course and death in less than four months. Secondary tumors cause death within seven months from cachexia, ascites and intercurrent disease, as pneumonia—or in a shorter period, from hemorrhage, peritonitis, icterus, galloping carcinosis or acidosis.

Treatment.—Treatment is palliative for the pain and ascites except when, in very exceptional cases, early operation (for primary growths) can be performed. Lücke reported the first recovery, which afterward proved to be a gumma; Keen collected 76 operated neoplasms with 63 recoveries.

ECHINOCOCCUS CYSTS OF THE LIVER.

The parasite is considered on page 305. Its hepatic localization constitutes 60 per cent. of echinococcus disease.

Symptoms.—*Fully half the cases* of liver echinococcus are clinically *latent*. Cases with symptoms show the following *liver findings:* (i) A *cyst on the lower border* or anterior surface may produce a visible, palpable, round and *smooth bulge* which everts the right costal arch or much less often distends the left lobe; cysts in the right lobe may lead to compensatory hypertrophy of the left lobe. The tumor is as large or larger than an orange. It is tense, elastic and dull; on percussion it may fluctuate and give the "hydatid thrill," a vibration elicited by laying three fingers over the cyst and tapping with the middle one; the thrill may also occur in ascites and sarcoma. The cysts rarely contain gas, due to

the Bacillus coli. In large cysts (36 to 70 pints are reported), the liver may tip so that the right lobe is lower and the left higher than normal. (ii) *Cysts on the convexity* crowd the diaphragm upward, cause symptoms resembling pleurisy (*q. v.*), produce a dome-like dulness, as in cancer and may compress the lung or heart. (iii) *Liver pain* and *tenderness are rare* without suppuration, which may induce perihepatitis; pain may be referred to the right shoulder. (iv) *Exploratory puncture* may precipitate severe nervous toxemia and cardiac collapse, as described on page 306, where also the finding of pus, membrane, chemical contents and hooklets are enumerated.

Rupture may occur into the lungs (in 11 per cent. of liver echinococcus); it may simulate phthisis or gangrene of the lungs; cysts may rupture into the pleura, peritoneum (with eosinophilia), kidney, stomach, bowel, bile passages, pericardium, portal vein and cava; heart embolism or external rupture may occur.

Pressure symptoms are rare, as icterus (5 per cent.), ascites, albuminuria, *caput Medusæ*, edema of the legs, dyspnea or vomiting. In some cases there is a distaste for fat or meat. The eosinophiles are usually increased in non-suppurating cysts. Urticaria may occur after puncture or rupture or without either incident.

Diagnosis.—Diagnosis depends on the history of the case, including contact with dogs; the smooth, round tumor; the thrill; slow development; the infrequency of constitutional disturbance, pain and fever, the signs predominating over the symptoms.

Differentiation.—(*a*) From *other liver cysts;* the small cysts in cirrhosis and obstructive icterus are of no clinical importance. Simple serous cysts may, in exceptional cases, hold several (even 13) pints; they rarely cause symptoms, rupture or hemorrhage. Diffuse, congenital, cystic disease may cause dystocia; in adults also the cysts are probably developmental in origin and are associated with cystic kidneys, in 15 per cent. of cases; Moschcowitz collected 85 cases; they are usually multiple and subcapsular and are often associated with other congenital anomalies, such as hare-lip, and are due to inflammatory hyperplasia of aberrant bile ducts or to congenital obstruction in them. Hoffman reported 18 operated cases. (*b*) *Malignant disease* causes cachexia, more pain than echinococcus, multiple tumors, ascites and icterus. (*c*) *Syphilis of the liver* (*q. v.*). (*d*) From *pleurisy;* the *x*-rays may show the localized tumor on the liver convexity; the upper line of pleuritic dulness is much less often convex upward; there may be thoracic symptoms, as hemoptysis, when there is rupture into the lung; the sputum or aspirated fluid shows the diagnostic hooklets. (*e*) In rare cases a pendulous, pedunculated, movable cyst may resemble a pancreatic cyst, enlarged kidney, gall-bladder or ovarian cyst. Rupture into the bile passages may simulate the pain of gall-stones. (*f*) Liver abscess; there may be doubt in suppurating cysts; the eosinophiles are not increased in liver abscess (page 587).

Prognosis.—Cysts may calcify or indurate. The mortality of rupture into the peritoneum is 90 per cent., into the pleura 80, bile ducts 70, bronchi 57, stomach 40, intestines 15 and externally 10 per cent.

Treatment.—When drainage by the trochar is performed (Hippocrates), 60 per cent. of cases recover. A few die suddenly from a toxalbumin, formed when the parasite dies; it causes urticaria, convulsions, cardiac failure and other symptoms similar to mussel-poisoning. *Direct surgical intervention* is attended with the least risk.

Multilocular or Alveolar Echinococcus.—This form is due to another parasite (see page 307); 100 cases are recorded.

Symptoms.—The right lobe of the liver alone is involved in 65 per cent. of cases and the left alone in 10 per cent. The liver is fibrosed, nodular and seldom fluctuates. This rare affection is attended by splenic tumor (90 per cent.), icterus (80 per cent.), dyspepsia, ascites and, less commonly, by fever, polyuria, collateral circulation, emaciation and metastases in the heart and lungs; death may occur after years from cholemia or gastro-intestinal hemorrhages. The diagnosis is very difficult, especially from cancer. The only hope of recovery is in extirpation.

FATTY LIVER.

"Fatty liver" (*hepar adiposum*) includes infiltration and degeneration; they may be considered together.

Etiology.—(*a*) *Obesity* and (*b*) *decreased oxygenation* from carcinosis, rickets, profound anemia, stasis and kindred causes may produce fatty liver. (*c*) *Toxemias*, as tuberculosis, the acute infections, infantile diarrhea, acute yellow atrophy or suppuration, (*d*) *poisons*, as phosphorus, arsenic or chloroform, and (*e*) *local diseases* of the liver, as cirrhosis or nutmeg liver, are etiological factors.

Pathology.—The fat may amount to over 40 per cent. (normally 2 to 5 per cent. of its weight). The *liver* is *enlarged* and may weigh 4500 gm. Its *surface is smooth*, its edges rounded, *consistence is decreased* and its color yellow. On section it is anemic, smooth and yellow when oleic acid predominates or pale when there is more palmitin and stearin. Slight quantities are detected only on microscopic examination. When cut with a warm knife the fat is seen on the blade. Microscopically, fat is seen which, *in fatty infiltration*, is located principally in the periphery of the lobules and obscures the liver cells; when the fat is dissolved by ether, the normal liver cells are clearly seen. *In fatty degeneration*, fat forms at the expense of the liver cells, *i. e.*, the percentage of albumin is decreased. The statement that fatty infiltration occurs in large and fatty degeneration in smaller globules is not wholly correct. Pathologists consider infiltration more important.

Symptoms.—(*a*) *The positive findings* are as follows: the liver is *symmetrically enlarged* and *smooth* and rarely extends below the navel. Its *edge is rounded*, soft and usually palpable, even in obese persons. (*b*) The *negative findings* are absence of pain, tenderness, icterus, splenic tumor, ascites or gastro-intestinal hemorrhage.

The *diagnosis* is considered on page 587.

AMYLOID LIVER.

Amyloid degeneration was first clearly described by Rokitansky.

Etiology.—(*a*) *Ulcerative tuberculosis* of the lungs or bones causes over 50 per cent. of amyloidosis, and (*b*) *inveterate syphilis,* particularly ulcerative and osseous types, 25 per cent. (*c*) *Chronic suppuration* has become uncommon since the introduction of antisepsis. Krawkow produced amyloid degeneration experimentally by inoculations of the staphylococcus. (*d*) As exceptional causes, intestinal ulcerations, exhausting diarrheas, rickets, leukemia, pseudoleukemia and ulcerating neoplasms may be mentioned; 78 per cent. of cases occur in males.

Pathology.—Amyloid degeneration is much less frequent in this country than in Europe. Its *localization,* in order of frequency, is in the spleen, kidneys, liver and intestines. The liver is uniformly enlarged and may weigh 5000 to 6000 gm., even in a child. It is hard, smooth and very pale. Its edges are blunt. On section the appearance is waxy, speckled, resembling raw ham, the edges slightly translucent and the lobules indistinct. If a thin slice is placed in diluted Lugol's solution, the amyloid areas assume a brown color (Virchow), the name (amyloid) referring to this starch-like reaction; the waxy substance, however, is a proteid and reaches the liver by the hepatic capillaries, whence it is first deposited in the middle zone of the lobule, and later infiltrates the central and peripheral zones. It is essentially an infiltration.

Symptoms.—The large, tense, symmetrical, painless, smooth liver, with rounded edges, is characteristic. Its dimensions may attain those of cancer. The onset and development are usually slow, but acute amyloid may develop in a month. *Splenic* amyloid *enlargement* is usually also noted, as well as *albuminuria* from renal amyloidosis (*q. v.*). In only 0.8 per cent. of amyloid degeneration is the liver alone involved. *Gastro-intestinal symptoms,* as nausea, vomiting and diarrhea, may result from amyloidosis of the alimentary capillaries. In advanced cases *cachexia, edema* and ascites may develop. Hepatargia is rare.

Diagnosis.—Diagnosis depends on (*a*) the recognition of a *cause;* (*b*) the characteristic, smooth, *symmetrical enlargement,* which is less hard than that of cirrhosis; (*c*) the associated *amyloid disease* of the *spleen, kidney* and alimentary tract; (*d*) the *absence of icterus* (very rarely from amyloid portal glands) and of *portal stasis* (ascites is an infrequent, late and renal symptom); and (*e*) the small amount of urobilin in the stools. *Differentiation* from cirrhosis (*q. v.*) and cancer (*q. v.*) presents no difficulties. Gummata in the liver plus amyloid disease may cause confusion. No diagnosis is possible in the exceptional cases in which the liver is slightly enlarged (because slightly diseased) or in which splenic tumor and albuminuria are absent (*v.* page 587).

Prognosis.—The prognosis of pronounced cases is unfavorable though light grades may exceptionally regress.

Treatment.—Treatment is prophylactic and symptomatic.

ANOMALIES OF FORM AND LOCATION OF THE LIVER.

In *situs viscerum inversus* the liver lies on the left side. Fenger encountered a case of icterus and gall-stones in situs inversus which was diagnosticated as acute yellow atrophy!

Tilting of the liver, *dislocation downward* by pleuritis, pneumothorax or tumors, or *upward luxation* require but passing mention.

"Wandering Liver."—Hepar migrans s. mobile occurs in women (88 per cent.), usually in multiparæ. Its *causes* are those of enteroptosis, with which it usually occurs, viz., flaccid abdominal walls, congenitally lax suspensory ligaments, lacing or very rarely from traction by a tumor. Clark and Dolley collected 118 cases; 1 had a mesohepar attached to the diaphragm and absence of the coronary and right lateral ligaments.

Symptoms.—The onset is usually gradual; in 5 per cent. it is acute. *Subjectively,* oppression over the liver, gastro-intestinal or nervous symptoms may be present. *Objectively,* there is sometimes cardioptosis, usually gastroptosis and in 20 per cent. nephroptosis. The hepatic notch can be distinctly felt, often also its convex surface and sometimes the tense, stretched ligaments. It can be replaced. In a recent case there was tympany between the left costal arch and the prolapsed spleen and tympany between the right arch and the liver, even though the patient was five months pregnant. In very rare instances there may be pain similar to gall-stones and ascites or splenic tumor from torsion of the portal vein.

Treatment.—The *treatment* is (*a*) *mechanical,* replacing and supporting the liver with binders; (*b*) *symptomatic,* for the neurasthenia; (*c*) *dietetic,* to increase the body weight and (*d*) *operative;* hepatopexy cures about 75 per cent.

Corset Liver.—The corset liver is found in 25 per cent. of female cadavers, but much less frequently here than abroad. Corsets cause pressure atrophy of the liver substance, followed by induration, deformity and peritoneal thickening, most conspicuously and usually in the right lobe. The liver may be merely constricted, dislocated upward or the part below the constricting zone may hang loosely as upon a hinge, causing confusion with floating kidney or other tumors. The gall-bladder is dilated in 60 per cent., but icterus is infrequent. Corset liver is regarded as a factor in gall-stone formation. Subjective manifestations are often absent. Sometimes paroxysmal syncope and vomiting may occur, due to congestion of the snared-off portion. Respiratory excursion prevails in most cases. Confusion arises when the intervening gut causes a narrow zone of tympany between the hard, hinged segment and the liver proper, but even then careful palpation frequently demonstrates its continuity with the liver. *Congenital tongue-like lobes* may be confused clinically with corset liver; they are often the seat of cirrhosis, malignancy, suppuration or syphilis. Treatment is seldom important.

AFFECTIONS OF THE BLOODVESSELS OF THE LIVER.

I. Active Hyperemia of the Liver.—A degree of arterial hyperemia is physiological during digestion. An accentuation of this condition is common in heavy eaters and drinkers and acute infections, in which it may be combined with venous hyperemia. Heat is probably not the cause of "tropical hyperemia." The alleged vicarious hyperemia from suppressed menstruation, the menopause or diseases of the internal female genitalia is based on theory. The symptoms resemble those of passive hyperemia (*v. i.*). A simple or milk diet, calomel and saline purgation are indicated.

II. Passive Hyperemia of the Liver.—Etiology.—Passive congestion of the liver results from stasis, the stress of which is felt in the efferent intrahepatic (sublobular) veins. It results (*a*) from *cardiac insufficiency*, valvular, particularly mitral diseases, myocardial or pericardial lesions; (*b*) from *pulmonary* stasis, emphysema, chronic bronchitis, asthma, induration or obliterative pleurisy; (*c*) from stasis caused by *spinal deformity*, aneurysm, mediastinal tumor, *perihepatitis* or pressure on the cava inferior.

Pathology.—The essence of passive congestion is overfilling of the sublobular or the *hepatic venous circulation*, malnutrition of the liver cells (*fatty degeneration*) and in some advanced cases, *increased connective tissue.* The liver is usually *symmetrically enlarged, firmer* and darker red than normal, though hepatic hyperemia lessens after death. The capsule is often thickened or edematous. On section the so-called *nutmeg appearance* is noted, but seldom over the entire liver. The *central veins* with their radiating offsets are *dark* and cyanotic, while the periphery of the lobule is yellowish-white from cloudy or fatty change (malnutrition or impaired oxygenation). Long-standing congestion results in atrophy and pigmentation of the cells in the periphery of the lobule (Virchow's *red atrophy*); there is usually increased connective tissue with fine granulations, the *atrophic cyanotic liver* or cardiac cirrhosis of the French; fibrosis is for the most part relative or apparent, for the liver parenchyma atrophies; true cirrhosis may coexist with cardiac disease.

Symptoms and Diagnosis.—(*a*) The *causal heart* (or lung) lesion has its appropriate symptoms. (*b*) The *liver* is *evenly enlarged,* on palpation and sometimes on inspection; if ascites is present thrusting palpation with the finger-tips usually outlines the organ; the liver may reach below the navel or evert the ribs. Its *edges are firmer,* somewhat rounded, *sensitive* (from capsular tension) and *descend with inspiration;* the incisure is clearly felt. If there is relative tricuspid insufficiency, a systolic expansile pulsation is detected by palpating with both hands, which are separated from each other with each systole, and is differentiated from the non-expansile pulsation transmitted from the aorta. (*c*) The *hepatic intumescence varies* greatly, as the heart responds to rest and digitalis, or compensation fails. (*d*) The *skin* shows *cyanosis* and *slight icterus;* to the experienced eye it has a characteristic appearance, probably due to a static catarrh of the finer intrahepatic bile radicles. (*e*) For other symp-

toms and treatment see Valvular Disease; the spleen is seldom enlarged save from cardiac infarcts or complicating liver cirrhosis. *Ascites* is usually *late* in onset and moderate in degree; a cardiac ascites sometimes occurs without general edema. Before death there may be signs of renal and hepatic insufficiency. In some cases with somnolence, delirium and a terminal toxemia, Oertel describes a cytolytic necrosis of the liver with stasis, fatty infiltration of the middle zone and hemorrhagic extravasations.

Differentiation is seldom complicated, for the fundamental heart lesion, the evenly enlarged, tender liver, which varies in size from time to time, and the slight icterus with cyanosis are usually unmistakable. Certain features may sometimes mislead, as the occasional unequal distribution of the liver changes, which may suggest tumor, or the tenderness which may resemble suppuration. (See page 587.)

III. Pylethrombosis—Pylephlebitis.—Thrombosis or phlebitis of the portal vein is practically always *secondary* to *compression or obstruction,* as liver cirrhosis, with which 33 per cent. of pylephlebitis cases occur; to cancer, ulceration, abscess or *inflammation* of contiguous structures, gall-stones, cholangitis and proliferative peritonitis; and to *diseases* of *the intima*, as phlebosclerosis and syphilitic endophlebitis. Pylephlebitis occurs chiefly in males over forty years of age.

Symptoms.—These may be entirely absent because of collateral compensation through the hepatic artery or Sappey's accessory portal vein. The *onset* is usually sudden, with symptoms of *acute portal stasis.* The *spleen is enlarged* in 75 per cent., unless fibrous changes in its pulp or capsule prevent its swelling. *Ascites* develops in 66 per cent. and rapidly recurs after tapping. *Gastro-intestinal* symptoms are usual, as *hemorrhage* (44 per cent.), intense, *sudden epigastric pain,* vomiting, diarrhea or sometimes acute intestinal obstruction. The *portal vein* is sometimes *varicose* below and is generally *obliterated* at and above the point of obstruction (pylephlebitis adhesiva); this leads to *shrinking of the liver,* particularly if the hepatic artery is sclerotic, in which rare event the entire liver may be hemorrhagically infarcted. Liver lobulation results from antecedent syphilis. Hepatargia and alimentary glycosuria may develop. Edema of the abdominal wall, development of a collateral circulation and icterus are uncommon.

Diagnosis.—Diagnosis is difficult, unless the onset is typically sudden; otherwise the findings are those of atrophic cirrhosis; a caput Medusæ indicates occlusion of the portal radicles. The ascites may suggest carcinoma or peritonitis or the hemorrhage may simulate gastric ulcer or hepatic cirrhosis.

Course and Treatment.—Death may occur in a few days or six to twenty years. Treatment is ineffectual, save in early syphilis. Citric acid (℥j) may be given to lessen thrombosis.

Pylephlebitis suppurativa is a pyemia of the portal vein; metastasis or extension occurs in the main portal trunk and its branches from infection of its radicles of origin; inflammations in the gastro-intestinal tract are its cause in 75 and appendicitis in 40 per cent. ("appendicular liver") and gall-stones, infected piles and pancreatic or umbilical suppuration cause most of the other cases (*v.* page 575).

DIFFERENTIAL TABLE OF LIVER DISEASES.

	Size.	Edge.	Surface.	Consistence.	Icterus.	Ascites.	Spleen.	Pain.
Acute Yellow Atrophy	Very small.	Even.	Smooth.	Soft.	Intense and invariable.	None.	Enlarged in 66 per cent.	Usual.
Portal Cirrhosis	Somewhat decreased. (Sometimes normal or enlarged.)	Sharp (possibly finely nodular).	Smooth (possibly finely granular).	Hard.	Seldom (15 per cent.).	Usual in 50 to 85 per cent.	Usual (75 per cent.).	Inconstant.
Biliary Cirrhosis	Very large.	Sharp.	Smooth.	Hard.	Constant.	None.	Constantly: very large.	None.
Abscess	Enlarged (mostly right lobe or on convexity).	Uneven, if abscess is on lower part.	Nodular.	Soft, even fluctuating.	Seldom (16 per cent.).	None.	None (save from sepsis).	Common.
Tumors	Greatly enlarged.	Nodular.	Nodular.	Hard.	Frequent (50 per cent.).	Frequent (50 per cent.).	Rare (in 14 per cent.).	More than in any other liver lesion.
Hydatid	Enlarged (right lobe).	Usually even.	Nodular.	Semifluctuating.	Seldom except in multilocular (80 per cent.).	None (save in multilocular).	None (save in multilocular 90 per cent.).	Seldom (except in multilocular).
Fatty Liver	Moderately enlarged	Even.	Smooth.	Soft.	None.	None.	None.	None.
Amyloid	Greatly enlarged.	Sharp yet more rounded.	Smooth.	Hard.	None.	None (except late from cachexia or nephritis).	Usual.	None.
Passive Congestion	Moderately enlarged; may shrink late.	Rounded.	Smooth.	Firmer than normal.	Subicterus plus cyanosis.	Some from cardiac stasis.	None.	Tenderness usual.
Syphilis	Enlarged (save in rarer atrophic type).	Nodular or lobulated.	Nodular (Wassermann).	Hard. (See p. 205).	In 33 per cent.	Seldom (save from atrophic type).	Frequent. Answers to therapy.	Frequent.

The *symptoms* are those of the *original disease, sepsis* and *portal stasis.* In very rare cases the thickened vein may be palpated. The spleen is enlarged, chiefly from sepsis, the liver is tender and somewhat enlarged and icterus occurs in over 50 per cent. The diagnosis is always difficult, but is based on *symptoms* suggesting liver abscess (though *without the findings*) and sepsis. Treatment is futile.

IV. Affections of the Hepatic Artery and Vein.—*Infarction* of the hepatic artery with complete necrosis of the liver is very rare. *Aneurysm* (*v.* page 409).

Thrombosis of the hepatic veins, of which only 12 cases are reported, may follow perihepatitis or, less often, compression by a neoplasm or fibrous obliteration of the inferior cava. Ascites and splenic tumor sometimes result. Suppurative thrombosis is always secondary to liver suppuration. Retrograde embolism (Heller) is mentioned under Liver Abscess.

DISEASES OF THE GALL-BLADDER AND BILE VESSELS.

ICTERUS.

JAUNDICE is a symptom of various affections of the liver and bile vessels; it is characterized by staining of the skin, mucosæ and urine by retained bile pigments.

Etiology.—Most cases of icterus are due to *obstruction* within or about the liver and biliary system; little obstruction is necessary because the bile is secreted under low pressure. (*a*) *Hepatic diseases.* It is (i) frequent in acute yellow atrophy, biliary cirrhosis, multilocular echinococcus, carcinoma, passive congestion and syphilis; (ii) infrequent in atrophic cirrhosis, unilocular echinococcus and abscess; (iii) absent in fatty and amyloid liver and in simple pylephlebitis. (See page 587.) Possibly functional liver disturbance may produce icterus (parapedesis of bile or "diffusion-icterus"), analogous to transient albuminuria (in its relation to Bright's disease). (*b*) Diseases of the *biliary tract,* as (i) catarrhal, croupous or purulent inflammation; (ii) calculous obstruction; (iii) congenital and (iv) cicatricial occlusion; (v) malignant growths; (vi) parasitic obstruction by ascaris, distoma or hydatid vesicles. (*c*) Disease in *contiguous structures,* as (i) duodenal ulcer, cicatrix, catarrh or cancer; (ii) tumors of the colon or lymph glands; (iii) tumors or inflammation of the pancreas, stomach, kidney, omentum, mesentery and peritoneum, and (iv) rarely from aneurysm. (*d*) *Hematogenous* or *urobilin icterus.* It was formerly maintained that toxic and toxemic conditions, breaking down the blood cells (hemolysis) cause hemolytic icterus. It is now thought that most of these cases are due to blocking of the capillaries by fibrin, thickening of the bile or cholangitis of the smallest bile radicles, though the obstruction is not complete; Eppinger maintains that the smaller bile radicles dilate and sometimes rupture, letting the bile into

the lymph vessels which absorb it. The jaundice in these cases is slight, bile pigment (bilirubin) is not found in the urine and the stools are bile-stained. This type occurs in various infections, pernicious anemia, and poisoning by phosphorus, chloroform, etc.

Icterus neonatorum. (*a*) The *physiological* type occurs in 66 per cent. of children; it appears on the second day, lasts two weeks and is never fatal; hemolysis, increased blood-pressure within the liver, etc., are suggested as causes. Perhaps every jaundice falls under (*b*) the *pathological* type, caused by sepsis, syphilis, Winckel's disease, or more exceptionally by congenital obstruction, gall-stones, acute yellow atrophy or catarrhal icterus. Glaister reported a family in which 6 cases of jaundice developed after birth and Rolleston an instance in which the mother, and also the babes, had icterus in four pregnancies (*v.* page 590).

Symptoms.—Bilirubin, found normally in bile, enters the blood in biliary obstruction, by the bloodvessels and produces the following symptoms:

1. Skin and Mucosa.—In three days the scleræ, skin and mucosæ become yellow and in chronic cases dark brown. The skin itches because of its dryness or the action of the bile on the nerves.

2. Urine.—The urine contains bilirubin before icterus is seen in the skin. Adding nitroso-nitric acid to the urine in a porcelain dish or on filter paper gives the Gmelin play of colors, of green, yellow, violet and red. If the urine is agitated the yellow color appears in its foam. Confusion is possible with melanuria, hematuria, urobilinuria or staining of the urine with rhubarb, picric acid, santonin, senna, etc. *Urobilinuria* is thought to indicate disease of the hepatic cells; urobilin is formed in the intestine by the reduction of bilirubin, whence it is absorbed and excreted by the urine; it never stains the skin and icterus results from bilirubin alone. There are three classes of cases; (*a*) in complete biliary obstruction with icterus only bilirubin is found in the urine, for none of it reaches the intestine to be converted into urobilin; (*b*) in partial obstruction bilirubin is found in the blood and urine and also some urobilin; (*c*) in slight obstruction there is no bilirubin but much urobilin in the urine. *Albuminuria* may occur or *cylindruria* alone and the hyaline casts and desquamated epithelium are then bile-stained. Increase in the *sulphates* and *indicanuria* are due to greater intestinal putrefaction.

3. The Digestive Tract.—The coated tongue, disagreeable breath, bitter taste in the mouth and nausea are toxemic. There may be an especial aversion to fats and sometimes to proteids. The *abdomen is distended,* possibly from absence of the antiseptic action of the bile. Starch and albumin digestion are impaired and fat in the stools (*steatorrhea*) reaches 50 or 80 per cent. (normally 7 to 10). The function of the pancreas in this respect is far more important than that of the bile. The *stools* are *clay-colored, constipated* and *stinking;* they may shimmer when the fatty soap crystals are abundant; they are also *dry* (the bile normally amounts to nearly a quart a day). If the obstruction is incomplete—as in many intrahepatic diseases—the stools are merely somewhat lighter than normal. The feces may appear brown from a nitrogenous diet or dark if voided with the urine which lends a yellow color to their surface.

4. Cholemia.—Cholemia (hepatargia or hepatic insufficiency) may

finally develop, probably from injury to the liver cells which fail to destroy the intestinal toxins. Its symptoms resemble acute yellow atrophy, *i. e.*, nervous excitation followed by stupor, irregular breathing or cutaneous and other hemorrhages. The hemorrhages are thought to result from pancreatic involvement (diminished lime salts or fat necrosis). Most subjects of icterus are depressed, irritable, sleepless and somewhat emaciated.

5. Other Symptoms.—(*a*) The *heart rate* is *slow*, particularly in catarrhal forms or recent cases and in the absence of pain, fever and intoxication; it may fall to 21; it is probably caused by vagus stimulation, for atropine raises the rate. The arterial pressure is low and the capillaries wide. A functional heart murmur is usually caused by myocardial weakness; Potain holds that the lung capillaries contract reflexly, thereby causing dilatation of the right heart. The *respiration* rate is *decreased*, even to seven a minute; the temperature is usually normal. (*b*) The *blood* shows the bile stain and in severe cases delayed coagulation, anemia, leukocytosis and sometimes a Widal reaction (typhoid cholecystitis?). (*c*) The *liver cells* may suffer mechanically and chemically from bile stasis and may be smaller, altered in form, pigmented, necrotic and bile-stained. The gross and minute anatomy varies with the cause. The bile vessels are often dilated, sometimes thickened and occasionally ruptured. The condition of the gall-bladder will be discussed under Gall-stones and Pancreatic Cancer. Simple biliary stasis never causes induration of the liver. (*d*) Splenic tumor is no part of bile stasis except from infection or portal stasis. (*e*) The *tissues, secretions and excretions are bile-stained;* the muscles and nervous tissues, except in the severe type of icterus neonatorum, are seldom icteric. (*f*) Xanthopsia, yellow coloring of objects seen, is due to the action of the bile pigment on the nervous tissues. The explanation of nyctalopia and hemeralopia is uncertain. (*g*) Of xanthelasma multiplex 23 cases were collected, and Futcher in 1905 reported 3 cases; 80 per cent. occur in chronic icterus, due to gall-stones, stricture of the bile ducts, cirrhosis, hydatids and cancer. They are often symmetrical, often begin on the eyelids or the folds and creases of the skin, in 1 case were found in the bile ducts, rarely disappear and are probably toxemic.

Diagnosis.—Icterus is not noticed in artificial light and daylight is absolutely essential for its certain detection. It is seldom confused with anemia, cachexia, Addison's disease or vagabondism. Pingueculæ are only mistaken on superficial examination.

In *ictero-anemia* (Widal's syndrome) there are icterus, splenic enlargement, urobilinuria and blood changes. The icterus is non-obstructive; the splenic tumor results from hemolysis and urobilinuria is marked. The anemia, fatigue, low blood-pressure and hemorrhages are explained by the fragility of the red globules (not observed in ordinary icterus); the red cells number 1,000,000 to 3,000,000, the index is over one and the leukocytes are increased (to over 40,000). The course is chronic and attacks like gall-stones are common.

In *chronic family jaundice* (Minkowsky) also, hemolysis and globular fragility are causal. There is *anemia* (1,000,000 to 4,000,000 reds), with free hemoglobin in the plasma; in the bone-marrow there is intense

reaction, with normoblasts and myelocytes. *Icterus* is congenital, perhaps through four generations, or appears early in life; it is neither intense nor obstructive and, lasting through life, the patients are icteric rather than ill (Chauffard). The spleen is enlarged, the urine contains no bile but urobilin, the stools are dark and attacks of gall-stone colic are frequent. Over 100 cases are reported. Iron and *x*-rays are helpful and in some instances splenectomy appeared justifiable.

Prognosis and Treatment.—The prognosis and treatment cannot be considered, as icterus is but a symptom (*v. i.* CATARRHAL JAUNDICE). Icterus may subside after six or even fifty-two years.

CHOLELITHIASIS.

Etiology.—1. IMMEDIATE CAUSE.—The causes are (i) catarrhal inflammation of the gall-bladder or bile vessels and (ii) stagnation of bile. Bacteria play an important role, especially the typhoid (found in 33 per cent.) and colon bacilli; they have been found in calculi, and cholelithiasis has been experimentally produced by their inoculation; the Bacillus typhosus may live seven years in the gall-bladder. (iii) Hypercholesteremia, in typhoid, pregnancy, nephritis, diabetes and obesity, is considered a frequent factor by Aschoff.

2. PREDISPOSING FACTORS.—(*a*) *Age;* 75 per cent. of cases occur in persons *over forty years* of age and only 1 per cent. under twenty years. (*b*) *Sex;* 75 per cent. occur *in women,* in whom pregnancy, menopause, lax abdominal walls and lacing are promoting factors; it is said that 40 per cent. of women who have gall-stones also have the corset liver. Gall-stones are found in 25 per cent. of autopsies on women over sixty years; 90 per cent. of women affected have borne children (Naunyn). They are found in 8 per cent. of autopsies.

Properties of Gall-stones.—(*a*) *Site;* calculi are formed in the gall-bladder, very rarely in the intrahepatic ducts. (*b*) *Size;* they range upward from the size of sand or gravel; a calculus 12 inches in length is recorded; the heaviest reported was 135 gm. (*c*) *Form;* they are usually round and rarely spinous; when multiple they are polygonal and facetted. Intrahepatic calculi may be cylindrical or branching like coral. (*d*) Their *number* averages a dozen; according to Riedel 44 per cent. are single; Otto reports 7802 stones in 1 case. In Chopart's case the liver could scarcely be cut because of the numberless stones in the intrahepatic ducts. (*e*) *Consistence;* gall-stones can usually be indented by the finger. (*f*) The *time required* for their growth is about six months. (*g*) *Chemistry;* the most common form consists of *cholesterin* and *bile pigment* with lime as a cementing substance; these calculi are small, numerous, usually yellow, laminated and contain about 75 per cent. of cholesterin. Pure *cholesterin* stones are not common; they are translucent, become opaque when dry, are light in weight and color and are combustible; they present a crystalline fracture like that of camphor, they dissolve in ether and alcohol, from which the crystals are precipitated and produce a red tinge on addition of chloroform and sulphuric acid; cholesterin comes from the blood, bile or gall-bladder mucosa. *Pigmentary stones* are

uncommon; bilirubin imparts a brownish and biliverdin a greenish tint to them. *Calcium carbonate* stones are rare, are grayish in color and heavy. Stones in the liver are constituted of calcium and bilirubin. Traces of magnesium, bile acids, fatty acids, iron and copper have been found. The nucleus is mucin, bacteria, epithelium, blood clot, cholesterin, calcium, bile pigment or rarely a foreign body.

Symptoms.—Kehr and Riedel assert that 95 per cent. of persons with gall-stones have no symptoms, especially elderly subjects with atrophic gall-bladders, and stones very rarely may be felt in the gall-bladder, or be found in the feces in these cases. The writer believes that latency is less common than above figured and that *gastric symptoms are exceedingly common* (75 per cent.).

Biliary Colic.—(*a*) Colic is usually *sudden in onset* and frequently occurs at night. Many women state that the pain is more severe than labor pains. It begins in the epigastrium and may radiate to the back, navel, hypogastrium, left side and even to the arms, legs, testes or chest. Irregular or large stones cause the greatest agony, controlled only by large or repeated hypodermics of morphine. The pain usually endures for a few hours and may soon recur. The cause of biliary colic is *mechanical*—the attempt of the stone to migrate produces the spasm of the cystic and common ducts during its passage—or *inflammatory*, cholecystitis starting the stones moving by the exudation it induces; pain is less or shorter if the ducts are patulous. Quiescent gall-stones may be started onward by palpation of the gall-bladder, trauma, exercise, reduction of flesh, menstruation or delivery. (*b*) *Reflex nausea* and *vomiting* are frequent; they may indicate cholecystitis. Vomiting relieves the pain, whence the frequently erroneous diagnosis of gastralgia. A cramp of the stomach has been seen with the *x*-rays. Torsion of the cystic duct may cause vomiting but not colic. (*c*) *Chills and fever* occur in 66 per cent. and indicate bacterial inflammation. (*d*) The *gall-bladder* is *tender*, there being hyperesthesia over the ninth costal cartilage or over a point two-thirds of the distance between it and the navel, and around the chest to the spine. In 33 per cent. the gall-bladder is palpable. The right rectus muscle is rigid (*défense musculaire*). (*e*) *Icterus*, present in but 15 per cent., may be calculous, infectious or compressive. (*f*) *Other symptoms* of the paroxysm are rapid pulse, sweating, prostration, concentrated urine, constipation, abdominal retraction and, after the seizure, marked prostration. Attacks of migraine are not infrequent. There may be reflex coughing or reflex contraction of the pulmonary capillaries, which overtaxes and dilates the right heart. The patient may collapse and the writer observed death in an attack. Glycosuria, albuminuria, leukocytosis and splenic swelling are inflammatory in origin. The stone may fall back into the gall-bladder or pass into the bowel if it measures under 1 cm.; confusion with lumps of olive oil, given to relieve the colic, is unnecessary; a round calculus suggests that it is the only one. Stones passing into the bowel may be dissolved, unless coated with cholesterin. The *great importance of gall-stones lies in their complications:*

Complications.—They may be grouped as *mechanical* or *inflammatory*, but are best combined topographically.

1. Gall-bladder.—Mechanical complications include pressure on the duodenum or pylorus and rupture of the gall-bladder, which, however, are generally inflammatory; pressure on the common duct; hyperchlorhydria and hypersecretion and cancer of the gall-bladder (*q. v.*). Inflammatory complications are more serious; all forms and sequences of cholecystitis (*q. v.*) may develop, as hydrops, empyema, gangrene, ulceration, deformity, hemorrhage, atrophy or calcification; also pylephlebitis, adhesions producing pyloric obstruction with hyperchlorhydria, dyspepsia and sometimes hemorrhage into the stomach; adhesions causing duodenal obstruction; and peritonitis, either local (near the gall-bladder or subphrenic abscess) or diffuse. A friction-rub may be heard over the gall-bladder a day or two after the colic. The distended gall-bladder rarely exceeds the size of the fist, is pyriform, can be moved laterally and moves with the liver, from which it may seem separated by a groove; it may be confused with appendicitis, the kidney, ovarian cyst or ascites and has been found in a femoral hernia.

2. Cystic Duct.—Mechanical impaction causes icterus by catarrh of the common duct (in 12 per cent.) or by directly compressing it. The duct may become twisted, stenosed or obliterated.

3. Common Bile Duct.—Its mechanical occlusion is usually preceded by colic; impaction occurs oftenest at its lower end (50 to 67 per cent.), and is caused most frequently by 1 stone; 88 have been found. Permanent occlusion by stone in the common duct or in the cystic duct pressing upon it causes marked icterus, usually without sepsis. The occlusion may be partial or intermittent, by a ball-valve action of the stone, described by Osler and Fenger, which allows some bile to enter the gut and some bacteria to enter the duct, thus adding bacterial to mechanical injury. Usually the icterus is sudden in onset and slow in subsidence. In partial or intermittent obstruction there is often the *intermittent hepatic fever*, first described by Charcot and comparable to urethral fever. During the paroxysms the fever rises to 103° or 105°, often with chills, sweats, leukocytosis, vomiting and increased tenderness, pain and icterus. The spleen may swell, and also the liver from saccular dilatation of the channels and cholangitis. Osler correctly insisted that these symptoms occur without suppurative cholangitis, though with infection. The *gall-bladder* is not enlarged, but *is usually shrunken*, following *Courvoisier's law;* Courvoisier found the gall-bladder enlarged in 92 per cent. of non-calculous obstruction of the common duct and shrunken in 80 per cent. of calculous obstruction; this is caused by infection (Hanot). In some cases suppurative cholangitis may complicate stones of the common duct, with remittent fever (rather than intermittent fever), enlarged liver, subcapsular abscess, septic symptoms and more rapidly fatal course. Cicatricial stenosis of the common duct is rare; pylephlebitis may develop. Malaria is excluded by examination of the blood; malignancy will be considered under tumors of the pancreas, though in common duct obstruction the nutrition is little impaired. Practically, stress may be placed on (*a*) the periodic fever, (*b*) the icterus, sudden in onset and subsiding slowly, and (*c*) tenderness (without pain over the liver).

BREWER'S DIAGNOSIS OF DISEASES

Pathological condition.	Pain.	Fever.	Vomiting.	Jaundice.	Tumor of gall-bladder.
I. Calculous Disease.					
(*a*) Stone in healthy gall-bladder, ducts free.	No.	No.	No.	No.	May be present from large number or size of stones.
(*b*) Stone in healthy gall-bladder, cystic duct temporarily obstructed.	May be absent; generally present during obstruction; paroxysmal.	No.	May be present when colic occurs.	No.	May be present from distention.
(*c*) Stone impacted in cystic duct.	No.	No.	No.	No.	Present; may attain large size.
(*d*) Stone in hepatic duct.	Frequently present; irregular type.	Occasionally present.	May be present during pain.	Frequent.	No.
(*e*) Stone in common duct; acute obstruction.	Present; acute, paroxysmal, radiating to back.	Generally present.	Present.	Present.	No.
(*f*) Stone in common duct; movable; chronic.	Periodic attacks of acute radiating pain	Present with chills and sweats.	Present.	Present; intermittent.	Rarely present.
(*g*) Stone in common duct; impacted; chronic.	May be absent; frequently present early; may be intermittent; variable.	May be present; variable.	Often present.	Present; progressive; may vary in intensity.	Rarely present.
II. Inflammatory Disease					
(*a*) Cholecystitis subacute.	Present; paroxysmal during periods of cystic duct closure from stone or swollen mucous membrane.	Present during attacks of colic.	May be present.	No.	Present during attacks of cystic duct obstruction.
(*b*) Cholecystitis acute.	Acute paroxysmal radiating pain; extending to back and shoulder; may be very severe.	Present with chills and sweats.	Present often; severe.	No.	Present; tenderness; often muscular rigidity.
(*c*) Cholecystitis chronic (empyema of gall-bladder).	Severe radiating pain at first; may disappear later; tendency to recur.	Present; severe at first, may diminish later.	Present at first.	No.	Present; with tenderness; may attain large size.
(*d*) Cholecystitis in previously diseased and contracted gall-bladder.	Present; often severe; paroxysmal.	Present; often with chills and sweats.	Present.	No.	No (occasionally present dūe to pericystic exudate).
(*e*) Cholangitis of hepatic and common ducts.	May be absent; generally present when obstruction exists, or severe infection; tenderness and pain over liver in intrahepatic cholangitis.	Present; chills; sweats; severe prostration; general sepsis.	Present.	Present; variable.	No.
III. New Growths.					
(*a*) Carcinoma of gall-bladder.	No; may occur late in disease.	No.	No.	Present late (portal glands).	Hard, irregular, movable tumor at first, later diffuse infiltration.
(*b*) Tumor of cystic duct.	No; may occur late.	No.	No.	No.	Present when obstruction exists.
(*c*) Tumor of hepatic or common duct.	No; may occur late.	No.	No.	Present; progressive.	May be present from distention with bile.
(*d*) Tumor of neighboring viscera producing chronic obstruction of common duct.	No; may occur late.	No.	No.	Present; progressive; may become extreme.	Present; generally from distention with bile.

OF THE GALL-BLADDER AND DUCTS.

Urine.	Stools.	Liver.	Spleen.	Ascites.	Remarks.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Generally discovered by accident; often unrecognized.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	All symptoms promptly relieved as soon as obstruction removed.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Hydrops of gall-bladder often unrecognized.
Contains bile pigment at times.	May be clay-colored if obstruction occurs.	Frequently enlarged.	Not enlarged.	No.	Diagnosis extremely difficult; symptoms generally due to co-existing cholangitis.
Contains bile. pigment.	Clay-colored.	Not enlarged.	Not enlarged.	No.	Symptoms rapidly disappear when stone passes papilla.
Contains bile pigment.	Clay-colored.	May be enlarged during attack.	May be enlarged from pressure of stone on vein.	No.	"Fièvre intermittente hépatique" of Charcot resembles malaria; all symptoms disappear during interval.
Contains bile pigment.	Clay-colored.	Enlarged.	May be enlarged.	No.	Condition may remain for many years; may only be jaundice with digestive disturbances and loss of weight; history of previous attacks (?). Ascites may be present from pressure of large stone or possibly from hydremia.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Tenderness over gall-bladder; tendency to recurrence; generally associated with stones in gall-bladder.
May contain albumin and casts.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	May follow typhoid or other septic diseases; onset often sudden; rapid development of severe symptoms resembling appendicitis; may be necrosis of walls of gall-bladder with perforation; local or general peritonitis.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Frequently follows acute cholecystitis; occasionally becomes quiescent, presenting practically no symptoms.
Negative.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	Generally tenderness over gall-bladder area, but no tumor; local peritonitis; diagnosis often extremely difficult.
May contain bile pigment, albumin and casts.	May be clay-colored.	Enlarged.	Enlarged (sepsis).	No.	Often follows severe infections of gall-bladder; generally associated with stones in common or hepatic duct; severe sepsis; generally fatal in virulent infections (streptococcus).
Negative (at first).	May be clay-colored late.	Enlarged late in disease.	May be enlarged late (pressure on vein).	Present late.	Digestive disturbances, progressive loss of weight and asthenia; cachexia; rapidly fatal.
Negative.	Normal.	Not enlarged.	Not enlarged.	No. (?)	Very rare; both benign and malignant growths have been reported; diagnosis difficult.
Contains bile pigment.	Clay-colored.	May be enlarged.	Not enlarged.	May be present late.	Very rare; diagnosis difficult.
Contains bile pigment.	Clay-colored.	Enlarged.	May be enlarged late.	Present late.	Malignant tumors most common; chronic interstitial pancreatitis from previous infection of biliary passages may remain after cause has disappeared; enlarged portal glands; Hodgkin's disease.

4. Ampulla of Vater.—Calculous retention in the ampulla may obstruct the flow of the pancreatic juice, though the accessory duct may carry it into the intestine; retention may allow bile to enter the pancreatic duct, and result in (*a*) acute pancreatitis; (*b*) chronic induration of the head of the pancreas resembling malignancy; (*c*) pancreatic fibrosis with pancreatic calculi due to stasis and infection; or (*d*) rarely glycosuria.

5. Intestinal Obstruction.—The obstruction may not occur for some time, the stones sometimes receiving new layers of magnesia or phosphates; it is in the ileum in 66, duodenum in 21, ileocecal valve in 10 and sigmoid in 3 per cent.; 50 per cent. of cases die. (See page 551.)

6. Fistulæ.—Courvoisier assembled 499 cases. The external form constitutes about 50 per cent. of cases; they are most often located near the navel and about 40 per cent. of them heal. In the duodenal form (28 per cent.) the fistula is between the duodenum and gall-bladder and less often between the common duct and gall-bladder. In 13 per cent. there is a fistulous opening into the colon. Courvoisier and Graham collected 34 cases of bronchial fistula. In rare cases stones enter the stomach (5 cases recorded, Snively, 1903), portal vein (as in the autopsy upon Ignatius de Loyola), hepatic artery, kidney, urinary bladder (200 gall-stones evacuated in the urine in the case of Barraud-Palletan), pericardium, retroperitoneal tissue, vagina or even the pregnant uterus.

Diagnosis.—The diagnosis is easily made when colic, vomiting, tenderness of the gall-bladder and icterus are present. The *x*-rays are positive in 50 per cent. (Case).

1. The Colic.—This is most apt to be confused with (*a*) *gastralgia* or *hepatic neuralgia;* due regard for other symptoms of gall-stones and reserve in the diagnosis of gastralgia save many mistakes. (*b*) *Gastric or duodenal ulcer,* hyperchlorhydria and hypersecretion (*q. v.*) are easily distinguished. (*c*) *Lead colic* has the characteristic gingival lead line, obstinate constipation and often neuritic manifestations. (*d*) *Tabetic crisis* is attended by the Argyll-Robertson pupil, absent knee-jerk and ataxia. (*e*) Renal colic is distinguished by pain reflected down the ureter, hematuria and pyuria; and Dietl's crisis in floating kidney, by bimanual examination of the abdomen. (*f*) *Mucous colic* is differentiated by the characteristic stools. (*g*) *Appendicitis* is characterized by its cardinal symptoms (*q. v.*); Ochsner finds 10 per cent. of his appendicitis cases complicated by gall-stones and 33 per cent. of his gall-stone cases by appendicitis. (*h*) *Acute pancreatitis* (*q. v.*) which may complicate gall-stones, causes greater collapse and more frequent intestinal obstruction. (*i*) *Febrile syphilis* may simulate gall-stones as may also the abdominal crises observed in angioneurotic edema.

2. Other Biliary Affections (see pages 594 and 595).

Prognosis.—The prognosis is difficult to formulate. Stones may pass without recurrence of colic and the passage of smooth calculi may justify some hope that they are the only ones. The first attack is usually the worst. Fever, infection, cancer and obstruction must be covered in forecasts to patients refusing operation. Riedel held that 10 per cent. recover spontaneously, while 90 per cent. require surgical intervention.

Treatment.—1. PREVENTION.—Exercise, deep breathing and plenty of water aid the flow of bile. Lacing is a factor to be regarded. Digestive derangements should be corrected. Alcohol is contra-indicated. The predisposing gall-bladder inflammation seldom comes under the physician's care, but in suspicious cases, sodium salicylate, gr. x–xv t. i. d., given alternate weeks, acts as a cholagogue and antiseptic. Hexamethylenamine, ℥j per diem, is excreted by the bile. Potassium iodide, and sodium phosphate, ℥ss–j are beneficial.

2. COLIC.—Hypodermics of morphine must be given, gr. $\frac{1}{4}$ being combined with nitroglycerin gr. $\frac{1}{100}$ and (for the first injection) atropine gr. $\frac{1}{100}$; repeated doses of morphine and nitroglycerin are often necessary. Inhalation of chloroform is indicated in severe cases until the morphine acts, but none of these drugs should be left in the possession of any patient. A full hot bath and hot fomentations over the liver are valuable accessories. Drinking warm water facilitates vomiting, which relieves the pain.

3. MEDICAL TREATMENT.—This in no way influences the *solution of gall-stones.* Durande's solvent (oil of turpentine 1 part to ether 4 parts) is useless and, like chloroform, is only antispasmodic. Olive oil only facilitates their final passage. Salicylates, calomel, salines (*v. s.*) and various "cures," as at Carlsbad, Vichy, Kissingen, Las Vegas or Bedford Springs, are useful in quieting concomitant or causative inflammation. Local massage is obviously injurious.

4. SURGICAL TREATMENT.—The author believes that gall-stones should be operated on when recognized, thus saving time, suffering and danger. Riedel's argument for early operation—the removal of the calculi while they remain in the gall-bladder—holds good, as it is the only salvation against perforation, cholemia and carcinoma; it is refused, however, in general practice. Hydrops, empyema of the gall-bladder and pericholecystitic abscesses, cholangitis, incapacitation, jaundice, impaction, and frequent resort to morphine are strong indications. Corpulent men do not bear the operation well. Women who have borne children lend themselves well to surgical procedure. If possible one should refrain from operating in cases of diabetes, arteriosclerosis, chronic nephritis and diseases of the lungs and heart.

In Mayo's 4000 operated cases the mortality was 1.8 per cent. in uncomplicated cases, while those complicated by suppuration or cancer brought the average mortality to 2.75 per cent.; the death-rate in operations on the common duct was 8 per cent. These figures appear to the writer as the best possible argument for early operation. Cholecystotomy is often inferior to cholecystectomy.

CHOLECYSTITIS.

Etiology.—Inflammation of the gall-bladder may occur before, after, with or without inflammation of the bile passages. (*a*) *Predisposing factors are gall-stones* (especially large ones which cannot pass, which cause 65 to 90 per cent. of cholecystitis and cholangitis), bile stasis, foreign bodies or previous attacks. (*b*) The *exciting cause* is *bacterial,*

the colon, typhoid, pyogenic, pneumococcus, and other microörganisms. They may ascend the bile channels or enter with the blood current. Inflammation from typhoid infection (Louis and Andral) may occur without actual typhoid or may develop twenty years later.

Symptoms.—The symptoms vary with the intensity of infection, which is almost synonymous with the type (simple, membranous, suppurative, phlegmonous, ulcerative or gangrenous). In light forms the symptoms may not be recognized; this corresponds to postmortem adhesions in cases with no history of cholecystitis. In other, perhaps more severe cases, symptoms are obscured by the causative typhoid or by gall-stones. In the average case there are: (*a*) *pain over the part,* which may be severe or paroxysmal and may radiate toward the appendix or back. (*b*) *Tenderness* as in gall-stones; it is often first general and then local and the right rectus is tense; it is clearly elicited by a thrust over the part during inspiration. (*c*) *Fever, increased pulse-rate, vomiting* or nausea; fever is absent in mild infections; in typhoid, cholecystitis may be confused with typhoid relapse. There may be *colic.* (*d*) There may be a *palpable gall-bladder.* Its contents may be purulent (empyema cystidis felleæ) or serous (hydrops cystidis felleæ). Hydrops may enlarge the gall-bladder to the size of the fist or in extreme cases so that it weighs 50 to 60 pounds and fills the entire abdomen, simulating ascites; the fluid is alkaline or neutral, serous or mucoid. If very tense the gall-bladder may not fluctuate or if very lax may not be palpable (see FLOATING KIDNEY). There may be pus in the gall-bladder without enlargement. In chronic cases it may shrink. (*e*) *Severe sepsis* may intervene in diffuse suppurative cholangitis or pylephlebitis, evidenced by septic fever, chills, leukocytosis, bacteriemia, swollen liver and spleen, nephritis and ulcerative endocarditis. Icterus occurs in 33 per cent. (See pages 594 and 595.)

Prognosis.—Many cases with cholangitis die in spite of operation. Mild cases may subside, but recurrence and formation of gall-stones are frequent. Under expectant therapy the outlook is serious in severe cases, for perforation, peritonitis, adhesions (in 75 per cent.) or pyloric obstruction may result.

Treatment.—Rest, milk diet, local heat and morphine may suffice for light cases, but in the more severe infections drainage is indicated.

CHOLANGITIS. CATARRHAL ICTERUS. CONGENITAL OCCLUSION.

Suppurative Cholangitis.—It usually coexists with suppurative cholecystitis and infection is usually *hematogenous,* but may be ascending—cholangitic sepsis (*v.* pages 594 and 595.)

Acute Catarrhal Jaundice.—Cholangitis catarrhalis, icterus simplex, results from gastroduodenal catarrh which causes swelling with obstruction of the papilla; in the few autopsied cases a small plug of mucus occludes the opening of the common duct; Eppinger found swelling of the lymphoid follicles where the duct lies in the wall of the bowel. Its usual causes are practically those of acute gastritis or enteritis. Favoring factors include passive congestion, acute infections, toxic causes, nephritis

and other diseases of the liver or bile tracts. Epidemic forms seem to be a separate type of infection (*v.* WEIL'S DISEASE).

Symptoms.—*Gastro-intestinal catarrh* occurs first in half the cases and after a few days *icterus* appears, with its usual symptoms, sudden decolorization of the stools and bright yellow staining of the skin and urine; in the other half, there is primary infectious catarrh of the bile ducts. The liver is not enlarged at first, but may become slightly swollen later; there is no pain or emaciation, the spleen and gall-bladder are seldom enlarged and the *diagnosis* depends on the mode of onset in young, healthy subjects, sequence of symptoms and exclusion of other causes of obstruction; in advanced life catarrhal jaundice is less common than gall-stones and cancer. (See page 594.)

Prognosis.—Cases may clear in a week or last five months; a course of over the average—four to eight weeks—renders the diagnosis uncertain. Cases with fever may run a prolonged course. Death rarely occurs from hemorrhage, exhaustion or sudden heart failure.

Treatment.—(*a*) *Causative* factors, as gastritis or stasis, are appropriately treated; dyspeptic symptoms yield most readily to gastric lavage and fractional doses of calomel at night followed by a mild saline in the morning; active catharsis augments or initiates catarrh.

℞—Acidi hydrochlorici diluti . . . . . ℥j
Tincturæ nucis vomicæ . . . . . ℥iv
Tincturæ gentianæ co. . . . . q. s. ad. ℥iv
M. et S.—One teaspoonful after meals in half a glass of water.

The green "bilious stools" from calomel are not due to bile but to mercuric sulphide. (*b*) In the *diet* fats are particularly to be avoided, as neither the stomach nor intestine can digest them; tea, coffee, alcohol and coarse foods should be interdicted; skimmed milk, well-cooked carbohydrates, toast, eggs and mealy soups may be given; meat is often poorly tolerated, seemingly because of exclusion of the pancreatic secretion. (*c*) As long as acute dyspepsia exists, *rest in bed* is indicated, in order to maintain nutrition on the lowest possible diet. (*d*) The bowels are regulated by *copious injections* of water, to incite peristalsis and dislodge the obstructing mucus plug. (*e*) The *itching* is allayed by 1 per cent. carbolic solution, the use of which requires care, as the urine sometimes becomes cloudy; acetanilide, gr. v, q. i. d.; potassium bromide, ℥ss, well diluted, by rectum once or twice daily; an occasional hypodermic of pilocarpin hydrochloride gr. $\frac{1}{8}$; and by warm baths.

Chronic Catarrhal Cholangitis.—(*v. Gall-stones, Pancreatic Cancer and Icterus.*)

Congenital Occlusion of the Bile Ducts.—Seventy-six cases are recorded (Howard and Wolbach), due perhaps to cholangitis or congenital atresia. Icterus develops within a month after birth and death results in weeks or months from cholemia or hemorrhages.

TUMORS OF THE GALL-BLADDER AND BILE VESSELS.

I. Cancer of the Gall-bladder.—Tumors other than cancer are rare, as sarcoma, papilloma, fibroma, lipoma or adenoma.

Etiology.—The most striking etiological factor is *gall-stones,* present in 70 to 91 per cent. (Courvoisier); the estimate that 9 per cent. of cases of gall-stones result in cancer of the gall-bladder seems very high; 80 per cent. occur in women over fifty years of age.

Pathology.—The tumor may be scirrhus or medullary, but usually adenocarcinoma; spheroidal and squamous types are rare.

Symptoms.—(*a*) *Gall-stones* which irritate the mucosa and cause most cases, usually produce no symptoms, though previous colic may be noted in the history. (*b*) A *tumor of the gall-bladder* is palpable in over 66 per cent., first as a smooth oval and later as a larger and more nodular swelling; it is usually due to the growth, for the gall-bladder itself is generally shrunken. The fundus is the usual point of origin, being most irritated by calculi, much more rarely the neck of the gall-bladder or least frequently the cystic duct. The tumor appears below the edge of the liver with which it moves. There may be local discomfort and, in rare cases, severe pain like that of gall-stones. (*c*) *Cachexia* appears with all its attendant symptoms.

Complications.—*Secondary growths* in the liver occur in 58 per cent. by lymphogenous routes or by invasion from contiguity and sometimes cause hepatic enlargement; from the liver extension by the hepatic veins may occasion systemic metastases. *Icterus* occurs in 69 per cent.; cholemia is a common termination. Growths in the peritoneum, lungs or pleura, abdominal lymph glands and into the colon occur in about 10 per cent. each. Warthin records adrenal metastases with vitiligo and pigmentation like that of Addison's disease. Pressure on the pylorus may cause its stenosis, vomiting or dyspepsia. *Ascites* occurs in 25 per cent. and is due to compression of the portal vein or to malignant peritonitis. Suppurative cholangitis may develop, with death from sepsis. The differential *diagnosis* is considered under gall-stones. The clinical course averages six months. *Treatment* is palliative, unless the affection is diagnosticated early or is accidentally found in operating for other conditions.

II. Tumors of the Extra-hepatic Bile Ducts.—Rolleston collected 80 cases, and Zezas 34 cancers at the junction of the three great bile ducts. These tumors are small, seldom infiltrate and are of the columnar type. The common duct is most often affected, the hepatic duct less and the cystic duct least frequently. The salient symptom is intense *icterus,* which develops early, increases steadily, occurs in nearly all cases, causes icteric necrosis of the liver cells in the central zone and precipitates early cholemia before metastases or cholangitic sepsis have time to develop. The gall-bladder is almost always dilated anatomically in tumors of the common duct and is felt clinically in over 50 per cent. Pain is not common, though gall-stones may be simulated. Ascites, dyspepsia and gastro-intestinal hemorrhages from cholemia may develop. Differentiation from duodenal or pancreatic tumors (*q. v.*) is usually impossible. *Treatment* consists of draining the gall-bladder or extirpating the tumor.

DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS; FAT NECROSIS.

Etiology.—*Infection* ascending from the intestines is the leading cause. (*a*) *Calculus obstructing the common duct,* by allowing bile to regurgitate into Wirsung's duct, may cause acute pancreatitis (Opie), especially when the gall-bladder, the natural reservoir for obstructed bile, is shrunken; gall-stones cause 42 per cent. of cases of acute fat necrosis. Bile salts may initiate inflammation and fat necrosis, without infection. The accessory duct of Santorini may carry off the pancreatic juice when there is obstruction of Wirsung's duct, but it is too small for much compensation in 66 per cent. of cases; (*b*) *metastatic infection* is an infrequent cause; the author observed one case in which an acute ulcer of the leg was the atrium; (*c*) suppuration or *ulceration in contiguous tissues* may be causative.

Predisposing Factors.—(*a*) *Age and sex;* 90 per cent. occur in *males beyond middle life.* (*b*) *Obesity;* (*c*) *alcoholism;* (*d*) *trauma;* (*e*) gastro-intestinal catarrh; and (*f*) parturition in 6.6 per cent. of Peiser's 128 cases.

Symptoms.—The clinical picture was clearly described and defined by Fitz (1889), though cases were reported earlier by Oppolzer. The *onset is sudden* in adult, stout males who have enjoyed previous good health or who have a history of alcoholism or of gall-stones.

1. *Epigastric pain* is the *initial symptom;* it is very sudden and agonizing, constant or paroxysmal and may be confused (or coincident) with gall-stone colic, though it is even more intense; it is diffusely epigastric or rarely over the head of the pancreas. The author has seen radiation into both axillæ. The intensity of the pain is referable to pressure on the celiac plexus (neuralgia or neuritis celiaca).

2. *Epigastric tenderness* is usual, often over the head or sometimes more over the tail of the pancreas, whence the inflammation may extend to the left pleura with tenderness and friction. The recti are tense and the epigastrium is swollen.

3. *Vomiting* is early, severe and sometimes brings up blood.

4. *Collapse* follows; it often threatens immediate dissolution and too often prevents surgical interference. It is due to pressure on, or actual inflammation of, the closely contiguous celiac plexus and semilunar ganglia. In one of the author's cases there were relapsing shock with cyanosis, dyspnea and a pulse of 140, which was so weak that no surgeon would operate during the two weeks before recovery. Collapse usually causes death in 2 to 4 days in unoperated cases.

5. *Fever* is not constant; there may be no elevation or an irregular rise to 103° or 104°. Chills are most common in late suppuration.

6. The *pancreas can rarely be felt* because of its deep position and the tense and tympanitic epigastrium.

7. The *bowels* are constipated, simulating obstruction, though flatus is passed. Operation or autopsy at this stage shows an acute pancreatitis

with swelling, a variegated yellowish-red or black color, exudation of fibrin, pus cells, blood (hemorrhagic pancreatitis) and areas of acute *fat necrosis* (Balser, 1882). Fat necrosis is caused by the fat-splitting steapsin (Langerhans), which produces glycerin and insoluble fatty acids from the pancreas and contiguous adipose tissues, *i. e.*, omentum, mesentery and peritoneum; the glycerin is absorbed, the fatty acids are precipitated with lime as opaque white areas of necrosis and some of the ferment occasionally escapes into the blood, producing toxemic symptoms or necrosing fatty tissue elsewhere, as in the pericardium or skin.

8. If the patient lives a *localized peritonitis* develops in the upper abdomen; pus may fill the lesser peritoneal cavity; the pain may become general from diffuse fat necrosis or dyspnea may develop from diaphragmatic pleurisy.

9. *Septicopyemic symptoms* may appear, as rigors, hectic fever, pleurisy, icterus, diarrhea and loss of weight and strength; the author saw splenic tumor, nephritis and universal lymph adenitis.

10. Acute *glycosuria* was present in one of the author's cases.

In the third week *rupture* into the colon may occur, attended by lancinating pain and intestinal hemorrhage.

Suppurative types, of which Körte collected 46 cases, may begin gradually, without acute gangrene, fat necrosis or hemorrhage; the symptoms are less acute and severe. The suppuration is localized or there is a diffuse purulent infiltration; it may cause pylephlebitis, liver abscess, subphrenic abscess, burrowing of pus into the peritoneum, loin (which Körte considers rather characteristic), peripheral tissues or alimentary tract. Protracted suppuration is said to cause glycosuria, skin pigmentation and anasarca. Körte collected 40 cases of *acute gangrenous pancreatitis*.

Diagnosis.—The cardinal features are the sudden onset, violent epigastric pain, distention and tenderness, vomiting and collapse. The absence of indican, leucin and tyrosin in the urine is suggestive. Robson finds calcium oxalate crystals in 30 per cent. of all varieties of pancreatic disease. The *pancreatic reaction* of Cammidge is unreliable.

Differentiation.—(*a*) *Acute poisoning* is eliminated by the history and gastric contents. (*b*) *Intestinal obstruction* is less severe in onset; collapse is attended by distention, intestinal rigidity or peristalsis above the obstruction; indicanuria is usual; epigastric distention alone is unusual; in one pancreatic case, the author saw obstruction lasting nine days. Operation may be necessary for diagnosis. (*c*) *Gall-stone colic* is rarely as severe as pancreatic pain; collapse or epigastric distention is infrequent. (*d*) *Perforating ulcer*, mesenteric embolism and appendicitis must be considered.

Prognosis.—The malady generally evinces a severe progressive tendency. A fatal attack may follow milder seizures. At autopsy fibrous tissue and crystals or granules of hematoidin may be evidence of previous inflammation. Death may occur within a few hours to three days from collapse, in two or three months from sepsis or in six months from diabetes (Fitz). The pancreas may slough into the intestine, as in Trafoyer's and Chiari's cases, with recovery.

Treatment.—Rectal feeding and stimulation, morphine hypodermically and heat are indicated during the initial shock. Though early operation is dangerous because of shock and hemorrhage, it is indicated lest local extension or general toxemia develop; the surgical mortality is 60, and the medical, 90 per cent.

CHRONIC PANCREATITIS.

Etiology and Pathology.—(*a*) *Gall-stones* in the common duct may induce chronic catarrh of Wirsung's duct, leading to chronic pancreatitis (Robson). (*b*) *Cancer*, gastroduodenal *catarrh* and *ulceration* (alcoholism, syphilis and pancreatic calculi) may produce the same results. In these instances *ascending infection* appears to be the potent factor, causing *interlobular fibrosis* around the pancreatic lobules (Opie); diabetes is a late and uncommon sequence. (*c*) *In liver cirrhosis* and *hemochromatosis* (see pages 572 and 574) *an interacinar fibrosis* occurs, developing in the lobules, diffusely invading the islands of Langerhans and causing glycosuria; it does not result from obstruction of the common duct.

Symptoms.—Riedel, who first described the affection, holds that it is found more frequently with increasing thoroughness of operations for gall-stones. Symptoms may be lacking. They vary with the cause. In catarrh due to gall-stones there is a history of *colic, jaundice* and sometimes *intermittent fever. Tenderness* in the epigastrium, a fulness above the umbilicus and loss of flesh are noted, and if pancreatic symptoms predominate the pain passes from the epigastrium to the left side, to the scapular or renal region. If gall-stones are not the cause, the pain may be moderate or absent. The gall-bladder may enlarge from obstruction of the common duct, when the liver becomes enlarged. Marked *enlargement of the spleen* is frequent, bile is found in the urine in 60 per cent., *calcium oxalate crystals* in 40 per cent. and glycosuria in 5.5 per cent. (Robson). Opie found glycosuria but once in 22 cases. A *tumor-like induration* of the head of the pancreas simulates cancer because of the emaciation and deep icterus; differentiation may be impossible, even during laparotomy; it is more common in subacute than in chronic cases. As in tumor, excessively copious, pale stools, laden with fat and undigested muscle fibers, are noted. Polyuria is reported.

Prognosis.—Without surgical intervention death is likely to occur from emaciation, cholemia, diabetes or hemorrhagic diathesis.

Treatment.—Drainage of the gall-bladder or anastomosis is indicated. Robson operated on 62 cases and Quénu and Duval on 62 cases with 13 per cent. mortality. Hemorrhage is a dreaded complication.

PANCREATIC APOPLEXY.

Pancreatic hemorrhage has been described by Spiess (1866), Klebs (1870) and Zenker (1876); Anders (1899) collected 40 cases. The anemias, syphilis, alcoholism, acute infections and local lesions of the pancreas seem to be *predisposing causes;* 66 per cent. occur *in men.* Doubtless many reported cases are acute hemorrhagic pancreatitis.

Sudden onset, collapse, epigastric pain, ileus, and normal or subnormal temperature mark the affection; death sometimes occurs within a few hours. If the patient survives the initial stage suppuration may develop in the pancreas and tissues into which the hemorrhage burrows; hemorrhagic cysts may form. The treatment is surgical.

LITHIASIS.

About 100 cases of sialolithiasis pancreatica are recorded. The *etiology* is probably infection of Wirsung's duct; stagnation of secretion is a predisposing factor, whence the occasional association with gall-stones, pancreatic inflammation and tumor.

Symptoms and Diagnosis.—Confusion with gall-stones is often inevitable, for colic without icterus is indicative of either condition and their coexistence is not uncommon. The pain may radiate to the left side and shoulder. In some cases stones were found in the feces during life, in others at autopsy. Salivation is reported. In Lichtheim's case there were colic, diarrhea and glycosuria (found in 45 per cent. of cases). The stones are single or multiple (even 300), whitish-gray, round, oval, irregularly outlined or branching, hard or friable and vary from almost impalpable gravel to the size of a hazel-nut. They consist largely of calcium carbonate and phosphate. Shattuck described an oxalate calculus. The *x*-rays may detect them. Their sequelæ are obstruction and inflammation of Wirsung's duct and acute suppurative pancreatitis. The treatment in fortunately diagnosticated cases is surgical.

PANCREATIC CYSTS.

To Nicholas Senn (1885) belongs the credit of establishing the clinical picture. Bessel-Hagen (1900) collected 149 cases.

Etiology and Pathology.—(*a*) Many reported cases are extra-pancreatic, especially the *traumatic* cysts, which constitute three-quarters of cysts. (*b*) Duct obstruction (*ranula pancreatica,* Virchow) by calculi, strictures, parasites, tumors or inflammation in the smaller ducts is an etiological factor. (*c*) *Cystadenoma* is the cause of most true cysts (Lazarus). (*d*) Cysts rarely result from an *old hemorrhage.* In all forms the cyst causes parenchymatous atrophy. (*e*) *Sex:* Traumatic and inflammatory forms are most frequent in men and the cystadenomatous type in women. (*f*) *Age:* Over 50 per cent. are found in the third decade of life, though there is a congenital form, sometimes with cystic liver and kidneys (Richardson).

Symptoms and Diagnosis.—1. As to the *cyst,* (*a*) a *deep retroperitoneal* swelling is found, (*b*) which is more or less *central,* and *above the navel* in 87 per cent., where other cysts are most uncommon; it lies behind or between the inflated stomach and colon, sometimes below the colon but rarely above the stomach; very large cysts which contain 10 to 20 quarts may fill the abdomen. Proliferative cysts developing in the tail of the pancreas, point toward the spleen or left kidney. (*c*) Its form is *round* or *oval* and *smooth;* (*d*) its consistence *varies;* (*e*) it is *immobile* on

palpation and on inspiration. (*f*) The *fluid* is alkaline, 1.010–25 sp. gr., mucoid, blood-stained in 82 per cent. (Körte), and in 45 per cent. contains one or more pancreatic ferments (diastatic, saponifying or tryptic) of which the latter alone is characteristic, for the other ferments occur in various exudates. The fluid sometimes contains fat and cholesterin; aspiration is dangerous, because the tension of the cyst is great and fat necrosis or peritonitis may result from leaking after exploratory puncture.

2. So-called *pancreatic symptoms* are rare, as emaciation, salivation, glycosuria or stools laden with undigested fat or muscle fibers.

3. Symptoms of *peritonitis* or *intestinal obstruction* may occur in traumatic or inflammatory types and after rupture. The author saw a case in which the picture after each rupture was that of subacute tuberculous peritonitis. Rupture into the bowel is followed by diarrhea and disappearance of the tumor.

4. *Pain* like that of cholelithiasis may be present, but it more often radiates to the left side, shoulder and neck. Pressure symptoms include dyspnea, vomiting, icterus, emaciation and polyuria.

Differentiation from other cysts is largely a topographical question. Ovarian cysts have their pelvic origin and connection, but hydronephrosis is movable and originates in the flank to which pancreatic cysts seldom penetrate. Fluid in the lesser peritoneal sac or mesenteric, omental and retroperitoneal cysts are difficult to distinguish. Cysts of the liver present respiratory excursion.

Treatment.—Aspiration is never permissible. Enucleation of the sac is more dangerous than simple drainage. Bessel-Hagen's operative mortality was 6.7 per cent. Diabetes has followed operation.

TUMORS OF THE PANCREAS.

Cancer constitutes 67 per cent. of all pancreatic diseases but confusion with benign induration has been frequent until of late years. It occurs in 0.6 to 1 per cent. of autopsies. In 132 cases of pancreatic tumors 127 were cancer, 2 sarcoma, 2 cysts and 1 gumma (Segré). *Sarcoma* is usually secondary from contiguous lymphosarcoma in the retroperitoneal glands; 21 cases are reported.

Cancer is usually scirrhus, less often medullary, colloid or adenomatous. It most often begins in the head of the organ. In some cases it may be secondary to pyloric cancer.

Symptoms.—(1) *Tumor* is the commonest and surest symptom, occurring in 25 to 50 per cent.; it lies *deep*, beside the spine at the level of the navel and between the pylorus and colon. It is tender and *immobile;* small tumors are commonest; in some instances it may show propagated pulsation from the aorta or a bruit from its compression. Palpation should be made with the stomach and bowels empty. (2) *Icterus* occurs in 72 per cent., increases gradually and is more often *continuous* than remittent. Occasionally, just before death, the growing anemia seems to lessen the jaundice. Bard and Pic state the liver is usually rather small. (3) *The gall-bladder* is usually *enlarged* (perhaps shrunken when the hepatic or cystic duct is obstructed.) (4) *Pain* and *early vomiting* are usual.

The pain is epigastric, severe and sometimes neuralgic (neuritis celiaca) or radiating, as in pancreatitis or lithiasis. (5) *Cachexia* is marked and rapid; great prostration, emaciation and a syncopal tendency occur as in other pancreatic lesions. The temperature is subnormal. (6) The *stools* may contain much fat (Kuntzmann, 1827, and Bright, 1833); in *steatorrhea* the fatty acids and neutral fats are more abundant than the fatty soaps (Müller); its significance grows if diarrhea and icterus are not present. The stools also contain many undigested muscle fibers (*lientery*) and sometimes blood is present. (7) The *urine*. Mirallié encountered *glycosuria* in 26 per cent.; in Robson's 15 cases it was absent. It is said to be an early symptom and to disappear with the increasing icterus. *Albuminuria* is more frequent. The *indican is decreased*. Peptone, maltose and fat have been found. Polyuria and bronzing of the skin are rare findings. *Complications* include compression of the aorta or cava; retention cysts of the pancreas; and extension by contiguity to the portal vein, pylorus, bowel, peritoneum, ureter or lungs.

Differentiation.—1. Chronic *pancreatitis* with icterus and tumor may not be differentiated even by laparotomy (Riedel), though ascites indicates neoplasm and recovery means inflammation.

2. *Duodenal cancer* above the papilla causes rather the picture of pyloric stenosis; in cancer below the papilla pancreatic juice may be recovered from the stomach.

3. *Liver disease*, with the frequent icterus, ascites and splenic enlargement, causes little difficulty in differentiation.

4. Calculus in the Common Duct.——*vs.*——Compression of Common Duct by Pancreatic Tumor.	
1. History of colic usual.	Absent.
2. Tenderness of gall-bladder usual.	Absent.
3. Icterus, often remittent.	Permanent.
4. Liver less large than in—	pancreatic tumor.
5. Intermittent fever, and rigors.	Subnormal temperature.
6. Gall-bladder (see gall-stones) shrunken in 80 per cent.	Dilated in 92 per cent. (Courvoisier's law).
7. Slower course.	Very rapid emaciation.

According to Bard and Pic the icterus, enlarged gall-bladder, rapid emaciation, cachexia, subnormal temperature and normal or smaller liver of pancreatic cancer can only be confused with some few cases of gall-stones, cancer of immediately adjacent parts, primary cancer of the liver or bile-passages and primary duodenal or gastric cancer with early diffuse generalization in the liver. Kehr states that chronic obstruction of the common duct is cancerous in 70 per cent., pancreatic in 20 per cent. and calculous in 10 per cent.

Treatment is largely symptomatic; morphine should be given for pain, the gall-bladder should be drained (cholecystotomy) or it should be sutured to the intestine (cholecystduodenostomy). Very few permanent recoveries follow extirpation.

DISEASES OF THE PERITONEUM.

ACUTE DIFFUSE PERITONITIS.

Peritonitis was once far more important as an independent affection than at present; we now regard it as chiefly symptomatic of some other disease, *e. g.*, appendicitis, salpingitis, puerperal sepsis, etc.

Etiology.—The *actual cause* is *mycotic*. Some writers classify peritonitis as bacterial (suppurative forms), chemical (serous and serohemorrhagic forms) and mechanical (adhesive forms). The Streptococcus pyogenes (puerperal and traumatic forms) and Bacillus coli (forms due to intestinal lesions) are the two most frequent organisms; the staphylococcus, pneumococcus and tubercle bacillus are frequent and less often the Bacillus pyocyaneus, proteus, anthracis, typhosus, aërogenes encapsulatus, the ray fungus, gonococcus, Ameba coli, etc. Generally speaking, mono-infections are less common than *mixed infections*.

Factors *lessening physiological resistance* include nephritis (in 10 per cent.), alcoholism, cardiac disease, etc.; in other words peritonitis may be a terminal infection, fatal to reduced subjects.

Atrium.—Compared with pleurisy and pericarditis, peritonitis is seldom primary. Though it may follow inflammation of every abdominal viscus, it is most commonly a secondary manifestation of intestinal disease (*e. g.*, appendicitis) or of pelvic disease in women. (*a*) Infection through the *alimentary tract;* appendicitis, bowel ulceration (typhoid, carcinomatous, tuberculous), bowel obstruction, perforation, traumatism and infarction may cause it. Gastric are less frequent than intestinal lesions and ulcer and cancer rank first. (*b*) The *female genitalia;* it may result from gonorrhea (Nögerrath), extra-uterine pregnancy, puerperal infections and septic abortion; peritoneal infection from diseases of the male genitalia is rare. (*c*) *Liver and bile vessels;* local or general peritonitis may result; peritonitis is more common as a result of hepatic abscess and syphilis than of echinococcus, cancer, pylephlebitis or cholecystitis. (*d*) *Pancreas;* acute pancreatitis. (*e*) *Kidney* and bladder; pyonephrosis, calculi or ulcerations of the bladder. (*f*) Rarer causes are splenic affections, retroperitoneal adenopathies, spinal or costal caries, lesions of the thoracic duct or the abdominal parietes or extension through the diaphragm. (*g*) *Metastatic peritonitis* is not common, though observed in septicopyemia and acute infections. (*h*) In *children* fetal peritonitis has been observed by transplacental infection; in the newborn the umbilical vein may convey infection, though peritonitis occurs in but 8 per cent. of fatal cord infections. It occurs more commonly through the lymph vessels of the cord.

Age and Sex.—Most cases occur between the ages of fifteen and forty and women are more often affected than men.

Symptoms.—1. Local or Abdominal.—(*a*) *Pain,* the usual initial symptom, is almost invariable, is continuous, begins near the navel and remains greatest there; a causal gastric ulcer may produce pain in the back, salpingitis is attended with early pelvic pain and appendicitis

has its own initial localization, but the pain of diffuse peritonitis is felt at or below the navel. (*b*) *Tenderness* accompanies pain and is deep rather than superficial; they are very rarely absent and are inflammatory; at the operation or autopsy the visceral and parietal investments are reddened, lustreless, covered with a thin film of fibrinous exudate which binds the intestinal coils together; not infrequently the peritoneum is dotted with minute petechiæ. (*c*) The *attitude* is characteristic; the head is lifted and the knees are drawn up to relieve tension or to keep the bedclothes from contact with the abdomen. (*d*) *Vomiting* is reflex; it occurs early and greatly aggravates the pain; the vomitus contains food and later bile; it may be green (*vomitus herbaceus*), rarely dark brown or fecal. It is accompanied by redness and dryness of the tongue, great thirst and singultus which is a sensory phrenic symptom; before the fatal issue vomiting and singultus cease. (*e*) The *abdomen* is at first *tense, retracted* and *scaphoid,* but *later* becomes *tympanitic* from paresis of the inflamed intestinal coils (Stokes) and decomposition of their contents; the diaphragm stands high and its movement is restricted; in some, especially muscular subjects, the abdomen may be retracted throughout the course. Percussion may elicit *dulness,* usually due to serous, sero-purulent or perhaps serohemorrhagic exudation, which may be effused in small pockets between fresh adhesions or may fill the entire cavity, amounting to even 30 or 40 quarts and sometimes fluctuating to the hand; *as a rule* a *tympanitic note* prevails; in some cases dulness suggests the presence of fluid which, however, is not found on exploration; because of adhesions, the fluid changes little on change of posture. On auscultation a friction-rub is sometimes heard (Beatty and Bright), chiefly over the liver. (*f*) *Constipation* results from inflammatory infiltration of the intestine (Stokes); in the puerperal streptococcic and in pneumococcic peritonitides, diarrhea is common.

2. General Symptoms.—(*a*) *Shock* is a conspicuous symptom. Collapse may occur early and directly from perforation or later from toxemia. The *pulse* is *frequent, hard, wiry* and about 120; later it becomes thready and runs to 140 or 170. The *facies Hippocratica* is pronounced, with its anxious expression, wrinkled, clammy, cyanotic, lead-colored skin, pointed nose, hollowed temples and sunken eyes. The voice whispers (*vox cholerica*) to avoid movement of the diaphragm and the *breathing* is costal, *shallow,* difficult and rapid. (*b*) *Fever* is usually present. It may rise abruptly with a chill at the onset but follows no set type; it may be continuous or remittent and is usually found by rectum, even during collapse. The fever and pulse curves may run parallel in peritonitis with a slow course. (*c*) The *lungs and heart are crowded* upward by the abdominal distention. The apex-beat is high and further to the left as the heart lies more horizontally. (*d*) The *urine* is scant from lessened absorption and from cardiac weakness; albuminuria is frequent; *indicanuria* is constant from increased putrefaction in the paretic bowel; dysuria is usual; tenesmus is less common than retention.

Course.—The issue is fatal within two days in the *rapidly fatal toxemic form* in which there is only roughening of the peritoneum without exudation; the slower cases rarely last more than five days. Consciousness

is usually preserved to the end, though stupor or delirium occasionally intervenes; the pulse becomes thready, the breathing shallow and sighing, the superficial temperature is low and the patient succumbs to toxemic cardiac failure. In exceptional cases death occurs within a few hours or relative recovery results with encapsulation of pus. Treves found inhalation pneumonia in 17 per cent. of his cases and Tilger found serous pleurisy in 25 per cent.; of the latter, 63.3 per cent. were right-sided, 3.3 per cent. left-sided and 33.3 per cent. bilateral.

Types.—(*a*) *Peritonitis serosa* usually follows the fibrinous form; the fluid is yellow or light green, its specific gravity is 1.015 or more, it contains 2 or 3 per cent. of albumin and flocculi of fibrin, epithelial cells, red cells and leukocytes. (*b*) *P. purulenta* may occur in pockets or as a massive pyoperitoneum, amounting to 30 or 40 quarts. (*c*) *P. putrida* occurs in septicopyemia and puerperal fever, but especially in perforating carcinoma; fetid peritonitis may occur without perforation. (*d*) *P. perforativa* is characterized by two groups of symptoms, (i) those of peritonitis and (ii) those of gas in the peritoneal sac (pneumoperitoneum, pneumatosis peritonei); there is often the initial subjective sense of perforation, sudden pain, collapse and retracted abdomen; in four to twelve hours diffuse peritonitis sets in; free gas may obliterate the dulness of the liver and spleen, though this also results from great tympanites without perforation; the liver dulness may be found only in the posterior axilla, but when the patient is turned on his left side the free gas entirely obscures it; the abdominal contents may be putrid or fecal; when due to perforating gastric ulcer there is seldom any odor. The diaphragm is pushed to its highest possible level; a succusion splash may be elicited and the intestines may be compressed against the spine. Gas may develop through the action of the Bacillus pyogenes fetidus and B. aërogenes encapsulatus, but pneumoperitonitis without perforation is rare. (*e*) *P. puerperalis*, usually streptococcic, is described on page 22; it is characterized by moderate pain, enormous tympanites, diarrhea and the usual septic manifestations. (*f*) *P. pneumococcica;* Barling (1912) collected 234 cases; 73 per cent. occur in young girls. There are three types: (i) the very *acute form*, in which the lower abdomen is especially involved, often simulating appendicitis, with abrupt onset, severe pain, high fever, vomiting and early, continuous, fetid diarrhea; one-half of the cases die. (ii) With the above symptoms or perhaps pneumococcic involvement of the joints, ear, meninges, etc., there are signs of *pneumonia* and *peritonitis;* most patients die. (iii) The last type is more subacute, uniting symptoms of *sepsis* with those of pneumonia and peritonitis; nearly all of this form die. The mortality of all types is 80 per cent. The process is diffuse and fibrinopurulent; operation is indicated later. It often points at the navel, through which a creamy, greenish pus may rupture. Sometimes it ends by crisis; an encysted pus sac often remains. The pneumococcus may be found in the blood. (*g*) The *streptococcic*, *colon* and *staphylococcic peritonitides* are described under Appendicitis.

Diagnosis.—Peritonitis is usually diagnosticated with ease from the cause (appendicular, salpingitic, gastric ulcer, septic abortion), by the

febrile onset, pain, tenderness, vomiting, distention, effusion and collapse. Aspiration is dangerous. In certain cases only the fact of peritonitis can be established, perhaps even at operation.

Differentiation is necessary from (*a*) intestinal *obstruction* (*q. v.*); (*b*) biliary or renal colic; (*c*) acute *pancreatitis* (*q. v.*); (*d*) *ruptured tubal pregnancy,* in which the menstrual history, pelvic localization, acute anemia and shock are present; (*e*) *ruptured abdominal aneurysm;* or infarction of the superior mesenteric artery in which a cause for embolism, acute obstruction and bloody vomiting or diarrhea with later peritonitis are often distinctive; (*f*) acute *enterocolitis,* which is attended by less pain and tenderness and by more diarrheal colic, tenesmus and collapse; (*g*) *hysteria* (*q. v.*), which may simulate peritonitis, as it may resemble every other disease; if its presence is once suspected the other stigmata are usually definitive. (*h*) The *referred abdominal pain* of *thoracic disease* (pleurisy and pneumonia) is very seldom accompanied by tenderness, and is distinguished by examination of the chest, especially of its posterior parts. (*i*) Mild infections with much exudation may simulate *ascites* (*q. v.*).

Treatment.—*Surgical* treatment alone is efficacious. In older statistics the percentage of recoveries was small (Krogius, 680 cases, 28 per cent.). At present, 90 per cent. recover under drainage, semi-erect posture and continuous enemata of normal salt solution at low pressure.

Palliative treatment is of little value. (*a*) *Opium* controls pain; the doses recommended by Alonzo Clark (over 1000 grains in one week!) are now only of historical interest. Morphine, given hypodermically, produces the best results. (*b*) The patient should have *absolute rest.* (*c*) Tait's saline purgation to increase osmosis is productive of more harm than good. (*d*) *Vomiting* is treated as in acute gastritis; the stomach should be washed out; all medication, food and water by mouth should be withheld and given only by rectum. (*e*) Tympanites should be treated by the turpentine stupe and by enemata containing emulsum asafetidæ ℥iij, spts. chloroformi ℥j and ol. terebinthinæ ℥ss.

CHRONIC DIFFUSE PERITONITIS.

Aside from tuberculous peritonitis, chronic diffuse peritonitis is uncommon. (*a*) The serous or serohemorrhagic form will be considered under ascites; (*b*) the diffuse adhesive form results from acute and tuberculous peritonitis; the peritoneal sac is obliterated and at operation or necropsy the knife cuts directly into the intestine. (*c*) *Chronic proliferative peritonitis* may occur in peritoneal cancer or tuberculosis, chronic alcoholism, cirrhosis of the liver or cardiac disease. It may develop with or without adhesions, as a diffuse sclerosis of the peritoneum and as a subperitoneal proliferation. Its extreme form was described by Virchow (1885) as *peritonitis deformans.* The peritoneum is white, lustreless and diffusely thickened, though in some places more than in others. The omentum is retracted upward, so that it lies as a hard, transverse roll above the navel; the mesentery is indurated (mesenteritis retrahens), as described under Tuberculous Peritonitis and as it retracts it may

gather the intestinal coils into a ball the size of a child's head, lying centrally or to the right of the median line. Here and there may be found multilocular serous encapsulations or palpable tumor-like thickenings. The spleen, liver, stomach, cecum or colon may be particularly indurated. In some cases it is but part of a multiple serositis (polyserositis, polyorrhomenitis), invading also the pleura and pericardium. Miliary fibromata resembling miliary carcinosis or miliary tuberculosis, may sometimes form around parasitic ova or cholesterin crystals.

LOCALIZED PERITONITIS.

I. Suppurative Forms.—1. **Subphrenic Abscess and Pyopneumothorax Subphrenicus.**—First diagnosticated by Barlow (1845) and especially described by Leyden (1879), Piquand collected 890 cases (1909), upon which the following figures are based:

Etiology.—(*a*) *Gastric ulcer* and cancer accounted for 251 cases. (*b*) *Appendicitis* caused 191 cases; (*c*) *trauma;* (*d*) *intestinal ulceration* (duodenum 36 cases and the rest of the intestine 20 cases); (*e*) cholangitis and cholecystitis (66 cases); *liver disease* (echinococcus, abscess) 70 cases; (*f*) *splenic disease* (40 cases); (*g*) renal disease (28 cases); (*h*) *thoracic disease* (32 cases); (*i*) *pancreatic disease* (27 cases), tuberculosis (23 cases), female genitalia (17 cases), trauma (20 cases) and unknown causes (69 cases).

Symptoms.—Pus is ensacculated beneath the diaphragm (pyothorax subphrenicus), associated in over 25 per cent. with gas formation (pyopneumothorax subphrenicus). The *subphrenic abscess* is right-sided in 59 per cent., left-sided in 38 and bilateral in 3 per cent., is limited by the suspensory ligament of the liver and may be intra- or extraperitoneal; in the latter instance the abscess is most often appendicular or perinephric and lies well back in the abdomen. Simple pus formation is often difficult to localize; the symptoms are those of sepsis, with pain, tenderness or dulness on the liver convexity, which suggest liver abscess. The retroperitoneal forms give most dulness posteriorly, and intraperitoneal forms chiefly anteriorly; edema of the chest wall may occur without suppuration; the *x*-rays may produce a subphrenic shadow.

In *pyopneumothorax subphrenicus* there are signs "of a cavity beneath the diaphragm, filled with gas and pus, pushing the diaphragm upward and simulating pyopneumothorax" (Leyden). (*a*) *Tympany* and *absence of vesicular murmur* and of vocal *fremitus* are found from the third rib downward, above which is the compressed lung and below which is (*b*) the *dulness of the liver* whose exposed surface and edge, dislocated downward, are easily percussed and palpated. Sometimes the tympany entirely covers the liver dulness. As in pneumothorax (*c*) *succussion* and, on change of posture, the *clearly shifting line of dulness* may be elicited. (*d*) *Puncture* evacuates gas and putrid pus; sometimes on a higher puncture serum is found in the pleura, for pleurisy develops in 66 per cent. of the cases. The needle ascends during inspiration and descends during expiration, which is the converse of the findings in pleural

exudation (Fürbringer). If a manometer is attached to the exploring needle, a rise in the indicating column during inspiration and a fall during expiration denote a subphrenic lesion (Pfuhl). (*e*) *The x-rays* may show a shadow between the diaphragm and the liver and no respiratory excursion. (*f*) *Perforation* into the lungs occurs in 16 per cent. (more often in extra- than in intraperitoneal forms), into the pleura in 19 per cent. and into the pericardium in 1 per cent. At the beginning and until perforation there is generally an entire absence of thoracic symptoms, as cough or sputum; this is of value in differentiation from true pyopneumothorax. (*g*) The *history* of the subphrenic pyopneumothorax is usually that of some *abdominal lesion* (*v.* ETIOLOGY).

Treatment is surgical. Without operation the mortality is over 90 per cent.; with operation 30 per cent.

2. **Suppuration in the Lesser Peritoneum.**—This may result from gastric or duodenal ulceration, acute pancreatitis, etc. Closure of the foramen of Winslow confines the pus; a tumor appears in the epigastric, umbilical or left hypochondriac region, with relations like those of pancreatic cyst; variability in size occurs when the stomach is filled with gas and fluid. When gas and pus coexist the diaphragm and liver are pushed upward. Treatment is surgical.

3. **Other Forms.**—The *appendicular abscess* has been described. The *pelvic abscess* occurs especially from tubal, but also from uterine and ovarian inflammation; it follows abortion, puerperal fever, gonorrhea, tuberculous and pneumococcic peritonitis in young girls. Other types are suppuration around the gall-bladder, colon and sigmoid and in other rare localizations.

II. Adhesive or Indurative Forms.—Local thickening or adhesions may occur over any viscus, especially over the spleen, next the liver and less commonly the intestines, which may become strangulated.

Perisplenitis is most common over chronic malarial, leukemic and pseudoleukemic enlargements of the spleen.

Perihepatitis may cover small areas of the liver like soldier's spots in the pericardium or may engross its entire surface, as described by Budd (1852) and Curschmann (1884), who called it the "icing liver" (*Zuckergussleber*). It is frequently associated with deforming proliferative peritonitis. Nichols describes it as a hyaline degeneration, hyaloserositis. There are three groups of cases: (*a*) *multiple serositis* (polyorrhomenitis, Concato's disease); this borders closely on Pick's "pseudocirrhosis pericarditica." (*b*) *Arteriosclerosis* and *interstitial nephritis* occurred in 86 per cent. in Hale White's series. (*c*) Rarer factors are syphilis, tuberculosis, malignancy and alcoholism (page 616).

III. Chronic Hemorrhagic Peritonitis.—This rare disease was described first by Virchow, who likened it to pachymeningitis hemorrhagica. It occurs chiefly in the pelvis and is most often circumscribed. Pachyperitonitis is characterized by hemorrhagic inflammation; from the laminæ of the resulting fresh connective tissue, deposited layer by layer, repeated fresh hemorrhages arise.

CARCINOMA OF THE PERITONEUM.

Primary *endotheliomata* are very rare.

Cancer is most frequently secondary to cancer of the stomach, ovaries, intestines, pancreas and liver. Histologically it is that of the primary growth. Its gross forms are (*a*) *miliary carcinosis,* resembling miliary tubercles; (*b*) *vegetative;* (*c*) *ulcerative;* (*d*) *cystic* and (*e*) *infiltrative.* The primary tumor may be latent clinically, so that the peritoneal complication is apparently primary. The secondary growths spread by contact, the lymphatics and implantation of free particles. They may be felt in the omentum, mesentery and Douglas's *cul-de-sac.* The diagnosis is reached by the symptoms and signs of the initial growth or by those of an ascites, which is serous, hemorrhagic, pseudochylous or adipose; its progress is rapid and attended by cachexia. Groups of cancer cells may be found in the aspirated fluid and secondary cancer may develop at the point of puncture (page 616). In Hodenpyl's remarkable case, which recovered, the ascitic fluid exerted a curative action when injected into animals with cancer.

ASCITES.

Hydroperitoneum is a symptom of various diseases.

Etiology.—(*a*) *Stasis* explains most of the cases; the chief varieties are *cardiac* lesions (40 per cent.), pulmonary, mediastinal or pleural disease and *portal* stasis (10 per cent.), due to disease of the liver or portal vein. Stasis causes increased pressure in the portal system, followed by malnutrition of the vessels and pouring out of serum. (*b*) *Hydremia or cachexia* causes malnutrition and increased permeability of the vessel walls; it occurs in amyloidosis, chronic nephritis (10 per cent.); marantic conditions, profound anemias and occasionally in acute infections. (*c*) *Inflammation,* as simple, tuberculous (10 per cent.), suppurative or cancerous peritonitis (20 per cent.). (*d*) *Abdominal* tumors, leukemic spleen, etc. More than one factor may operate; tuberculous peritonitis may complicate liver cirrhosis or ascites may be due to the hydremia of nephritis, accentuated by weakness of the heart (*v. i.* Etiological Diagnosis).

Symptoms.—1. Inspection.—A *symmetrical enlargement* of the abdomen in the average case presents rather more lateral than anterior bulging. The abdomen resembles that of a batrachian (*ventre de batracien*). The skin is pale, tense, striated, edematous; the *pouting navel* and diastasis of the recti muscles are noted. A *caput Medusæ* may be noted in cirrhosis cases. Dilated veins over the ribs and upper abdomen indicate compression of the cava inferior. Sometimes the heart imparts systolic waves to the fluid.

2. Palpation.—Fluctuation (*ballottement*), due to the transmission of a fluid wave from side to side, is usually present but may be absent because of tense abdominal walls or great accumulation of fluid. Pseudo-

fluctuation may be caused by accumulation of fluid other than ascitic, as intestinal contents, or by lax, obese abdominal walls; in the latter condition a third hand placed in the median line of the abdomen will break the deceptive wave, due to parietal vibration. In women the *water-pillow* fluctuation of Landau may be elicited *by vaginal examination* and the uterus seems remarkably movable. *Thrusting palpation* may disclose the outline of the liver, spleen or possibly some neoplasm even through the abundant fluid (placing the fingers vertically on the abdomen and suddenly thrusting them inward, thus anticipating any muscular rigidity and pushing aside the ascitic fluid).

3. Percussion.—A pint to a quart of fluid must be present for clinical detection. With small effusions the patient should be examined in the genupectoral position. Elevation of the buttocks directs the scanty fluid to the flanks where it is more easily demonstrable. Classically, the *fluid* in dependent parts *gives dulness* and the *supernatant intestines* are located by *tympany* in the highest parts of the abdomen. These areas *vary with change of position.* This rule is void when the mesentery is retracted and does not allow the gut to float; adhesions between the intestinal loops prevent shifting of the fluid; when the gut is adherent there is permanent tympany in that location. Light percussion is employed, because heavy tapping elicits tympany from the more deeply situated intestine. The author withdrew two gallons of ascitic fluid from a patient in whom no dulness could be elicited in any position.

Diagnosis.—*Fecal accumulations* cause dulness in either flank, simulating fluid, but its immobility and the use of enemata preclude error. Küssner published a case in which fluid in the intestines was mistaken for ascites. Leube confused ascites with an enormously *dilated stomach* and John Hunter thrust a trocar into a dilated bladder. Hydronephrosis, hydatid cyst, pregnancy and tympanites are frequently sources of error. Rostan (1837) noted that tympany might be absent in ascites when the intestines contained little or no gas, a point sometimes forgotten in differentiation between ascites and *ovarian cyst;* the latter may thrust itself in between the coils of gut and simulate ascites; ovarian cyst usually produces a central flatness with tympany in the flanks, and neither the dulness nor tympany shifts with change of posture. Adami collected 42 cases of *fibrolipoma* and *myxolipoma* in which ascites was closely simulated.

Etiological Diagnosis.—1. Stasis.—In *cardiac disease* the dyspnea develops early; dyspnea caused by ascites pressing upward on the diaphragm is a late symptom. Swelling of the feet usually antedates the ascites, though pericarditis and valvular disease may produce ascites without anasarca. The *liver* is peculiarly an etiological factor in ascites (see page 587). Portal ascites commences in the peritoneum; the legs swell secondarily from pressure on the cava inferior or from cardiac weakness due to great ascites (see page 616).

2. Hydremia. (Cachexia, Increased Vascular Permeability.)—In renal disease the eyelids often become edematous before other symptoms develop and the nephritic ascites is seldom great unless the liver and heart are involved. Cachectic ascites is not uncommon in leukemia

and kindred affections and in acute infectious diseases in children; the author has seen three instances of ascites in typhoid convalescence.

3. Peritonitis.—The specific gravity and the percentage of albumin in the fluid are important factors in differentiating between exudate and transudate. Three divisions are distinguished: (*a*) A specific gravity of 1.010 or lower indicates a *cachectic transudate, e. g.*, in nephritis, with less than 1 per cent. albumin. A very low specific gravity and percentage of albumin occur in amyloidosis. (*b*) A *hypostatic transudate* has a specific gravity which ranges between 1.010 and 1.014 and has between 1 and 3 per cent. of albumin. (*c*) An *exudate* has a specific gravity of more than 1.015 (or 1.018) and the albumin reaches 4 to 6 per cent. The lower strata are heavier and the specific gravity should be measured several times during the paracentesis. Less than 1 per cent. of albumin excludes disease of the peritoneum or portal vein. Reuss's formula computes the albumin percentage from the specific gravity—the percentage of albumin = $\frac{3}{8}$ (specific gravity minus 1000) minus 2.8. Ascites due to a carcinoma in the liver substance has a low specific gravity and albumin is present to 1 or 2 per cent.; if carcinomatous peritonitis develops, the specific gravity and percentage of albumin increase. An ascites due to nephritis has a low specific gravity but the figures rise when stasis due to weak heart or intercurrent inflammation develops. In simple transudation (ascites and hydrothorax), the characters of an exudate may develop, due to the long-continued irritation of the serosa.

Ascitic fluid contains the constituents of the blood—albumin, globulin, urea, uric acid, sugar, etc. (*a*) Ascitic fluid due to stasis is clear yellow or yellowish-green, opalescent and alkaline and microscopically contains some lymph cells, erythrocytes and peritoneal endothelium. (*b*) Exudates show a microscopic picture varying with the cause; fibrin threads, pus, blood, adipose, chylous or chyliform fluid, intestinal contents, cancer cells, tubercle bacilli, diplococci or pyogenic organisms, may be found. In exudates are found the polymorphonuclear leukocytes and some erythrocytes; lymphocytes and red cells are detected in tuberculous exudates (see page 460); the fluid must be examined at once. Rieder and Dock describe cells in carcinomatous peritonitis in which asymmetrical karyokinetic figures appeared. Quincke states that red disks, lymph cells and peritoneal endothelium are found in every ascites.

Primavera found that a drop of glacial acetic acid will leave a white cloud as it falls to the bottom of a vessel containing an exudate, while its addition to transudates gives no reaction.

"Essential" ascites is usually tuberculous peritonitis.

4. Tumors.—Tumors, as uterine fibromata may produce hydroperitoneum. Very small uterine myofibromata may excite considerable peritoneal effusion. There is a direct relation between the malignancy of the tumor and the amount of the fluid found. Ascites is infrequent in uterine tumors but when observed is most common in adenomyoma (Freund). It is more often seen in papillomatous cysts, whose walls rupture by fatty degeneration and pour out their contents into the abdominal cavity; serum is secreted by the peritoneal surface because of the irritation, even though the ruptured cyst be very small (Quénu).

The fluid in cysts contains more solids (50 to 60 pro mille) than does the ascites of renal disease (25 pro mille, Méhu). Ascites is most often observed in solid ovarian tumors.

	"Zuckergussleber;" chronic perihepatitis.	Atrophic cirrhosis of liver.	Chronic tuberculous peritonitis.	Carcinoma of peritoneum.
Age.	Occurs about middle life or later.	Oftenest about middle life.	Between ages of 20 and 40.	Late in life.
Sex.	Both sexes equally liable.	More frequent in males.	In females.	More frequent in females.
Previous History.	Often a history of acute pericarditis or perihepatitis.	History of alcoholism, syphilis or digestive disturbances.	Often a chronic cough, diarrhea, or genital tuberculosis. Often with pleurisy (polyserositis).	In some cases a history of cancer of stomach, ovaries, etc.
Alcoholism.	No influence.	Frequent.	None.	None.
Syphilis.	No influence.	Occasionally.	None.	None.
Heredity.	No influence.	Unimportant.	May be familial.	Unimportant.
Incidence.	Acute becoming chronic or insidious from first.	Insidious.	Onset may be acute or insidious.	Insidious.
Chronicity.	Cases last for 2–20 years.	May last for years	Prolonged.	Rapid course.
Fever.	Generally absent except during exacerbation or some complication.	May be afebrile; when present is slight.	Usually slight; often absent.	Rarely absent; due to complication or cachexia.
Pain.	Indefinite and trifling.	Slight.	Often marked.	Variable; often marked.
Digestive Disturbance.	Slight or none.	Constant; dyspepsia, nausea, vomiting, gastric hemorrhage, melena.	Fairly common.	Often marked; alimentary origin or pressure.
Ascites.	Constant and extreme; nutrition long maintained: may be tapped over 800 times; may resemble peritonitis; albumin 3 per cent.; fibrin.	Constant. Low specific gravity. Low albumin.	Never extreme, may be absent, may be hemorrhagic. Often sacculated. Tubercle bacilli; lymphocytic formula. Higher specific gravity.	Moderate grade; may be hemorrhagic or pseudochylous.
Anasarca.	Constant but slight.	Slight.	None.	Frequent.
Jaundice.	Absent.	Occurs in 15 per cent. of cases.	Rare.	Common when liver is enlarged.
Liver.	Not cirrhotic; at first enlarged, then small; smooth.	Cirrhotic; at first enlarged, then small and warty. Hepatargia.	Seldom enlarged. Matting of omentum may simulate hepatic enlargement. May be nodes lower; doughy feel.	May be enlarged, with nodules.
Spleen.	Gradual enlargement.	Gradual enlargement; often marked.	No enlargement.	None.
Omentum.	Thickened and contracted.	Normal.	Often matted up. Mesenteritis retrahens.	Often matted up. Mesenteritis.

Treatment.—The treatment of ascites varies with its cause, for which reference should be made to valvular heart disease, atrophic cirrhosis, tuberculous peritonitis and nephritis.

Paracentesis in peritoneal carcinoma and chylous ascites (*v. i.*) hastens the fatal issue. Hale White asserts that cases of cirrhosis survive but few punctures, while cases of perihepatitis may be punctured more than 200 times. Lécanu reports a case of ascites in which 886 tappings were performed in fifteen years. In puncture, surgical antisepsis is most necessary, for reduced subjects are easily infected. The bladder should be emptied by catheterization. The trocar should be introduced in the *linea alba* to avoid injury to the bloodvessels. It should be pushed

carefully and is felt to penetrate the parietal peritoneum by the sense of something giving way. A dull-pointed probe should be sterilized, with which to push back the omentum if it should prolapse against the canula. The fluid should be evacuated slowly to obviate collapse which may result from dilatation of the abdominal veins, if they are suddenly relieved of the pressure of the effusion. A cat-o'-nine-tails bandage may be applied as the tapping progresses, to compress the abdominal contents.

Chylous and Adipose Ascites.—The peritoneal cavity, more frequently than any other serous sac, is the seat of those unusual exudates known as chylous, chyliform, lactiform or adipose ascites. The first authentic case is Poncy's (1699). The literature gives about 200 cases. Seven have come under the author's observation.

Chylous ascites properly designates an effusion of chyle. *Adipose ascites* contains a large percentage of fat, with no chylous admixture. Some authors use the terms "adipose," and "chyliform" interchangeably. Chyliform ascites is a chyle-like fluid in which lymph or chyle is mixed with exudate or transudate.

1. **Chylous Ascites.**—Straus's case is the clearest example, in which typical chyle was extravasated through two fistulæ. Ingested butter or olive oil is recognized in the fluid withdrawn by paracentesis. Chylous ascites contains sugar, which is said to be diagnostic when diabetes is excluded. Sugar when primarily present may disappear later. Its presence is very suggestive; it has been detected not more than two dozen times. Many clinicians discount the importance of the presence of sugar as a diagnostic test; Bock found sugar (0.04 to 0.07 per cent.) in all cases of hydrops, and Eichhorst in 10 out of 17 cases of pleural exudate. A small amount of fat (0.9 per cent.) indicates chyle. Chylous ascites is rich in solids, mineral salts and albumin. Albumin and fat occur in small punctiform granules, susceptible of chemical differentiation. The escape of chyle may occur through a visible rupture or by transudation through the altered walls of the chyle vessels; among the most frequent causes of lymph or chyle obstruction are compression of the thoracic duct or lymph system by glands, neoplasms, peritonitis, occlusion of left subclavian vein, lifting or coughing, filarial disease or occlusion of the thoracic duct. Rupture may occur in any part of the lymphatic system, in the thoracic duct, receptaculum, lacteal vessels, lymph glands and chylous cysts.

2. **Adipose or Chyliform Ascites.**—Adipose ascites is characterized by the absence of sugar and a higher percentage of fat. The opacity may be due to emulsionized albumin. Fat is found in most cases; its highest figure was in one of the author's cases, 6.5 per cent. The granules of albumin and fat are much coarser in adipose than in chylous ascites. Hydropic and fatty carcinoma cells may, if numerous, cause a creamy layer. Red blood disks may occur, also fibrin, casein, mucin, sodium alkali albuminate, peptone, lecithin, cholesterin, fibrinogen, etc. Among the numerous etiological conditions are tuberculosis of the peritoneum and glands; carcinoma of the glands, peritoneum and lymph vessels; chronic peritonitis; liver cirrhosis, heart disease and sarcoma of the omentum or mesentery.

Diagnosis.—A diagnosis has been made prior to puncture only in Morton's early case. The local signs and symptoms do not differ from those of serous ascites. Some cases diagnosed as the vulgar ascites, healing without treatment, are probably chylous hydrops. It is not easy to differentiate between chylous and adipose effusions, even at necropsy.

Prognosis.—About 90 per cent. of both types die. Continuous chylous fistulæ are always fatal. The immediate prognosis is seemingly more favorable in the adipose form.

Treatment.—Surgical interference is chiefly indicated in tuberculous peritonitis. Paracentesis should be avoided as much as possible for it is weakening and precipitates the fatal issue.

SECTION V.

DISEASES OF THE KIDNEYS.

ACUTE NEPHRITIS.

Etiology.—(*a*) Resulting from *infections*, the *scarlatinal* nephritis (*q. v.*) is the most frequent type and develops after desquamation. Acute nephritis occurs in diphtheria, at the height of the disease; it is found in a large percentage of cases of acute enterocolitis in children; it may result from malaria, variola, sepsis, ulcerative endocarditis, pneumonia, tuberculosis and syphilis, or occasionally in typhoid, typhus, measles, tonsillitis or epidemic meningitis; it is rare in German measles, varicella or purpura. Infections operate (i) more often by their toxins than (ii) by the causal microbe, though the typhoid bacillus, pneumococcus, streptococcus and others are found in the kidneys and urine; (iii) in some instances hemolysins apparently cause nephritis (nephritis hemoglobinurica). (*b*) *Toxic substances*, taken internally or applied to the skin, may induce acute nephritis, as turpentine, salicylic compounds, phenol, potassium chlorate, cantharides, etc. (*c*) Exposure to *cold and dampness* (with alcoholism) lessens the tissue resistance or acts as a hemolysin. (*d*) *Skin diseases*, burns, eczema and psoriasis, may impair the skin functions or produce hemolysins. (*e*) *Pregnancy* (*v. i.*), chronic Bright's disease, cardiac affections and the dyscrasiæ predispose to acute nephritis. (*f*) Acute nephritis may occur independently of any known cause, and sometimes epidemically.

Pathology.—*Gross changes* may not be apparent in mild cases, though in some the kidneys are swollen from marked interstitial exudation and are dark and heavy. In other cases the kidney is yellowish-white and perhaps mottled from small hemorrhages. The edematous capsule strips readily and the cut section drips blood. On section the cortex is swollen and granular, and its striations are blurred and pale in contrast with the dusky medulla; the glomeruli are frequently visible as red or later as yellow dots. Without the microscope, lesser degrees of acute nephritis may be confused with fatty or parenchymatous degeneration. *Microscopic changes* are usually diffuse. (*a*) *Glomerular* changes. Glomerulonephritis, best exemplified by the scarlatinal form (*q. v.*), is caused by toxins reaching the tufts through the blood current. The glomerular capillaries show infiltration with leukocytes, an albuminous or cellular exudate escapes into the capsule and the epithelium of Bowman's capsule degenerates, desquamates and later proliferates.

(*b*) *Tubular* changes. Cloudy swelling, hyaline and fatty degeneration occurring especially in the cortex may be difficult to differentiate from febrile and other degenerations, but interstitial exudation occurs in every case of nephritis. In severe or diphtheritic cases some cells necrose entirely and desquamate. The swelling around the convoluted tubules is one cause of the kidney enlargement. (*c*) *Vascular* and *interstitial* changes are essential to the pathological diagnosis and are more focal than diffuse. Serum exudes into the connective tissue, tufts and tubules and coagulates in them; leukocytes escape into the parenchyma and interstitium and in some cases red cells also (hemorrhagic nephritis). Councilman thinks that Unna's plasma cells are conveyed to the kidney from the spleen and bone-marrow.

Symptoms.—The *onset* may be *sudden*, especially following exposure. Again it develops *gradually* in the course or convalescence of a causal scarlatina, diphtheria, etc. Many cases may be unrecognized without frequent analyses of the urine or until uremia develops.

1. Urinary Findings.—(*a*) The *urine is decreased* to ten or twelve ounces or in severe cases is *suppressed;* this is due to glomerulitis, stoppage of the tubules by coagulated albumin and desquamated epithelium or swelling of the capillaries; the decrease usually runs parallel with the intensity of inflammation and hydrops. During convalescence the urine becomes abundant and clear. (*b*) Its *color* ranges from that of febrile urine to a *dark,* smoky tinge; it is seldom red or actually bloody; in the hemoglobinuric form it is chocolate-colored. (*c*) The specific gravity averages 1.025 to 1.030 and (*d*) the reaction is *acid.* (*e*) *Albuminuria* amounts to $\frac{1}{3}$ to 1 per cent. and 5 to 8 gm. daily. There is serum albumin, considerable globulin and if there are many cells, as in tubular nephritis, nucleo-albumin. The author has seen 6 fatal cases of acute diffuse nephritis, confirmed by autopsy, in which there was no albuminuria. (*f*) *Urea* is *decreased* even to $\frac{1}{6}$ its normal quantity, as are also the *chlorides* and phosphates; the uric acid remains normal and the xanthin (alloxur) bases may be increased. (*g*) The *sediment* in mild acute tubular nephritis consists of many *epithelial cells,* some red and *white corpuscles* and hyaline *casts* with crystals of oxalate of lime and uric acid and sometimes hemoglobin granules, which also may be found as free granules. In diffuse glomerular nephritis there are almost always *red cells,* leukocytes, epithelium, fatty, hyaline and granular casts and granules of fat. Senator maintains that the leukocytes from the kidney are mononuclear. Waxy and epithelial casts are found only in the severest types. Blood and casts may appear before and may also outlast the albuminuria. For the significance of cylindruria see page 625.

2. Anasarca.—This is often the first symptom to attract attention. (*a*) It occurs *about the eyes* early in the morning and later in the day in other parts, as the limbs and external genitalia. As the process advances it may become general. (*b*) It changes its location readily and with *anemia* gives a rather *characteristic facies.* (*c*) Anasarca occurs most commonly in cases due to *scarlatina* and *exposure,* less commonly in pregnancy, malaria, alcoholism and skin diseases and seldom in sepsis,

pneumonia, typhoid or diphtheria. (*d*) It is caused by *inflammation* of the *skin vessels,* which is identical and associated with the inflammation in the glomerular vessels. (*e*) Identical changes may occur in the *serous sacs* (pleura, pericardium or, far less often, peritoneum, meninges and joints), *mucous membranes* (glottis or intestinal tract) or viscera (pul-

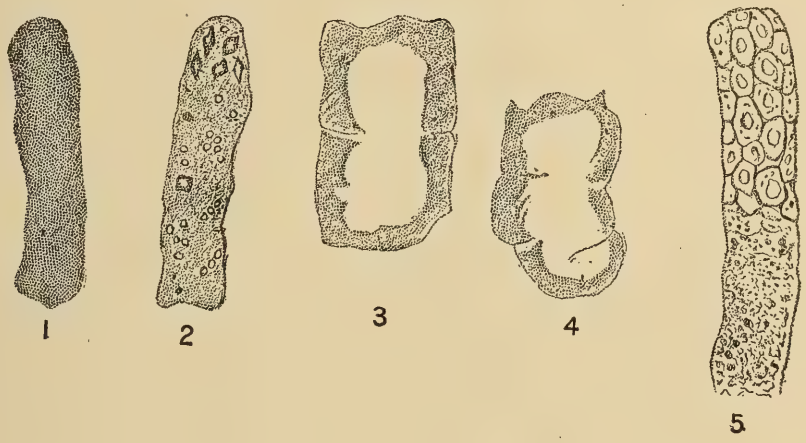


Fig. 46.—Casts. 1–4, amyloid; 5, cast, epithelial in its upper, and granular in its lower, part.

monary or cerebral edema). (*f*) It parallels the intensity of the nephritis and the decrease of urine.

3. Anemia.—The red cells and hemoglobin are reduced, often early and with the dropsy is suggestive of acute nephritis.

4. Cardiovascular Symptoms.—Hypertrophy of the left ventricle is uncommon, though observed within ten days of the onset. The pulse

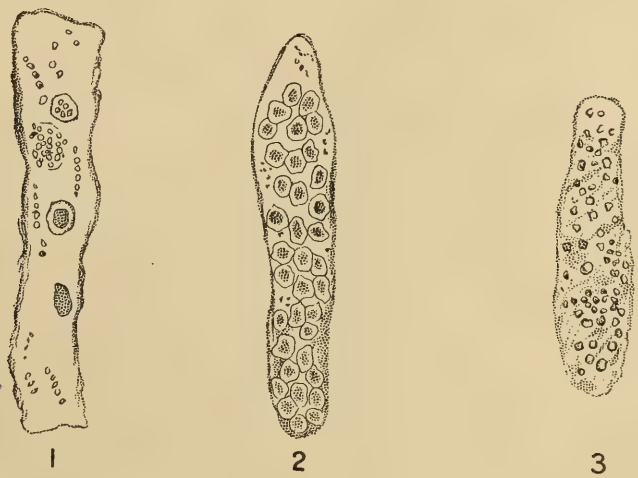


Fig. 47.—Urinary casts. 1, hyaline, with fat droplets and cells; 2, hyaline, with leukocytes; 3, cast with fat droplets.

is usually slow and tense; the sphygmogram shows increase of the tidal and decrease of the dicrotic wave and the sphygmomanometer shows the blood tension increased even to 180 or 200 mm.; these phenomena are probably due to vascular contraction induced by metabolic products retained in the blood or by glomerulitis. The second aortic tone may

become accentuated and, if heart fatigue develops, the gallop-rhythm is heard.

5. Temperature.—Fever is exceptional. There may be pain and tenderness in the back, frequent and painful urination, emaciation and epistaxis. Constipation is the rule. The skin is generally dry.

Diagnosis.—The urinary findings are characteristic; *albuminuria* (*q. v.*) may result from other causes, as fever, amyloidosis or stasis; casts, leukocytes, epithelia and even red cells also occur in chronic nephritis, but the history and evolution determine the diagnosis (*v.* page 640); cases without urinary findings are extremely rare. The greatest danger lies in confusing acute nephritis with an *acute exacerbation of a chronic process;* fully half of the author's acute cases belonged to this category. In *renal hematuria* the urine contains no casts (save perhaps red cell casts), few leukocytes and practically no epithelia; there is no anasarca; the urine is clear at one time and is laden with blood at another. *Anasarca* (*v. s.*) occurs in certain types only and also in various cachectic states.

Diagnosis of Type.—(*a*) The *kidney of pregnancy* usually develops in the last half of gestation and infrequently before the third month. Its pathogenesis is disputed, though toxemia due to the double task imposed upon the maternal kidneys seems the most probable cause. It has been suggested that anaphylaxis or decreased antitoxic activity of the thyroid gland is a causal factor or that the placenta elaborates toxins (syncytiolysins). The urine is decreased, pale and of low specific gravity; it contains much albumin and not much sediment, though red and white cells, lymphocytes, fatty epithelia and sometimes casts are found. The attendant anasarca, like that of stasis, develops from below upward. When retinitis develops its prognosis is more favorable than in other nephritides. The convulsive seizures (eclampsia) are often uremic, though Ingersley collected 106 cases in which there was neither clinical albuminuria nor renal alteration at necropsy; Fehling remarks on the slight anatomical changes in the kidney and on the fact that but 5 per cent. of pregnant patients having an old nephritis develop eclampsia. The embolism of placental cells in the brain or lungs (Schmorl) is probably rather a result than a cause of eclampsia. Zweifel maintains that the oxidation of the albumins is decreased, the nitrogen output decreased, the ammonia increased and that acidosis occurs. The maternal mortality is 30 per cent. and that of the child over 50 per cent. Very few cases develop chronic nephritis. (*b*) The *cholera nephritis* is parenchymatous and chiefly tubular and by some is considered renal ischemia rather than nephritis. (*c*) In *hemoglobinuric nephritis* hemolysins disorganize the blood and irritate the kidneys by the hemoglobin set free. It is found in burns, acute infections (typhoid, pernicious malaria, scarlatina, yellow fever, Winckel's disease of the newborn), exposure, hemoglobinemia and drug poisoning, notably that of chlorate of potash. (*d*) *Acute recurrent* hemorrhagic nephritis is uncommon.

Prognosis.—In general there are more recoveries than deaths. The following factors are important: (*a*) Recovery is usual in atypical or *light forms* not involving the glomeruli. (*b*) The *causal affection;* the

scarlatinal type has a mortality of 33 per cent.; the virulence of the epidemic is an important factor, as are also heart complications. (*c*) *Complications,* as (i) intercurrent *inflammations,* such as pneumonia, pleuritis, pericarditis or erysipelas; (ii) *edema* of the pharynx, larynx, lungs or serous sacs; and (iii) *uremia,* which will be fully covered under chronic interstitial nephritis; uremia is more often an adult than an infantile and a late rather than an early complication; headache, delirium, dyspnea, convulsions, coma and vomiting are among its salient signs.

The average duration is less than two weeks, though one or two years may elapse before recovery, and gradual transition into chronic nephritis sometimes results.

Treatment.—Most of the points mentioned will be considered more fully in the treatment of chronic nephritis. (*a*) *Prophylaxis* is of limited value. Drugs which irritate the kidneys should be withheld in acute infections or pregnancy. In fevers water should be given freely by mouth or by enema; hydrotherapy is prophylactic, save in scarlatina when cold baths are contra-indicated. Induction of labor is indicated in severe cases. (*b*) *Diet.* Milk is diuretic, non-irritant and contains little or no extractives or salt. It should be given as in typhoid (*q. v.*). Thoroughly cooked carbohydrates, as gruels, are well combined with it. Alcohol, broths, meats and beef-tea are contra-indicated. (*c*) *Absolute rest in bed* is imperative to protect the skin and spare the kidneys from the products of the tissue waste; the patient should lie between flannel blankets and should be dressed in flannel nightgowns. (*d*) *No drug* modifies the nephritis; tannin and ergotin (of each 5 grains three times daily) seem to restrict renal hemorrhage. Salt should be withheld (see p. 635). (*e*) The best *diuretics* are *water and milk,* which flush out detritus from the kidneys; excessive quantities are often given; potassium citrate is given in doses of half a dram with sweetened, weak lemonade. Sweet spirits of nitre ℥ss–j, q. i. d., is diuretic and also dilates the bloodvessels. All stronger diuretics are to be avoided. (*f*) *Skin. Warm baths* are diuretic and stimulate some vicarious elimination through the skin. Careful covering and rest in bed relax the skin, materially aid its functions and relieve renal hyperemia. (*g*) *Saline catharsis* lowers arterial tension and aids compensatory elimination. As salts often derange the stomach, an ounce should be given by rectum with two ounces of glycerin and one of water. One-half to one dram of compound jalap powder or $\frac{1}{10}$ grain of elaterin is indicated when uremia impends. (*h*) *Treatment of uremia.* For suppression of urine a full warm bath lasting fifteen to twenty minutes should be given; a hot pack should be applied, followed by wrapping in blankets and covering heavily with a rubber sheet; or the alcohol sweat or hot-air sweat may be given. Cups applied to the back may divert blood away from the kidneys, because the vessels of the loin anastomose with those of the kidney. Pilocarpin, catharsis, incision of the legs, venesection, saline infusions, stimulation, etc., are considered fully under Chronic Nephritis. (*i*) *In convalescence,* iron and other mild tonics should be given, irritating foods and beverages interdicted, exercise carefully restricted and cold and exposure avoided by sending the patient, if possible, to a warm climate.

CHRONIC NEPHRITIS.

Aetius, Avicenna, Cotugno (1770) and other writers mentioned the association of hydrops and albuminuria, but to Richard Bright (1827) is due the credit of correlating renal disease, albuminuria and dropsy; he also noted the etiological importance of alcohol and cold, as well as the cardiac hypertrophy, uremia, brain hemorrhage, coma, convulsions, blindness and the tendency to serositis.

The following classification of nephritis covers typical cases, though *combined* or *mixed types* are equally frequent. As a cause of death chronic nephritis ranks sixth, after pneumonia, tuberculosis, heart disease, endocarditis and unknown causes.

I. Chronic Parenchymatous Nephritis.—Definition.—Chronic Bright's disease, with special involvement of the parenchyma, much albuminuria and abundant formed elements in the urinary sediment. The affection constitutes 3 per cent. of all diseases.

Etiology.—It may result from (*a*) *infections,* as tuberculosis (which causes 25 per cent.), malaria and syphilis; (*b*) from exposure to *cold and dampness;* (*c*) from toxic factors, as alcohol, lead, less frequently mercury or arsenic; (*d*) from *cardiac disease,* which accounts for 14 per cent. of nephritis; (*e*) exhausting *discharges,* suppuration and ulceration; (*f*) *acute nephritis* is a somewhat predisposing factor. (*g*) Most cases occur between twenty and fifty years of age; it is rare in children. Males are affected more often than females.

Pathology.—There are *three types* besides the mixed forms.

1. The "Large White Kidney," or "Inflamed Fatty Kidney."—This is never decreased in size and may weigh 10 to 12 ounces and measure two or three times the normal size.

Gross Characters.—The capsule is non-adherent; the surface is smooth, yellowish-gray, with prominent stellate veins and sometimes dots of hemorrhage. On section the cortex is broader, the striations are poorly marked and when cut streaks of fat may show; the glomeruli and convoluted tubules are yellow and contrast with the gray bands of newly formed connective tissue. The medulla shows fewer changes and is red, in sharp contrast with the light cortex.

Microscopic Characters.—The most essential changes are hyaline and fatty degeneration and atrophy, chiefly in the convoluted tubules, in which lie desquamated epithelia and coagulated albumin. The glomeruli are always diseased, but to a varying degree; they are enlarged, their vessels show hyaline and fatty degeneration and nuclear multiplication; albumin is found in their capsules and the epithelium is swollen, fatty or even necrotic. The connective tissue is always increased, which histologically separates chronic nephritis from acute nephritis and mere degeneration.

2. The "Large Red" or "Variegated Kidney."—*Gross Characters.*—It is also large, but is firmer than the kidney just described or that of acute inflammation, for it has more connective tissue, which may cause some tearing and loss of the cortex when the capsule is stripped off. The surface may be perfectly smooth. The cortex is swollen, indistinctly

striated, dotted yellow from fatty glomerular change or red from minute ecchymoses and sometimes streaked yellow and red from similar tubular changes.

Minute Characters.—These are essentially those of the large white kidney, except that fatty degeneration is less marked and connective tissue alteration and hemorrhages are more conspicuous.

3. The Secondary Contracted Kidney.—This form may develop after a year or two from the above forms.

Gross Characters.—It may be as large as the "large white" or "large red kidney" and the name refers rather to its connective tissue than to any visible renal shrinkage. The capsule is adherent in places and, when stripped, tears off small areas of the renal substance. The surface is rough and shows in some places reddish, depressed areas (fibrous contraction or granulations and parenchymatous atrophy) and in others yellow areas (which are more normal, though fatty). Section shows decrease in the cortex corresponding to the reddish foci. It differs from the genuine contracted kidney (primary interstitial nephritis) in the following respects: it is secondary, much more rapid in development, contains fewer cysts, is more yellow and its red granulations and yellow prominences are larger.

Minute Characters.—Epithelial changes resemble those of the two forms described. The membrana propria of the tubules thickens, adheres and obliterates many tubules. The glomeruli collapse, thicken and shrink to small, dark gray granular bodies, which sometimes calcify. The retracted areas, capsular adhesions and cortex shrinkage correspond to connective-tissue contraction.

Symptoms.—The *onset* is in most cases *gradual* and *insidious,* the earliest symptoms being loss of strength, pallor, emaciation, simple decline of health, indigestion, anorexia, headache, shortness of breath on exertion or chronic bronchitis.

1. Urinary Findings.—Except in the secondary contracted kidney (*a*) the *urine is decreased* to 1000–200 c.c.; (*b*) the color is *dark* yellowish-red, opaque or rarely smoky; the urates are held in suspension by the albumin; sometimes its surface is shimmering from fatty cells and casts; (*c*) the specific gravity is 1.020 to 1.040 and (*d*) the reaction is acid; (*e*) *albumin* is *more abundant in the day time* than at night and amounts to 1 to 3 per cent. and 15 to 30 gm. daily; it coats the bubbles formed by agitation of the specimen and preserves them for a long time. (*f*) The absolute amount of *solids* is generally *decreased;* for example, the urea (which normally constitutes 90 per cent. of the nitrogen output and amounts to about 35 gms. daily), kreatin and sodium chloride are decreased and the freezing-point of the urine is lowered (*v. i.*). (*g*) The *sediment* is *abundant* and hyaline, granular, fatty and epithelial *casts abound* (first noted by Simon and Nasse); coarsely granular and waxy casts appear late in the disease; the granular type is due chiefly to fat particles, which may darken the casts. They have been long regarded as significant of nephritis. Key, Nothnagel, Rosenstein and others afterward observed their occurrence in the urine of normal individuals and of late there has been a tendency to underestimate their impor-

tance. According to Senator, casts originate in the kidney chiefly from epithelial cells and always indicate some renal disease. They may be dissolved in the bladder by the pepsin contained in the urine. Leukocytes and fatty epithelia and, in some forms of nephritis, red cells occur in considerable amounts. In Ackermann's case no casts were found in the urine, but were found in masses in the renal pelvis at necropsy.

In the secondary contracted kidney the urine amounts to 1500 to 2000 c.c., is clearer, is somewhat lower in specific gravity and contains somewhat less albumin and casts.

2. Edema.—Edema is very frequent. Its absence argues for little glomerular involvement. It commences *about the eyes, changes its location* readily and is at first evanescent, but soon *becomes general, obstinate* and *ominous*. It is more pronounced in the "large white kidney" than in other types and increases as the urine decreases. The puffy and prominent eyes, pale, swollen cheeks, dull expression and distended abdomen and shapeless wrists and ankles often suggest the diagnosis. The *serous* and *mucous membranes* are similarly involved, though to a lesser degree, and also as possible terminal events, pulmonary or cerebral edema may develop. Edema is caused (*a*) by hypalbuminosis and hydremia of the blood (Bright's theory), especially in hemorrhagic types, (*b*) by abnormal permeability of the vessel walls, a theory advanced by Cohnheim and Lichtheim, or (*c*) glomerular changes, causing chloride and water retention.

3. Anemia.—Anemia distinguishes chronic parenchymatous nephritis from the genuine interstitial type. There is usually retention of urea, uric acid and chlorides; sometimes the blood is cloudy.

4. Cardiovascular Findings.—The pulse is more often small, soft, rapid and associated with a weak apex, indicating dilatation, than tense and associated with a loud second aortic tone and strong apex, indicating hypertrophy. Secondary contraction and mixed nephritis are likely to develop findings similar to those of the genuine interstitial nephritis; Senator finds eccentric hypertrophy in parenchymatous and simple or concentric hypertrophy in interstitial nephritis. Myocardial degeneration is common.

Complications and Causes of Death.—(*a*) *Marasmus* results from the anemia, indigestion, diarrhea, edema of the alimentary mucosa or its irritation by decomposed urea. (*b*) Intercurrent *infections* may result, as inflammation of the pleuræ or pericardium, cellulitis and especially pneumonia; many pneumonia deaths really result from nephritis; in one instance the writer saw acute necrosis and sloughing of the scrotum. Other complications are (*c*) *edema* of the lungs, larynx, serous sacs and extremities, impeding respiration and obstructing the peripheral arterial flow; (*d*) *hemorrhages* (8 per cent.) in the brain or other organs; (*e*) *retinitis* (18 per cent.); (*f*) *uremia*, with its numerous nervous, circulatory and digestive symptoms (*v.* page 630); (*g*) *acute exacerbations*, particularly in the chronic hemorrhagic type; and (*h*) *cardiac insufficiency*.

Diagnosis.—The diagnosis rests upon three cardinal symptoms: (*a*) the *urinary*, which are characteristic when twenty-four-hour specimens are carefully and repeatedly examined in every patient; (*b*) the *anasarca*,

which is sometimes absent in secondary contracted kidney and in Wagner's hemorrhagic type; and (*c*) the *anemia.* (See pages 640 and 641.)

Prognosis.—Though some cases suggesting an arrest of the process are found at autopsy, the clinical course is usually progressive and fatal. Dropsy usually causes death in a few months to a year, though the author saw 5 typical cases recover after two and a half years; they did not result from acute infections like the recoveries reported by Senator and Rosenstein. The outlook is better in children than in adults. The large, white kidney causes death in one-half to one year, the large, variegated kidney and the secondary, contracted kidney in one and a half to three years. The treatment is considered on page 633.

II. Chronic Interstitial Nephritis.—Definition.—A primary, interstitial, contracting sclerosis of the kidney, characterized clinically by abundant urine of low specific gravity, with small amounts of albumin and few cellular elements; and by marked cardiac hypertrophy, arteriosclerosis, hypertension, retinitis and uremia.

This genuine contracted kidney must not be confused with secondary contracted kidney, arteriosclerotic contraction, embolic contraction or the contraction resulting from ascending infection.

Frequency.—In ten years Eichhorst treated 31,562 cases, of which 1.4 per cent. had contracted kidneys (six and a half times as frequent as the parenchymatous form).

Etiology.—The etiology is that of *arteriosclerosis,* with which contracted kidney has a threefold relation: arteriosclerosis may cause the arteriosclerotic kidney; contracted kidney may cause arteriosclerosis; or both may result from a common cause. The often unknown toxin reaches the kidney through the blood stream.

The disease may result from (*a*) chronic *alcoholism* combined with exposure or overeating; (*b*) chronic *lead poisoning;* (*c*) *gout,* caused by the alloxur bodies; (*d*) *syphilis;* (*e*) *diabetes,* caused by sugar or acidosis; the diabetes lessens or ceases when induration develops; (*f*) *age;* it begins between the years of *thirty and fifty* and is clinically manifest after fifty years of age. Sawyer collected 24 cases in children. (*g*) *Sex.* The disease is three times as frequent in *men* as in women. In the infantile form most cases occur in girls. (*h*) Infrequent causes are acute nephritis, the kidney of pregnancy, hereditary tendency, as in Dickinson's series through four generations, and endocarditis.

Pathology.—It is undecided whether the process is inflammatory or atrophic; probably induration follows cellular degeneration.

Gross Pathology.—(*a*) The kidneys are *contracted* even to one-third their original size and may weigh but 21 gm.; contraction is rarely symmetrical in both. (*b*) Their *consistence* is *increased.* (*c*) The *capsule* is *thick,* tendinous and *adherent* and strips off particles of cortex when removed. (*d*) The *surface is uneven;* the small reddish or sometimes paler prominences, measuring 0.5 to 5 mm. represent the more normal tissue; the paler retracted areas are fibrous. (*e*) Small *cysts* are frequently found, usually with clear contents; their size ranges from that of a pin-point to a cherry; they result from glomerular and tubular constriction by connective tissue (retention cysts). (*f*) On section the *cortex*

is shrunken, even to 1 mm., irregular and gray from fibrillar connective tissue. The medulla is less changed, though also shrunken; pale fibrous lines alternate with red, dilated vessels; uric acid or calcium salts are deposited.

Minute Pathology.—The changes are largely cortical, focal, asymmetrical and interstitial. (*a*) The *glomeruli* early exhibit round cells, nuclear increase, desquamated cells and albumin; later they *atrophy* from degeneration of the afferent artery; in the extreme stage the glomeruli become *hyaline and fibrous;* some tufts may visibly hypertrophy, Sabourin's compensatory adenomata. (*b*) The *epithelium* is always changed, though much less than in parenchymatous forms; in the sunken areas many tubules disappear and degenerated cells are seen in the prominent surface granulations; in the medulla the collecting tubules are larger, even cystic, and are filled with colloid material. (*c*) The *connective tissue* is increased everywhere, but more in the cortex than in the medulla; round-cell infiltration occurs early and induration late; the vessels are sclerotic in all their coats, but especially in the intima, which some consider is the earliest and causative alteration.

Symptoms.—*Long latency* for years, *insidious onset* and *protracted compensation* by *heart hypertrophy* are characteristic of this type. Many cases are first discovered during an acute infection, as pneumonia, on examination for life insurance or at the coroner's autopsy after accidents. It may appear for years, to be a physiological or cyclic albuminuria. Polyuria, cardiac symptoms, retinitis, indigestion or depreciation of health may be the first symptom. The edema, pronounced urinary findings in a single specimen and anemia of chronic parenchymatous nephritis are seldom conspicuous and almost never early symptoms in this type. The condition may be first declared by heart incompetence, uremic coma or convulsions.

1. Urinary Findings.—Urinary findings are generally the earliest signs. Twenty-four-hour specimens should be examined repeatedly in doubtful cases, with a fresh specimen for microscopic examination. As a general statement, the urine is increased, pale and voided frequently; it is low in specific gravity and contains small amounts of albumin and solids; and the scanty sediment contains few casts. In detail: (*a*) The amount is gradually increased (polyuria) as the kidneys lose their power to condense the urine; this is a frequent cause for consulting the physician. Nagel found polyuria in only 47 per cent. The urine is also passed frequently (*pollakiuria*), especially at night, always suspicious when the prostate is normal. In well-developed cases it totals 2000 to 3000 c.c., rarely 8000 to 12,000. (*b*) It is *pale* and weakly acid. (*c*) The specific gravity averages 1.010 (1.005 to 1.012). (*d*) *Albumin* is present *in most cases,* but *is not abundant;* the merest traces are found up to a daily total of 2 to 5, rarely 10 gm. It is often absent in single specimens, especially in the morning, whence a twenty-four-hour specimen should always be examined. Delicate tests are often necessary (*v.* Albuminuria). It may appear only after eating, exercise or fatigue, whence its greater frequency toward night; it may increase after dyspepsia or bronchitis. Senator's statement that its alleged absence is due to an

arteriosclerotic kidney and not to interstitial nephritis is not invariably correct. Albuminuria may be absent for weeks or months. A *permanent foam* due to the albumin is frequent. (*e*) *Solids are decreased, i. e.,* the urea, uric acid, ammonium, chlorides and phosphates. The determination of the freezing-point, *cryoscopy,* employed by Korányi to estimate the amount of soluble molecules in the blood and urine, is not of practical value in medical cases. (*f*) Tests of *renal function* show decreased activity (*v. i.* Therapy.) (*g*) *The sediment is scant,* absent or obtained only by the centrifuge. Hyaline, granular and epithelial casts are found in small numbers when the light is moderated by the diaphragm, sometimes only after repeated search; the granular casts are often wide, originating in the dilated tubules of the medulla. A few epithelia, leukocytes and crystals of uric acid and oxalate of lime are found. Red cells are infrequent.

The urinary findings are modified by intercurrent uremia, fever and heart failure, all of which concentrate the urine.

2. Cardiovascular Symptoms.—Cardiovascular symptoms were noted by Bright, but their significance was first established by Traube. They appear in 75 per cent. of cases. Briefly stated, they are the signs of cardiac hypertrophy and arterial hypertension—a heaving apex, a metallic, second aortic tone, hard vessels and raised blood-pressure. *Cardiac hypertrophy* is more quickly detected clinically than pathologically, especially in the young, because the urine is increased before the signs of hypertrophy prevail and concentric hypertrophy is present long before dilatation appears. Emphysema may obscure the precordial evidences of hypertrophy, such as the *heaving apex-beat* and double impulse at the apex, and one must therefore depend on the evidences presented by the *tense pulse,* the hardness of the arteries, the sphygmographic tracings with a square apex, the loud, ringing, second aortic tone or other manifestations of increased arterial tension, as epistaxis, vertigo, headache, tinnitus, palpitation or cardiac asthma. The systolic blood-pressure in interstitial nephritis may exceed 200 mm., and is one of the most constant and early symptoms. (See pages 395 and 396.) The cause of the hypertrophy or hypertension is not definitely known—mechanical obstruction to the blood flow by renal contraction (glomerular changes) and vasomotor constriction excited by retained waste substances are probable causes. Though hypertrophy occurs in interstitial rather than in parenchymatous nephritis, the two types often mingle, with the urinary findings of the "large white kidney" and the cardiovascular findings of a renal contraction. In the last stage of contracted kidney the hypertrophied heart weakens, the hypertension falls, the apex becomes weaker, a systolic murmur appears from myocardial insufficiency and Potain's gallop-rhythm, a sign of heart fatigue, is heard.

3. Retinitis.—Retinitis albuminurica may be the first declaration of the disease and thus come under the observation of the ophthalmologist. It occurs more frequently than in other renal lesions, 25 per cent. Its forms are stellate, white areas of fatty and fibrous tissue, a white zone surrounding the disk, degeneration of the disk or retina, "flame-like" hemorrhages and choked disk whose large veins are compressed where

the arteries cross them. Identical findings occur in other conditions. (See Plate VI, Fig. 8.)

4. Uremia.—Uremia is the most dreaded and frequent complication; its nature and immediate cause are unknown; it can only be stated that it is an auto-intoxication, due to disturbance of renal function, probably with retention within the blood of nitrogenous substances. Further explanations are theoretical, such as acidosis; failure of the "internal secretion" of the kidney; the toxic action of abnormal products of metabolism or breaking up of the proteids by nephrolysins; it is not due to retention of urea alone or to mechanical conditions alone, as edema or anemia of the brain. It occurs most often in chronic interstitial, next in acute, then in chronic parenchymatous nephritis and is far less common in other diseases of the urinary tract, as pyelonephritis, etc.

Uremic *symptoms* may be acute, perhaps developing precipitately; more frequently it is chronic; prodromal headache is followed by chronic gastric irritability, vomiting and epigastric oppression; dyspnea and high arterial tension, alternate with restlessness, anxiety, insomnia or disturbed vision; and finally psychic disturbance, polyvisceral scleroses, myocardial incompetency, gallop-rhythm, Cheyne-Stokes's breathing or angina pectoris terminates—rapidly or with miserable remissions—in uremic convulsions, uremic coma or apoplexy. These manifestations will be considered as nervous, digestive, respiratory and cardiac:

Nervous Symptoms.—(*a*) Acute *convulsions* (eclampsia) are due to irritation of the cortex by toxins, and closely resemble the epileptic attack. There are usually prodromes, as pain or pressure in the head, neuralgia, insomnia, vertigo, visual disturbance, tinnitus, nausea, vomiting or dyspnea; there is usually no "epileptic cry." Then clonic or tonic convulsions appear, usually generalized and involving especially the flexor or extensor muscles of the arms and legs; the abdomen is retracted; there are dyspnea, cyanosis and involuntary evacuations; the reflexes, especially the patellars, are increased, and even ankle-clonus and Babinski's sign may develop, sometimes unilaterally and thus simulating apoplexy; the pupils are wide and reactionless; the pulse is full and slow before the attack but becomes rapid during the convulsions. The skin is covered with sweat and the temperature is usually elevated. The convulsions cease after fifteen to thirty minutes and coma intervenes, during which the convulsions may recur. Death is the usual outcome, but recovery is possible, as in one of the author's cases, in which the anterior third of the tongue was bitten off. (*b*) *Other motor manifestations.* Landois's general statement is as follows: *Paralysis* affects especially the sensorium, to a lesser degree the special senses and rarely motility; *irritation* seldom affects the sensorium (*e. g.*, delirium), it acts principally upon the motor paths (convulsions) and seldom upon the special senses. Uremic convulsions may resemble focal symptoms, such as convulsions of only half the body or rarely as convergent strabismus, facial spasm, Jacksonian epilepsy, nystagmus, localized trembling and twitchings, muscular cramps, grinding of the teeth, tetanic convulsions, trismus, convulsions on one side and contractures on the other, rigidity of the neck or opisthotonos. In 7 cases seen by the author the symptoms

were those of an acute focal brain lesion; in 2, treated by lumbar puncture, relief was immediate.

Paralyses in uremia are infrequent, are chiefly transitory hemiplegias and are due to *cerebral edema*, capillary hemorrhage, softening, apoplexy or cortical overstimulation by toxins. Hemiplegia may occur with aphasia, amnesia, deafness, crossed oculomotor paralysis, glossoplegia or conjugate deviation of the eyes. The author reported 5 cases in which uremia very closely resembled meningitis. Other cases have been suggestive of brain tumor. (*c*) *Acute coma* often occurs without antecedent eclampsia, being preceded by headache, stupor or apathy; coma may last for days or even months. (*d*) *Psychic symptoms*. The most common is a *characteristic restlessness and anxiety*. Obstinate *headache*, of a dull, throbbing nature is common; it often begins in the early morning; it may be occipital or hemicranic. *Insomnia* is common, though during the day the patient is often sleepy. Delirium and mania are more frequent in chronic nephritis in adults than in juvenile cases; melancholia and delusional insanity occur; these mental states may assume medicolegal importance as to the testamentary capacity of the patient. (*e*) *Special senses*. Sudden, complete and bilateral blindness is cortical in origin; this *amaurosis* may be an initial symptom; there are usually no retinal findings and the condition lasts but a few hours, though in exceptional cases edema or choking of the disk is found. The pupils are wide and reactionless in acute eclampsia, but are small and mobile in chronic uremia. There may be sudden or gradual uremic deafness and tinnitus aurium. Barker remarked exophthalmos in half his patients, especially those with a suburemic state, retinitis and hypertension; most of these cases also exhibited the signs of v. Graefe, Möbius and Stellwag.

Digestive Symptoms.—These rank second to the nervous symptoms in frequency. The breath is often offensive and sometimes has an odor of urine, which Senator considers of prognostic value, for it often precedes eclampsia. The tongue is foul, the mouth inflamed and the saliva increased (uremic stomatitis). *Nausea and vomiting* are partly central and partly local from decomposed urea, eliminated vicariously into the stomach; the same may be said of *hiccough* and *diarrhea;* the HCl is decreased. All these symptoms may be very acute. Diarrhea may be serous or dysenteric from the B. dysenteriæ. Mathieu and Roux in a study of 23 cases of "uremic ulcers," described by Treitz (1859), found them chiefly in cases of advanced nephritis in subjects under twenty years old; they rarely occur in the stomach; diarrhea is common, but bowel hemorrhage and constipation are rare. They are seemingly produced by necrosis due to toxemia. The prognosis is almost always unfavorable.

Respiratory and Cardiac Symptoms.—*Dyspnea* is very common. It is sometimes toxic and central, occurring in close parallelism with cerebral manifestations. In other cases asthma uremicum is cardiac. *Prodromal slowing* of the heart to 60 to 40 beats is frequent in incipient uremia. The dyspnea may be continuous or paroxysmal and is often nocturnal; Cheyne-Stokes's breathing is an unfavorable sign, though recovery is

possible. Obstinate hoarseness and a rare form of laryngeal spasm are recorded.

5. Other Complications and Symptoms.—(*a*) Various *hemorrhages* occur; in the brain in 12 per cent. of cases; also in the dura, nose, bronchi, stomach, bowels, uterus, conjunctiva or retina. Purpura occurs especially in recurrent hemorrhagic nephritis. The author observed 11 cases of profuse gastro-intestinal hemorrhages in uremic subjects. (*b*) *Inflammations:* Chronic bronchitis always suggests kidney disease. Pericarditis, pleuritis and pneumonia especially, may develop as terminal infections. Many deaths from pneumonia are due to a previously unnoticed nephritis. Laryngitis and acute inflammatory edema glottidis may occur. (*c*) *Edema* is a late symptom and results not from the nephritis, unless it is of the mixed type, but from cardiac failure. Ascites is nearly always cirrhotic or cardiac. (*d*) The *skin* is usually dry, harsh and sometimes covered with urea crystals—"urea frost" or uridrosis. Itching, erythema, urticaria, eczema and numbness or "dead fingers," cramps in the calves, unusual susceptibility to cold (cryesthesia), neuritis or rheumatoid pains are probably toxemic.

Diagnosis.—There are *three cardinal findings, cardiovascular, urinary* and *retinal;* as Senator says, "He who examines the urine and heart in every case rarely fails to diagnosticate nephritis."

Cardiac Symptoms.—The very interdependence of cardiac and renal changes may prove confusing; thus primary cardiac disease may cause renal congestion, embolism or even acute or chronic nephritis; again alcohol, syphilis, etc., may be a common cause of arteriosclerosis, myocarditis, high blood-pressure and nephritis; and finally, the heart lesion may be secondary to renal disease. Gallop-rhythm is more common in the cor renale than in primary cardiac affections.

Urinary Findings.—In *diabetes mellitus* there is polyuria, in which the high specific gravity and glycosuria are distinctive; in *diabetes insipidus* the absence of albumin, casts and cardiac changes are characteristic; and *pyelitis* is characterized by albumin and pyuria but not by casts or cardiac changes. In the *arteriosclerotic kidney* arterial and cardiac antedate the urinary changes (the converse is true in interstitial nephritis), the specific gravity is higher, albumin less in amount and in frequency of occurrence and the course is much more rapid (see page 640).

Uremia.—Miliary tuberculosis, meningitis, cerebral abscess and sepsis may be diagnosticated uremia, because nephritis is also present and there is danger of mistaking uremic symptoms, as hemicrania, nervous symptoms, dysentery, etc., for independent disease. Many authors have remarked on the lack of correspondence between the clinical and pathological findings in nephritis. Uremia is assumed to be the cause of nervous symptoms only when other causes may be excluded, because in the course of renal disease organic lesions, as cerebral hemorrhage, may simulate uremia. Though the urine is usually decreased, Liebermeister instances a case in which, immediately before a uremic seizure, both urine and urea were increased twofold. Biermer reports an anuria which lasted two hundred and twenty-two hours before uremia developed.

Apoplexy is probably present when pressure symptoms, as vomiting or slow tense pulse, complete hemiplegia or conjugate deviation exist; it is rarely completely simulated by uremia. Meningitis, insolation and alcohol or opium coma are rarely simulated. The author has seen a few cases in which uremia simulated typhoid or sepsis by the constant high temperature.

Prognosis.—It is often detected only when the patient suffers from cardiac insufficiency, beginning uremia, apoplexy or intercurrent inflammation; for this reason this incurable malady is seldom treated in its early stages. The clinical course usually runs five to ten years after albuminuria is found; a course of twenty to thirty years is reported. Much depends on the docility and financial ability of the patient. Marked arteriosclerosis hastens the fatal issue. *Retinitis* is an ominous symptom and increases the gravity of the outlook fourfold; in Belt's 419 cases 72 per cent. died in one and 90 per cent. in two years. Webster, Wert, Hare and the author report cases which lived from four to ten years after retinitis was found. Uremia, pneumonia, apoplexy and other complications are unfavorable.

Treatment of Chronic Nephritis, Parenchymatous and Interstitial. — Both forms are considered together, because they often blend.

1. Prophylaxis.—Prevention and treatment of the cause have a very limited practical application. Irritating foods, excessive eating, all forms of intemperance, overwork, neglect of infections and dyspepsia come under this head, but are usually treated too late.

2. Rest, Exercise.—The kidneys may be relieved by rest in the horizontal posture, as recognized by Bright. Rest should be absolute as long as the heart is weak, the urine scanty or hydrops conspicuous. In moderate renal involvement or in intervals of improvement moderate exercise is advisable. Rest in bed lessens the amount of waste products to be eliminated by the kidneys and so protects the skin and dilates its vessels that the cutaneous functions become more active. The author treated 2 severe cases of parenchymatous nephritis by rest in bed for a year and permanent recovery resulted. Fatigue, dampness and cold must be avoided, by rest in bed during severe symptoms or acute exacerbations and by relative rest and warm clothing in cases of moderate severity. In men leading a strenuous life, moderate nephritis is often improved when nervous tension is relieved; in any event exercise should supplant work and not be taken after hard, nervous strain.

3. Springs, Climate.—At water resorts the importance of diet and hygiene is impressed on the patient; the suggestive element is also important. Diffuse nephritis (with both parenchymatous and interstitial changes) is most benefited. Only patients without edema or serious heart involvement should be sent to such places as Poland Springs, Bedford Springs, Saratoga, Vichy or Neuheim. A stay in California, Mississippi or Texas during the winter, at least, will materially benefit.

4. Diet.—The diet must be non-irritative, must not especially tax the kidneys in its elimination, must not burden the circulation and must maintain nutrition. Disease of the glomeruli alone may be compensated

by vicarious excretion of water through the lungs, skin and intestines, but disease of the tubules whose function is elimination of the end-products of protein metabolism, cannot well be compensated by other structures, whence the indication for *limitation* of *albuminous foods* and substitution by fats and carbohydrates. It is less important what protein the kidney can eliminate than to determine what amount of protein must be ingested to spare the body protein, *e. g.*, the myocardium from fatty change; 50 gm. is sufficient for a short time and 85 gm. over a longer period. The small amount of albumin lost through the kidneys can be offset by a glass of milk daily; in this respect nephritis differs from diabetes in which the system is irritated by unappropriated sugar and loses a vast number of units of heat and energy. It is a mistake to gauge the severity of a nephritis by the degree of albuminuria or to estimate improvement by the influence of diet upon the albuminuria alone. Fats as butter, olive oil and cream, are very important in replacing albumin. As in diabetes the restriction of diet most of all concerns the maintenance of nutrition, especially when the patient suffers from parenchymatous nephritis, the form in which restriction of diet is theoretically most needed, but in which anemia and malnutrition distinguish it from the interstitial type. The patient is considered more than the disease or the type of the disease.

The *milk diet* is free of extractives and poor in salt, and influences the albuminuria more favorably than other proteins. It has its restrictions; it is bulky, 3.5 quarts being required to maintain nutrition at rest (2200 calories), and it therefore may cause dyspepsia, containing an excess of about 60 per cent. of protein; it lacks iron, but this can be easily compensated; it contains too much water, thereby overloading the heart; and its phosphates tax the kidneys. One to one and a half quarts of milk with a pint of cream, fats and carbohydrates (baby foods, sago, rice or potatoes) meet the needs of metabolism; over half of the phosphates can be precipitated in the intestine by calcium carbonate, gr. x, with the milk. It should be taken in sips, as it is a food, not a beverage. Milk was a favorite article of diet with the older masters of medicine; a short time ago we heard that it contained an excess of water; now it is excellent because it contains little salt!

The present reaction in favor of *meat* is extreme, as it promotes uremia. The average daily amount is 100 gm. There is no difference between red and white meats as to extractives or purin bodies. Some meat is preferable to overloading the stomach with a bulky vegetable diet, save when uremia is imminent. Boiled meats contain less extractives. Game is interdicted.

Raw *eggs* increase (perhaps induce) albuminuria; three cooked eggs are allowed. *Substances excreted with difficulty* include the following, according to von Noorden: Urea (in meat), creatinin (in meat broths, extractives and to a less extent in eggs), urinary pigments (in hemoglobin), hippuric acid (in cranberries, fruits with pits, prunes, plums and gages), phosphates (in milk) and inorganic sulphates (in meats); uric acid and alloxur bodies found in glandular organs—the thymus, sweetbreads, liver and kidney—in broths and coffee are eliminated readily.

The *chlorides* and also sod. bicarb. and phosphate are especially avoided in parenchymatous nephritis with edema. As pointed out by Widal, Lemerre and Javal, in 1902, administration of salt (which is retained in the body) increases the body weight, induces edema and aggravates albuminuria, while "dechloruration" of the food reduces weight, edema and albuminuria. Salt probably raises arterial tension. The tissues ordinarily demand 2–3 gm., but most people take 10 gm. or more. Milk contains 2 gm. per liter and meat soups 6–8. The salt in bread and butter may be disregarded. Rice, potatoes, sugar and eggs may be given. In acute or early nephritis, salt is excreted in excess (polychloruria), as the tubules are oversensitive; later they are less sensitive and there is achloruria; the pot. iodide and phenolsulphonephthalein tests concern the same tissues. Widal differentiates two stages of edema, the deep or invisible and the obvious; the former is distinguished only by regularly weighing the patient. He designates the retention of chlorides as hydropic uremia and nitrogen retention as dry uremia. *Irritants*, as cheese, relishes, horse-radish, onions, rhubarb, garlic, radishes, asparagus, spices, sauces, broths, beef-tea and alcohol, should be absolutely avoided; tea and coffee because they may aggravate or produce nephritis and tobacco must be limited.

5. Water.—In regard to water, the same extremes of opinion are noted; fluids were once excessively restricted and later undue quantities were recommended; von Noorden states that flooding the vascular system with water overtaxes the heart; "water can only be excreted by the kidney if the blood-pressure is increased; many patients having weak hearts give a history of excessive water drinking; in such cases improvement is frequent if the ingestion of water is limited." The maximum elimination of waste substances occurs on about 1500 c.c. of fluids; 500–1000 c.c. are given when there is edema. We consider the weight of the patient and the in- and output of fluid from the kidneys and bowels. The urinary output is 70 per cent. of the water ingested. 1500 c.c. of water ingested (+ 500 in food) = 1500 c.c. of urine (+ 500 c.c. excreted by the lungs and skin). In early or acute nephritis, there is polyuria, due to increased sensitiveness of the bloodvessels, and loss of power to concentrate the urine; later there is normaluria and eventually oliguria or anuria (decreased sensitiveness of the glomeruli).

The following therapeutic measures are but symptomatic:

6. Diuretics.—Diuretin produces no renal hyperemia but acts directly on the epithelium and sometimes reduces the albuminuria (*v.* page 377). The use of diuretics is indicated by increase of hydrops, scanty urine and imminent uremia. Theocin is more efficacious, given well diluted to avoid nausea. Diuretics first increase, and then decrease the urine (fatigue of the kidneys); they are injurious when the organs have lost their power to secrete. The refrigerants (cream of tartar, ℥j) or potassium citrate (gr. xx–xxx) are the safest diuretics; they are thought to abstract water from the tissues and facilitate the excretion of urea, uric acid and similar products. Basham's mixture (liq. ferri et ammonii acetatis) as a tonic and diuretic should be given in doses of 1 or 2 drams. Digitalis perhaps is a renal stimulant.

7. Hydrops.—Hydrops necessitates treatment because it prevents movement, threatens suffocation by pulmonary or laryngeal edema, increases the danger of secondary infection, as erysipelas or cellulitis, embarrasses the circulation by pressure on the afferent capillaries and efferent lymphatics and venules, interferes with digestion and increases the danger of uremia. (*a*) Though *cathartics* may eliminate fluids as well as solids, drastics must be given with due care for the digestion and nutrition; vegetable are better than saline cathartics. Purging in nephritics may increase the protein in the movements even to 30 per cent. (normally 10 per cent.). Watery stools are produced by pulv. jalapæ co., ℥j, or elaterinum, gr. $\frac{1}{16}$, but not by cascara, rhubarb, aloes, senna or phenolphthalein; liq. magnes. citrat. contains an excess of water and Pluto, etc., an excess of chlorides; concentrated Epsom salts may in-

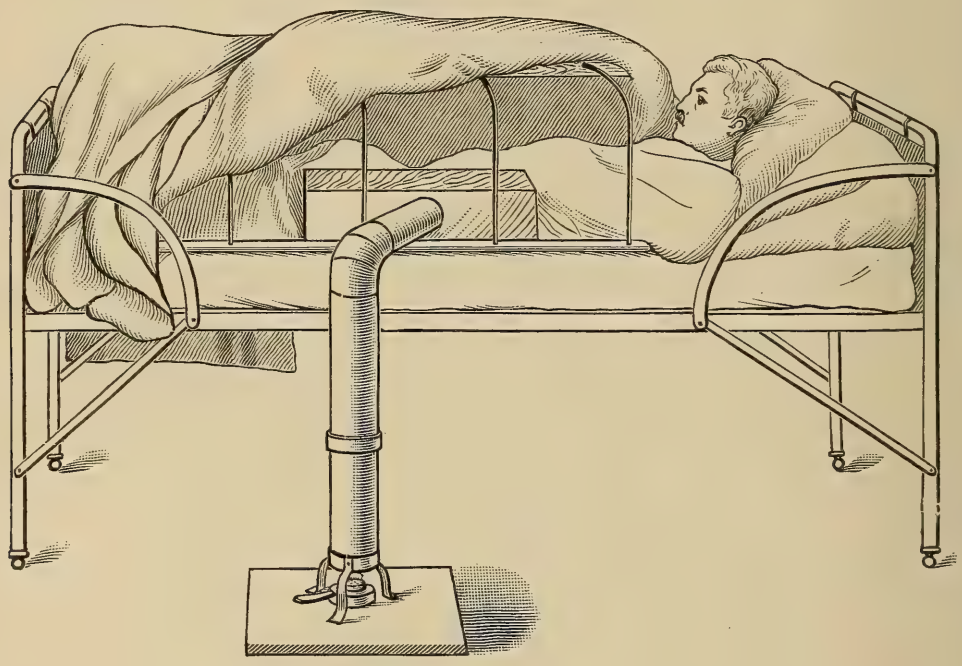


Fig. 48.—Sweat bath employed in nephritis.

duce fatal results. (*b*) *Sweats.* Though the functional relation between the skin and kidneys cannot be overestimated, profuse sweating eliminates fluid rather than solids (about 2 gm. each of Cl and N per liter). When a patient sweats one quart (1000 c.c.) 7 to 15 grains (0.5 to 1 gm.) of urea are eliminated. Sweats do not concentrate the blood and predispose to uremia, but lower the blood-pressure 10 to 20 mm., by diverting blood from the splanchnic to the cutaneous vessels. The author has seen a fatal issue result from sweating and prefers the old-fashioned full warm bath of Osborne and Liebermeister, to the more active methods; an ice-bag is placed on the head to prevent brain congestion. Commencing with water at body temperature this is raised five or six degrees. After twenty or thirty minutes the patient is removed and wrapped—without drying—in blankets. A hot-air bath may be given by placing a lamp under one end of a stove-pipe which runs by an elbow into a tent built

over the patient. In the alcohol sweat, hot bricks protected by flannel are placed around the heavily covered patient; equal parts of alcohol and water are poured on the bricks and the patient is covered again; the steam vapor is well tolerated unless the heart is weak, when fatal collapse may ensue. It is often unsafe to give pilocarpine, gr. $\frac{1}{5}$ (page 623), because it may produce vomiting, diarrhea, syncope, pulmonary edema and sudden death, even when the heart appears strong. (*c*) Mechanical relief by means of *incision* is superior to all other measures which must first bring the fluid with all its urea and extractives into the circulation, with the danger of inducing uremia. The author makes deep incisions, three inches long, over the lower leg (not over the malleoli where infection from the feet is likely). In testing the fluid evacuated there has frequently been 0.1 per cent. urea, a measure for other retained products of kindred nature; quarts often seep out. Free incision is obviously less dangerous than punctures or Southey's capillary tubes.

8. The Circulation.—Treatment often resolves itself into the care of the heart. Excitement, nervous strain, physical exertion, constipation, tobacco, coffee, alcohol and exposure to heat must be avoided, as cardiac dilatation is the inevitable fate of hypertrophy. In the uremic seizures, with the same symptoms in other respects, the heart may beat strongly or weakly. (*a*) *With cardiac overaction* observed early in uremia, the high tension must be corrected by *vasodilators* (see pages 399 and 400). Iodides are beneficial when there is no urgency, grs. x, t. i. d.; iodism, especially salivation, may result from renal retention. When extreme tension and imminent uremia exist, tr. veratri viridi, ♏v–x, every half-hour for three doses may be given; the author prefers spiritus glycerylis nitratis, ♏ij, until results are produced; vasodilators are often given to excess, as hypertrophy and increased arterial tension are less pathological than compensatory conditions. Chloral frequently operates better than any other remedy. Lowering the tension 30–40 points, decreases elimination of solids and fluids. *Bleeding* may relieve the intracranial blood-pressure, lessen the molecular concentration of the blood (*i. e.*, its toxicity), relieve the laboring heart, and avert for awhile the uremia; salines may be given afterward, either subcutaneously or by rectum; they "lavage the blood." An excess of salt may be given in the infusions (*v. s.*) and when they are repeated the salt should be left out, for it may induce edema of the lungs. Bouchard states that the abstraction of 32 gm. of blood removes more toxins than 280 gm. of fluid feces or 100 liters of sweat. Increased intracranial tension may be relieved by *lumbar puncture;* the normal pressure of 120 mm. is increased, sometimes to 600 mm.; the author remarked relief of the headache, vomiting, convulsions and coma. (*b*) When the *heart is weak*, rapid, irregular or galloping, cardiac stimulation by digitalis and strychnine is indicated. Infusion of digitalis by mouth may cause vomiting and ♏ij–v of the fluidextract may then be given hypodermically or by rectum. Its combination with calomel may cause the most intense salivation and in one instance necrosis of the entire tongue. Cardiac unrest, palpitation or dyspnea should be relieved by a hypodermic of morphine, by application of *ice to the heart*

or by champagne, nitroglycerin, bromides and valerian. Pilocarpine and sweats overtax or fatally depress the heart. Von Ziemssen gave champagne and digitalis by mouth as long as the patient could swallow and then digitalis by rectum and camphor subcutaneouslv.

9. Vomiting.—Vomiting and diarrhea are as often vicarious as toxemic and should not be checked at once. Gastric lavage and colonic flushings are beneficial. If improvement ensues, a starvation diet should be instituted.

10. Convulsions.—Hypodermics of morphine should be given (though medical opinion is generally against it), with chloral (gr. xv every hour for not more than four doses).

11. Operation.—Many cases of "decapsulated" kidney are not genuine nephritis, but albuminuria from floating kidney or ascending infection, and nephritis is more than merely a renal disease, perhaps a result of a general blood condition.

PASSIVE CONGESTION. EMBOLISM.

I. Passive Congestion.—Venous hyperemia is an important lesion compared with arterial hyperemia, of which latter little is definitely known, though it occurs in renal inflammation, diabetes and after the administration of diuretics.

Etiology—(*a*) Among the chief causes of *general stasis* are cardiac insufficiency from valvular disease, myocarditis and pulmonary emphysema. (*b*) *Local stasis* has scarcely any part in the renal pathology. Berkmann (1859) described renal stasis and thrombosis of the renal veins occurring in the newborn or nurslings after severe diarrhea; when unilateral there is renal pain, great albuminuria, some hematuria, swelling of the kidney and decreased urine; most cases of bilateral thrombosis die from uremia. Thrombosis of the cava and pressure by large tumors may cause passive congestion.

Pathology.—The slowing of the arterial flow and to a less degree venous hyperemia, alter the integrity of the secreting renal structures, notably the glomeruli. In experiments, arterial insufficiency causes initial glomerular alteration, while venous obstruction causes primary congestion in the medullary portion.

Early Changes.—The kidney is swollen, dark and firm and the stellate veins are prominent. On section it expands beyond its close-fitting capsule, which is readily stripped off. The organ is very vascular and may drip blood. The glomeruli are seen as dark red points and the medullary bloodvessels are clearly defined. Microscopically the conspicuous changes are hyperemia, occasionally with punctate hemorrhages, fatty and cloudy degeneration of the epithelium of the tufts and convoluted tubules, escape of albumin into them, forming hyaline casts in the tubules, and finally some thickening of the vessels.

Late Changes.—Prolonged stasis leads to greater atrophy of the secreting structures and to increase of connective tissue; this "cyanotic induration" has been rather overdrawn in clinical descriptions, but may result from extreme and protracted stasis. The kidney becomes pale and

firm, the cortex shrinks and connective tissue develops in the medulla. Microscopically, fibrillar connective tissue, thickening of Bowman's capsule and epithelial degeneration are found.

Symptoms.—The urinary findings are of chief interest. The urine is decreased and its amount directly follows the heart's activity. It is acid, dark, concentrated (specific gravity 1.025 to 1.030) and it has a lateritious sediment of urates and uric acid. Albumin and globulin are present in moderate amounts. Microscopically a few leukocytes, epithelial cells and hyaline casts are found; granular casts are seldom found; red cells indicate nephritis or infarction.

Diagnosis.—The urinary findings are significant in association with the causal lesion; edema begins in the legs; the nutmeg liver, dyspnea, cyanosis and pulmonary stasis are present. The percentage of albumin runs parallel with the strength of the heart, increasing with its failure or decreasing if digitalis or strychnine improve its action. If cardiants increase the urine, and if abundant albumin and casts still remain, there is probably coincident nephritis. (See page 640.)

II. Embolism.—Valvular disease of the left heart is its predisposing cause. Sixty-six to 75 per cent. of all embolisms are renal. Stoppage of a renal terminal artery causes the well-known wedge-shaped anemic infarct in twenty-four to thirty-six hours; the local ischemia causes fatty degeneration, necrosis and a finely granular transudation into the connective tissue and glomeruli. Fibrous substitution occurs with ultimate shrinkage and deformity. Repeated infarctions produce the irregular "embolic contracted kidney." The process may be innocent (mechanical) or less commonly suppurative.

Symptoms.—In most cases there are no symptoms. Exceptionally a diagnosis is possible, based on (1) the valvular lesion; (2) sudden onset with fever, chills and vomiting; (3) lumbar pain and tenderness and (4) hematuria and albuminuria. Treatment is symptomatic.

AMYLOID DEGENERATION OF THE KIDNEY.

Etiology.—The etiology of amyloid kidney is that of amyloid liver (*q. v.*), *i. e., ulcerative tuberculosis, protracted suppuration* and *inveterate syphilis.* First described by Rokitansky (1842), its symptomatology was developed by Wilkes, Todd and Traube.

Pathology.—Renal amyloidosis seldom occurs without amyloid of the liver and spleen. To the naked eye the kidneys generally appear *enlarged,* weighing together even as much as 870 gm.; when amyloid infiltration is slight, there may be no alteration in size or weight; in some cases the kidneys are large from coincident chronic parenchymatous or small from interstitial nephritis. The capsule strips readily, showing a pale, smooth surface. On section the *cortex is enlarged,* waxy and pale and its *edge translucent;* the *glomeruli stand out as glistening points* and become brown when treated with Lugol's solution. Microscopically the amyloid substance can be seen clearly; it is so named because of its starch-like reaction, though chemically it is an albuminoid. It is either deposited from the blood in the small vessels or more probably

DIFFERENTIAL TABLE FOR THE DIAGNOSIS OF THE DIFFUSE DISEASES OF THE KIDNEYS. (MODIFIED FROM LEUBE.)

		Passive congestion.	Acute nephritis.	Chronic nephritis.			Amyloid kidney.
				Chronic parenchymatous nephritis.	Secondary contracted kidney.	Genuine chronic interstitial nephritis.	
Etiology.		General stasis (heart, liver, kidney) or local stasis (thrombosis renal veins, cava, etc.).	1. Cold, exposure. 2. Poisons, *e. g.*, cantharides. 3. Acute infections scarlatina, sepsis, pneumonia.	1. From acute nephritis. 2. Cold and moisture. 3. Toxic. 4. Toxemic, phthisis, malaria. 5. Heart disease.	Secondary contraction may follow, but even then the kidney remains large.	Gout, alcoholism, diabetes, plumbism, syphilis, arteriosclerosis.	1. Protracted suppuration. 2. Old tuberculosis. 3. Inveterate syphilis.
Pathology.		Kidney, larger (may shrink), cyanotic, fatty, cloudy change, infarcts.	Some swelling, exudation of cells and serum; glomerular, epithelial and interstitial changes.	1. Large white kidney. 2. Large red (variegated) kidney. Fatty changes in tubules. Some connective change, especially in 2.	Contractions less than in genuine interstitial form, less cysts, larger prominences, larger granulations and more yellow and smooth.	Very small, atrophied, indurated kidney with fibrous hyperplasia, epithelial atrophy, adherent capsule, small granulations, etc.	Large, homogeneous waxy, vascular infiltration.
Urine	Quantity	Scanty; varying with heart's activity, whence prognosis from amount, and from action of digitalis.	Decreased (glomerular changes), suppression even.	Decreased (tubular and glomerular changes); 200 to 1000 c.c.	Somewhat increased, 1500 to 2000 c.c.; at least more than normal.	Very abundant, polyuria, pollakiuria, usually 2000–3000 c.c. (rarely 4000–5000).	Normal or increased; variable.
	Color.	Dark red.	Turbid (urates or blood)—pale red to dark red.	Opaque: "meat water" appearance; urates suspended in albuminous urine.	Moderately clear.	Pale, clear.	Clear yellow.
	Specific Gravity.	High, 1.025–30.	High, 1.025–30–40.	Normal or somewhat higher, 1.020.	Somewhat under normal.	Low, 1.010 being the average.	Normal or less.
	Albumin.	Due to arterial anemia, moderate, 0.5 per cent., varying with condition of heart.	Much; $\frac{1}{3}$ to 1 per cent.	Much; $1\frac{1}{2}$ to 3 per cent. (very rarely absent).	Considerable.	0.5 per mille, slight, intermittent, latent or long absent.	Usually abundant.
	Sediment.	Moderate, urates, hyaline casts, few red cells.	Great; much urates, red and white cells, blood, epithelial, waxy casts.	Great; red and white cells; casts, all kinds, granular and fatty especially; fatty cells.	Moderate—many casts of all types.	Scanty; few hyaline (granular) casts.	Usually none, unless coincident nephritis.

Solids.	Practically normal, urea decreased, chlorides normal.	Decreased chlorides, urea, phosphates.	Decreased.	Markedly decreased.	Decidedly decreased urea, ammonia, uric acid, chlorides, phosphates.	Normal.
Blood.	Absent, save for stray red cells; blood from infarct.	Much.	Usually some; more in large red kidney (chronic hemorrhagic nephritis).	Little or none.	Absent.	None.
Heart Hypertrophy.	Changes depending on valvular lesion, etc., present.	None as rule (very rarely early hypertrophy).	Occasional, eccentric hypertrophy; especially in diffuse nephritis.	Usually present.	Almost invariable (concentric hypertrophy).	None in pure cases.
Hydrops.	From stasis; stationary, mechanical, in feet first, then abdomen, pleuræ, etc.	Usually marked, rarely absent; mostly in scarlatinal and exposure nephritis; from vessel changes.	Usually extreme and obstinate (in skin, serous cavities and viscera); marked vascular change; somewhat less in large red kidney.	Moderate; less than in large white kidney but more than in true contracted kidney.	Absent, unless in stage of cardiac incompetence.	Marked, from cachexia or vessel infiltration.
Anemia.	Often present when is usually associated with cyanosis.	May be some.	*Very* frequent; a cardinal symptom with the urinary findings and hydrops.	Moderate.	Usually none (except in young subjects).	Marked.
Uremia.	Absent.	Frequent; especially when due to cold and scarlatina.	Moderately frequent.	Frequent.	Most frequent.	Never.
Visceral Symptoms.	Lung congestion, stasis liver, infarcts spleen, etc., dyspnea.	Pulse somewhat tense; often latent; fever due to other causes.	Anemia; retinitis; bronchitis. Fairly frequent.	Retinitis, bronchitis, etc.	Retinitis and arteriosclerosis more than in any other forms.	No retinitis. Amyloid degeneration of liver, spleen and alimentary tract.
Course.	Depends on age and extent of lesion; may be long.	Average duration 1½ weeks; usually wholly disappears if patient lives; sometimes becomes chronic (scarlatina).	½ to 1½ years (may be longer in chronic hemorrhagic nephritis).	Slower than in large white kidney, but faster than in the genuine contracted kidney; 1½ to 3 years.	Much longer than in other nephritides, 5 to 10 years.	Depends on causal disease.
Cause of Death.	Cardiac dilatation, embolism, coronary disease.	Death from causal disease; uremia, hydrops (glottis), edema of lungs, inflammation (pericardium, pleura, lung, etc.).	Uremia, edema glottis, hemorrhages, marasmus, sepsis, acute exacerbations, but mostly (visceral) inflammations.	Uremia, cardiac insufficiency, myocarditis, hemorrhage (cerebral), retinitis, inflammations (pleural, pericardial, pneumonic, cutaneous) very common.		Causal disease, hydrops, cachexia.

formed *in situ*, especially in the glomeruli, then in their afferent vessels and the straight vessels of the medulla. The renal interstitium is involved secondarily; the *membrana propria* may be affected but the cells may suffer only secondary compression, degeneration and atrophy.

Symptoms.—(*a*) *Urinary symptoms* are exceptionally absent. The urine is generally *limpid and increased*, but may be decreased when amyloid degeneration is associated with parenchymatous or greatly increased when associated with interstitial nephritis; some writers place undue importance upon variability in its amount. The specific gravity is 1.010 to 1.015 or higher in syphilitic cases. *Albuminuria* is *usual*, being absent only in exceptional cases which invade the vasa recta and evade the glomerular capillaries; albuminuria results from increased permeability of the glomerular vessels and may amount to 1 or 2 per cent.; Senator remarked the large amount of globulin in the urine. There is little change in the solids; indicanuria is not infrequent. Microscopically hyaline casts may be present and perhaps a few leukocytes and epithelia, but the sediment is scanty in most cases; the so-called waxy or amyloid casts found in severe nephritides have no relation to amyloidosis of the kidney. (*b*) Cardiac hypertrophy, uremia and retinitis do not occur in renal amyloidosis, except when it is associated with nephritis. (*c*) Amyloid degeneration of both the *spleen and liver* and sometimes the alimentary tract is associated in 66 per cent. of cases; these and the causal disease are necessary for diagnosis. (*d*) *Edema* and *anemia*, which is almost cachexia, are usual in diffuse amyloidosis. Hydrops of the serous sacs, especially *ascites*, is less common than edema and usually occurs as a late or terminal event. *Differentiation* (*v.* p. 640).

Prognosis.—Amyloid disease is incurable, though light grades may recede. Amyloid of the kidneys in most cases runs its course in a year or less, particularly when associated with parenchymatous nephritis. Cases may endure for several, even fifteen years, but these are generally complicated by contracted kidney.

Treatment.—With modern surgical methods and early treatment of tuberculosis and syphilis amyloid disease is less common than formerly; for the developed disease there is no treatment.

MALFORMATIONS OF THE KIDNEY.

I. Malformations and Structural Anomalies of the Kidney.—These are of little clinical importance. Complete absence or rudimentary development of both kidneys occurs only in unviable infants.

One kidney may be *absent or rudimentary*, whereon the normal kidney vicariously hypertrophies and may be mistaken for malignant disease, whence its removal is fatal. In Ballowitz's 184 cases 61 per cent. occurred in males and in 63 per cent. the left kidney was lacking. Absence of one kidney is usually associated with unilateral malformation of the internal genitalia, but this is not true in unilateral renal atrophy or in horseshoe kidney. Before operation cystoscopy is imperative in doubtful cases to determine the presence of two ureters. Two ureters may come from one kidney.

Fifteen cases of *supernumerary* kidneys are recorded (Graser) and in 2 cases there were four kidneys.

The "*horseshoe*" kidney (*ren arcuatus*) due to the fusion of both kidneys, is fairly common; it may cause pain. Errors in diagnosis are common, especially if the kidney is dislocated and are inevitable if the renal pelvis is inflamed or dilated. The *ren arcuatus* has been mistaken for a tumor and extirpated.

II. Movable Kidney.—*Congenital dislocation* occurs twice as frequently in males as in females and is usually left-sided, wherein it differs from the acquired form, and frequently coincides with congenital anomalies of the genitalia and intestines. The kidneys may lie one upon the other (dystopia renum cruciata), of which Chatelin could collect but 15 cases.

Acquired dislocation, described by Rayer a century ago, is much more common and important. It is called movable or floating kidney, ren mobilis s. migrans, ectopia renis acquisita or nephroptosis.

Etiology.—(*a*) *Sex;* 88 per cent. occur in *women.* (*b*) *Age;* most cases occur between the twentieth and fiftieth years. Comby reports 18 and Abt 5 cases in children. (*c*) The kidney is supported by the fat around it, the peritoneum in front of it and by its own bloodvessels. Among the *alleged causes* are: atrophy of the perirenal fat; the presence of a *mesonephron* surrounding the kidney to a variable extent; *weakening of the abdominal walls* by pregnancy and other causes; alterations in the intra-abdominal pressure by *malpositions of the uterus;* pressure by *corsets; congenital laxness of the supporting structures,* in connection with which Stiller claims that the tenth rib is usually "floating" like the eleventh and twelfth ribs (costa fluctuans decima); tumors of the kidney, which drag it downward; and trauma or sudden efforts. Enteroptosis is frequently observed with floating kidney.

Symptoms.—Symptoms are absent, probably in most cases; they may date from the time when the physician incautiously remarks upon the presence of a "floating kidney."

1. Pain.—Pain may be merely a dragging sensation or severe renal colic, from stretching of the renal nerves; it may be lumbar, abdominal or may shoot toward the chest, thigh or sciatic region; it may be constant or intermittent like a crisis (*v. i.*).

2. Objective Kidney Findings.—On bimanual examination with one hand over the kidney in front and the other pushing it up from behind, the findings vary; (*a*) frequently the *kidney* is not painful or tender, but *palpable,* which condition has no significance, especially in women; (*b*) the kidney may be *movable,* descending on deep inspiration, maybe to the navel, where it can be retained by pressure during expiration; movable kidney is found in 20 per cent. of women; (*c*) the most extreme type is *floating* kidney, which may wander to the lower abdomen or pelvis, even into a hernial sac. In 76 *per cent. of cases the right kidney* is involved, in 11 per cent. the left and in 13 per cent. both kidneys (Küttner); the right kidney has longer vessels, but the left renal vein is more firmly connected with the suprarenal vein and the pancreas. When the kidney is grasped a sickening sensation sometimes results, as when the testicle is compressed. The *smooth tumor* is recognized by its

shape. In rare cases the pulsating renal vessels are felt. It may be felt only in the standing posture or in the left decubitus. Occasionally tympany can be found along the spine, over the dull area normally occupied by the kidney, which merges with the liver and spleen above and extends outward 2.5 inches on the left and 3.5 inches on the right side. In thin subjects the sinking-in of the back and the tumor in front may sometimes be clearly seen.

3. Complications.—(*a*) The same train of *neurasthenic*, hysterical or hypochondriacal symptoms are present as in enteroptosis. Nervous dyspepsia is particularly common. (*b*) Dietl (1864) described sudden severe pain and collapse known as "*Dietl's crises;*" they are marked by fever, chill, vomiting, collapse, severe pain, sometimes with swelling and tenderness of the kidney, scanty urine containing blood, pus, uric acid and oxalates and in some instances peritonitic manifestations; Dietl's theory that the kidney became "incarcerated" between the connective tissue and peritoneum is now abandoned; in one of Sutton's cases the ureter was found twisted three times, causing acute renal retention and hydronephrosis. Perhaps the renal vessels suffer torsion or the crises are due to acute nephritis or peritonitis. (*c*) *Dilatation of the stomach* is only a coördinate and not at all a common coincidence. (*d*) *Dysmenorrhea*, constipation and enteroptosis are common complications. Colitis mucosa, compression of the intestine, icterus, abdominal aneurysm by tugging upon the aorta and compression of the cava inferior are rare and questionable sequences. Albuminuria may occur periodically, on palpation of the kidney or when the patient stands (orthostatic type).

Diagnosis.—The finding of the sensitive kidney-shaped movable tumor is most important. It may be mistaken for movable omental, gastric, intestinal and gall-bladder tumors or corset liver. Particular reserve is necessary in the diagnosis of left-sided floating kidney. Severe pain may be confused with a tabetic crisis, appendicitis or the passage of gall- or kidney-stones.

Prognosis.—Death is an extremely rare occurrence.

Treatment.—The patient should never be told of a movable kidney, because neurasthenic symptoms may date from the diagnosis. (*a*) A *rest cure* may develop adipose tissue and relieve the nervous symptoms. (*b*) A well-fitted *bandage*, with padding to prevent renal descent, is excellent in many cases. (*c*) In obstinate and extreme cases, *nephropexy* is indicated. Hahn's results are 75 per cent. complete recovery, 15 per cent. decidedly improved and 10 per cent. failures; the mortality is under 1 per cent. Nephrectomy is seldom indicated. (*d*) The crises are treated by morphine, reposition and later by operation.

ANOMALIES OF RENAL SECRETION.

I. Albuminuria.—Albuminuria, discovered by Cotugno, in 1770, results (*a*) from leakage of albumin through the glomeruli and tubules (*albuminuria vera, renalis*) or (*b*) from blood, pus, semen or fluid of tumors (*albuminuria spuria*). The spurious or accidental form will be considered under Pyuria and Hematuria.

The most common albumin is serum albumin (or simply albumin) with which serum globulin is frequently associated.

Etiology.—(*a*) Its incidence in the *diffuse renal diseases*, the nephritides, amyloidosis and passive congestion has been discussed. (*b*) *Blood alterations* are frequent causes: (i) blood diseases proper, as anemia, leukemia, pernicious anemia, etc., in which the poverty of the blood or toxins induce renal degeneration; (ii) toxemic states, as acute and chronic infections, in which "*febrile albuminuria*" evidences the renal degeneration; this condition borders very closely on mild acute nephritis; (iii) diabetes, icterus and poisoning by lead, mercury, etc. (*c*) *Nervous maladies* possibly act by lowering the local blood-pressure, as epilepsy, exophthalmic goitre or migraine. (*d*) Affections of the *digestive tract*, as intestinal incarceration, hepatic disease and lead colic may produce albuminuria. (*e*) In the *newborn* it was first noted by Virchow. It is almost regular in the first week of life and occurs with hyaline casts, urates and epithelium. Ribbert holds that it is due to imperfect development of the glomeruli. (*f*) *Pregnancy;* it occurs in 40 per cent. of cases in the last few months of gestation (from increased tax on the kidneys and from pressure) and in the same percentage after delivery (from effort and strain). (*g*) Is there a "*physiological albuminuria*"? Various types are described, as Pavey's cyclical albuminuria, which appears and disappears; intermittent albuminuria; adolescent albuminuria, in young, anemic subjects, often with vasomotor disturbances, or orthostatic albuminuria, also termed postural or lordotic, which appears only when the subject stands; the latter types give the euglobulin reaction, show a lower blood-pressure (by 10–20 mm.), when the patient sits than when he lies down and are harmless. Some bring under this caption albuminuria after heavy eating, severe exercise, coitus, mental strain or cold baths. Senator, Bradford and Grainger Stewart, whose names alone carry weight, insist that there is a physiological albuminuria, in which signs of nephritis, as casts or cardiovascular changes, are absent. The author regards most cases of albuminuria as pathological; its cause may be an insidiously developing nephritis, a receding nephritis, stasis or may be "constitutional," as Martius calls it, but it is always a sign of glomerular or tubular lesion (see Prognosis). The amount of albumin in this type rarely exceeds 0.5 per mille (Senator).

Detection.—The urine must be clear and acid; if turbid it should be filtered and if it then remains turbid it should be cleared by shaking with magnesia; it must be acidulated, if necessary, with acetic acid; if very concentrated, dilution with distilled water is advisable.

1. Boiling Test.—A test-tube, two-thirds filled with urine, is held by its lower part and the upper layers are boiled. Albumin produces clouding, which may also result from phosphates and carbonates (dissolving when nitric acid is added), balsams (dissolving in alcohol) and urotropin. Nitric acid should not be added before boiling, for a soluble acid-albumin may result.

2. Nitric Acid or Heller's Test.—Urine, somewhat diluted, is placed in a test-tube and nitric acid is run under it by a pipette. Albumin, globulin, mucin and albumose are precipitated at once or after a few

minutes at the contact point; albumose dissolves by heating, the others do not.

3. Ferrocyanide Test.—To a test-tube half-full of urine 10 drops of acetic acid are added, which may precipitate uric acid, urates, oleo-resins or nucleo-albumin. These are filtered out and 5 c.c. of 10 per cent. potassium ferrocyanide is added. A precipitate indicates albumin, globulin or albumose; the latter dissolves on heating.

4. Acetic Acid and Salt Test.—Adding to the urine *acetic acid* to an acid reaction and then an equal volume of *saturated sodium chloride* solution precipitates albumin and albumose.

5. Spiegler's Test.—Nucleo-albumin is removed by adding 15 drops of dilute acetic acid and filtering. To the urine is added a quantity of Spiegler's reagent (bichloride mercury 8, tartaric acid 4, glycerin 20 and distilled water 200) when albumin or albumose develops at the contact zone.

Globulin occurs chiefly in renal congestion, acute and chronic nephritis and amyloid degeneration. It dissolves in an excess of acetic acid. It is precipitated by boiling. Flocculi of globulin are thrown down, when 2 drops of dilute acetic acid are added to 1.6 ounces of filtered urine diluted ten times with distilled water.

Nucleo-albumin (euglobulin, *mucin*) occurs in acute desquamative nephritis, icterus, poisonings causing necrosis of the renal cells and regularly in catarrh of the urinary passages. It is precipitated by boiling and by acetic acid but unlike globulin, does not dissolve in an excess of acetic acid. Shreds of mucus, voided with colic, have been observed by von Jaksch and Baumüller; the condition seems analogous to colica mucosa and spasmodic asthma.

Albumose is albumin which does not coagulate by heat. When the urine is boiled the albumin is precipitated and filtered out. Albumose in the filtrate causes clouding and precipitation on cooling. (*a*) *Bence-Jones's albumose* is rather a peculiar albumin than an albumose; described by him in 1848, albumosuria has also been called Kahler's disease. De Castello, in 1909, collected 52 cases. It has been found in osteomalacia, multiple myeloma, leukemia and bone-marrow tumors. It is precipitated by cold nitric acid, dissolves on heating and reappears on cooling. (*b*) Albumose may be found in various acute infections, septicemia, peritonitis, empyema, etc. Albumin in the kidney, bladder or urinal may be converted by pepsin into albumose.

Diagnosis.—Blood or pus may give albumin reactions (*v. i.*).

Prognosis and Treatment.—These are etiological. The outlook is not favorable, though not necessarily bad. In seemingly "physiological" cases, the condition of the heart must be watched and casts carefully looked for. In youth and adolescence albuminuria may be "physiological;" Senator observed such a case develop contracted kidney after twenty years; in middle and later life it is usually nephritic. Shattuck and Osler, however, hold that "albuminuria is more common apart from Bright's disease than is commonly supposed, that it increases with advancing years and that a small amount of albumin with finely granular casts of small diameter in those over fifty years of age is of small

importance." Life-insurance figures show that it entails double the average death-rate. Hawkins reported a case in which albuminuria lasted, without any unhappy symptoms, for forty-three years after Bright had given a fatal prognosis.

II. Hematuria.—Etiology.—Like albuminuria, blood in the urine is only a symptom of many diverse conditions. (*a*) It is *most frequently renal.* (i) *General conditions* may cause it, as intoxications (by quinine, carbolic acid, turpentine or salicylic acid), acute infections (malaria, exanthemata, particularly hemorrhagic types, appendicitis) or blood diseases (hemophilia, leukemia and the anemias). (ii) Other causes are *local renal conditions,* as trauma, vascular maladies (thrombosis of the renal veins, *infarct,* aneurysm, *varicose veins,* arteriosclerosis and rarely passive congestion); inflammation (acute, chronic, hemorrhagic and suppurative *nephritis*); *tumors; tuberculosis; calculous* ulceration and pyelitis; rarely amyloidosis or parasites (distoma, filaria, nephrophages sanguinarius); sometimes floating and cystic kidney, especially after bimanual examination. (iii) Hematuria may be "*idiopathic*" *or* "*essential,*" occurring in very rare cases in "apparently healthy kidneys," to which phrase there has been a just objection; it has been called renal epistaxis, angioneurotic hematuria and renal purpura. Nephrotomy may give relief. In Eshner's 48 cases of unilateral renal hematuria nothing was found at operation in 33 per cent., floating kidney in 12.5, renal pelvic changes in 19, and diffuse renal disease in 24 per cent. (*b*) Of *lesions in the lower urinary tract,* the chief types are stone in the ureter or bladder; ruptured varices anywhere in the urinary tract; stasis from liver cirrhosis or thrombosis of the iliac veins; inflammation (pyelitis, ureteritis, cystitis, prostatitis or urethritis, sometimes called "Russian gonorrhea"); and tuberculous, malignant or other ulcerations.

Symptoms and Diagnosis.—Hemorrhage may be macro- or microscopic. The urine may be clear or turbid, red or smoke-colored. It is usually acid, or alkaline from copious vesical hemorrhage. The color is brighter when the urine is alkaline. Confusion is possible with concentrated, icteric, carbolic, fuchsin, hematoxylin and senna urines. The presence of blood is positively determined (*a*) by the *microscope;* the red cells remain normal for a long time in acid urine, but may become pale in dilute and mulberry-form in concentrated urine; (*b*) by the *spectroscope;* if no red cells are present with positive spectroscopic findings it indicates hemoglobinuria (*v. i.*); and (*c*) by *chemical tests,* as Heller's, Almén's guaiac or Teichmann's test.

Diagnosis of the origin and cause of hematuria is often most difficult. (*a*) In cases of *renal* origin, the blood is intimately mixed with the urine, as much with that first voided as with the last. The color is often that of water in which raw meat has soaked ("beef-water" color). Clots are infrequent in renal hematuria, save from trauma, tumors, aneurysm or varicosities. The red cells are seldom seen in rouleaux-form and are fragmented from action of the renal cells; this does not occur in vesical hemorrhage. If there is some other renal lesion, as nephritis, there is more albumin than the red cells would account for, and albumin is present after careful filtering; if there is albumin with a red cell count,

by the hemocytometer, of less than 3000 per c.mm., the albuminuria is renal. Blood casts, granular casts and renal epithelium are indicative of renal origin. (*b*) Hematuria originating in the *pelvis and ureter* is more often marked by clots and possibly by caudate epithelium. (*c*) Blood from the *bladder* is more apt to clot and the last urine voided is more bloody than the first. Tenesmus and alkaline urine often indicate a vesical origin. (*d*) *Urethral* hemorrhage stains most deeply the first urine voided. Hematuria may be accidental, *e. g.*, menstrual.

Treatment.—The treatment is etiological. Rest, morphine and aside from the local treatment of vesical disease, urethritis and calculous pyelitis, tannic acid, thyroid extract, calcium chloride āā gr. v., t. i. d., normal horse serum and adrenalin 1 to 1000 (given by the catheter) may stop hemorrhage. In idiopathic renal hematuria, simple incision of the kidney is frequently efficacious.

III. Hemoglobinuria.—This is hemoglobin in the urine without hematuria. Though first described by Dressler (1854), most of the early accounts are English (Pavy, 1855).

Etiology.—The *toxic form* may follow transfusion of blood, poisons, organic and inorganic, as potassium chlorate, phosphorus, mercury, quinine (in malarial cases), phenol; burns, icterus, pernicious anemia, scarlatina, acute nephritis, Winckel's disease and estivo-autumnal malaria. The *paroxysmal form*, of which Gilman Thompson collected 206 cases, occurs largely in England, France and Germany; 96 per cent. are males, chiefly between thirty and forty years of age. (i) After some *infection*, notably lues, (ii) a *hemolysin* is formed, capable of dissolving the red disks of the subject or of other individuals; cold is important in anchoring the hemolysin to the red cells but does not produce this toxin; besides this type of Donath and Landsteiner (hemolysin in the blood and hemoglobinemia), there is another, characterized by abnormal fragility of the red cells; exercise, excesses or menstruation may precipitate an attack. It is observed in horses after cold and exertion, with hemoglobinemia and hemoglobinuria, paresis of the hindlegs and death from disturbed circulation and respiration. In cattle and sheep epidemics, Babes and Krogius found a hematococcus. (iii) There is some *renal disturbance* and hemoglobin is excreted, chiefly by the convoluted tubules; genuine renal hemoglobinuria (without hemoglobinemia) seems to occur in some cases of nephritis and from certain intestinal nematodes. (iv) Hemolytic *icterus* and *splenomegaly* may result.

Symptoms.—Symptoms occur only after destruction of at least one-sixth of the blood. After a chill, fever, lumbar pain which radiates to the legs, cyanosis, oppression and gastric symptoms, the urinary findings appear; the urine is dark and contains clumps of blood pigment, peculiar granular (iron) casts and epithelium stained with hemoglobin, and albumin. In most cases the hemoglobin is already altered to methemoglobin, as shown by the spectroscope. There may be slight jaundice, edema or urticaria. The blood shows increased coagulability and diminution in the number and redness of the red cells, which do not form in rouleaux. There may be swelling of the liver and spleen from hemoglobin deposits in them. The paroxysm lasts one-half to several

hours and in the interval the general health is fairly good. The course is most chronic. No death is known from the paroxysm itself and very rarely from the disease.

Treatment.—Prophylaxis in regard to cold and exertion may modify the attacks. Attacks are sometimes aborted by nitrite of amyl inhalations and warmth may modify the seizure.

IV. Pyuria. — Etiology. — (*a*) Pus may occur intermittently, as in pyonephrosis or continuously, as in calculous or tuberculous *pyelitis*. The urine is usually acid in tubercle, colon and proteus infections and in some infections ascending from the bladder; the staphylococcus may render the urine less acid than normal or alkaline (*v. i.* Pyelitis). (*b*) In *cystitis* pyuria the urine may be acid or alkaline and contains ropy mucus and triple phosphates. Pus is seen especially in the last urine voided. (*c*) *Urethral* pyuria, usually gonorrheal, is greatest in the urine first evacuated. (*d*) *Accidental* pyuria from leucorrheal contamination is excluded by catheterization. In pyuria from rupture into the urinary tract of appendicular, pelvic or psoas abscess, much pus is voided at first, but it gradually lessens and disappears.

Symptoms.—The urine is yellowish-white, turbid, with a ropy sediment becoming gelatinous when caustic potash is added; it is often ammoniacal, from decomposition of the urea, by the proteus and staphylococci. Gas may be voided with the urine (pneumaturia), particularly in enterovesical fistula and diabetic cystitis. Alkalinity was formerly thought characteristic of cystitic, and acidity of pyelitic pyuria, but this distinction has been dropped. *Albumin* is found, corresponding in simple pyuria with the number of pus cells. Albuminuria may be absent if there are not over 600–800 pus cells to the c.mm. It is less or may disappear when the urine is filtered. The question often arises whether the albuminuria is simply accidental or whether, for instance, cystitis is combined with contracted kidney; besides consideration of the amount of urine, its specific gravity, cardiovascular findings, washing out the bladder and ureteral catheterization—leukocytes coming from the renal parenchyma in nephritis are mononuclear and those from suppuration are polymorphonuclear; DeLint found that (*a*) when there is one part per mille of albumin and less than 7000 pus cells the albuminuria is renal; and (*b*) it results from the pus alone when there are more than 40,000 pus cells; no conclusion can be drawn if the pus cells number between 7000 and 40,000 per c.mm.

Treatment.—The treatment is that of the fundamental tuberculosis, calculus, pyelitis, cystitis, etc. Phenylis salicylas and hexamethylenamina, āā gr. x, t. i. d., are excellent antiseptics for the urinary tract.

V. Chyluria. — The *tropical form* plugs the lymph vessels, which dilate and rupture in the kidneys, as proved by Mackenzie's autopsy (see page 314). The *non-tropical forms* are not understood and have rarely occurred in Europeans who have never been in the tropics; the rare autopsies have shown no marked renal alteration. Probably there is some communication between the urinary and chylous vessels.

The *urine* resembles chyle, is weakly acid or neutral in reaction, may present a *fatty, creamy surface* and contains 2 to 10 per cent. albumin

(serum albumin, globulin or albumose), cholesterin, lecithin, fat and stearic and palmitic acids. Sugar is seldom found. Microscopic findings are fat globules, leukocytes, sometimes red disks and the filaria parasites, usually in the fibrin clots, which sometimes occasion dysuria. Casts are never found. Chyluria is often intermittent; it occurs especially at night and in 66 per cent. of cases, it disappears in the recumbent posture. In some cases there is free fat in the blood (*lipemia*). Its course is chronic. Recovery is the rule, though death may occur from anemia and marasmus. *Treatment* is unsatisfactory. Potassium picronitrate, gr. $\frac{1}{6}$, t. i. d., is said to accelerate recovery. Prophylaxis regarding drinking water must be considered in the tropics.

VI. Lipuria.—Lipuria or adiposuria is symptomatic of (*a*) *lipemia* or fat in the blood. The clearest instances follow fractures, contusions of the liver or delivery (fat from the pelvic cellular tissue). Fatty foods, acute yellow atrophy, diabetes, disease of the pancreas and various poisonings are possible causes. (*b*) It is symptomatic of *renal lesions* (or fatty epithelial, degenerated tumor or pus cells).

Under the *microscope* small fat globules are seen which stain black with osmic acid or red with the alcanna test. The fat may be saponified by alkalies, emulsified by albumins or dissolved by alcohol, ether, xylol or chloroform. Lipuria is often simulated by bacteria or phosphates or by oil from catheters, suppositories or greasy urinals.

VII. Phosphaturia.—Phosphoric acid is chiefly excreted in combination, two-thirds with sodium and potassium, the alkaline phosphates, and one-third with calcium and magnesium, the earthy phosphates; it is chiefly derived from the food, but some is derived from decomposition of lecithin, nuclein and protagon. It averages two to three grams daily. Amounts over 4 gm. or alteration of the normal relation of the phosphorus to the nitrogen (17 or 20 to 100) are pathological. An excessive deposit which occurs in dyspepsia is not synonymous with an actual increase. Phosphaturia may be observed in neurasthenia, pulmonary tuberculosis, diabetes (largely from the diet), acute yellow atrophy, leukemia and anemia; 8 gm. daily were reported by Teissier and Ralfe, who termed the condition *diabetes phosphaticus*. Earthy phosphates may cloud the last part of the urine, which is mistaken by the laity for "spermatorrhea." The addition of an acid rapidly clears the urine. The *diet*, recommended for this condition, consists of meat, fish, bread, potatoes and apples. Coffee, nux vomica, arsenic, cod-liver oil and ammonium benzoate (gr. x–xxx, t. i. d.) are also given.

VIII. Lithuria.—No subject has occasioned more clinical absurdities than uric acid. It normally bears to urea a ratio of 1 to 70. Futcher has found a ratio of 1 to 300 or 500 (*v.* Gout). It is deposited in larger amounts than normal when the urine is very acid and the mineral salts or pigments are decreased. An increased precipitation as red granules resembling red pepper is far more common than an increased excretion of uric acid. Uric acid combines chiefly with ammonium and sodium, which form *urates* of the "brick-dust" color so frequently seen in febrile urine and urine of high specific gravity or excessive acidity (*sedimentum lateritium*). They are soluble when heated.

IX. Oxaluria.—Oxalates first detected by Donne (1838) in the normal urine amount to less than 10 mg. daily. Larger daily quantities are pathological. Helen Baldwin decided that they are not formed during normal metabolism and are taken in with the food. Oxaluria is observed in gout, hyperacidity, increased intestinal putrefaction, neurasthenia, icterus and diabetes. Oxalates occur as octahedral and dumb-bell crystals, which, according to Klemperer, occur in 50 per cent. of renal calculi. Cantani maintains that oxaluria is associated with *nervous depression, irritability, lumbar pain,* sweats and nervous *dyspepsia,* though the consensus of opinion is against any definite clinical picture or diathesis. Klemperer advised a diet of meat, fat and vegetables (except spinach and cabbage); he excludes milk and eggs and administers magnesium sulphate ℥ss. Nitromuriatic acid ♏ij, with tincture of nux vomica ♏x, is excellent.

X. Indicanuria.—Potassium indoxylsulphate is present in small amounts in normal urine. It is colorless as a rule, but may be voided blue or violet in alkaline urine. When acid or some oxidizing substance is added it decomposes and frees indigo. The action of the pancreatic juice on albumins produces indol, which is absorbed, oxidized in the tissues to indoxyl and unites with potassium sulphate to form potassium indoxylsulphate. Its absence in some pancreatic diseases has been discussed. It is rarely found in urinary calculi. It is increased in cachexiæ in which albuminous substances are decomposed, in intestinal obstruction, carcinoma, suppuration and peritonitis. Boiling the urine and adding nitric acid, as in the ring test for albumin, gives a blue ring; adding chloroform and shaking brings down the violet- or blue-stained chloroform.

XI. Other Anomalies.—*Alkaptonuria* was discovered by Bädecker. It is a crystalline, aromatic body, probably derived from proteid decomposition, whose chief clinical significance is that (*a*) it reduces Fehling's solution, thereby simulating glycosuria, though negative to polarization and fermentation tests; and (*b*) it imparts to the urine a dark brown color, usually apparent only after it has stood or after caustic potash is added (by derivation, urine which "absorbs" an "alkali" freely). Adding a drop of a very dilute solution of ferric chloride causes a dull green color which at once disappears. It is said to be homogentisic or uroleucinic acid. Garrod collected 47 cases, 73 per cent. of which were males; in 32 congenital cases, 19 occurred in 7 families. Virchow (1866) described a case of *ochronosis,* a discoloration of the cartilages and ligaments; Osler described 2 cases associated with alkaptonuria, making 11 in the literature; it may occur with pigmentation of the face, chronic arthritis and deformation of the lumbar vertebræ. Chronic phenol poisoning is a possible cause.

Hydrochinon, from phenol poisoning, causes similar discoloration.

Hematoporphyrinuria, discovered by Hoppe-Seyler, imparts a dark color to the urine. It occurs in tuberculosis, intestinal hemorrhage, sulphonal and lead poisoning, etc. Hematoporphyrin resembles hematin, save that it contains no iron. Barker reports a fatal familial form, with gastric dilatation, fits and polyneuritis.

PYELITIS. PYELONEPHRITIS. SUPPURATIVE NEPHRITIS.

I. Pyelitis.—Pyelitis and pyelonephritis are inflammation of the pelvis, and pelvis and kidney, respectively. They were first fully described by Rayer, though recognized by Hippocrates and Galen.

Etiology.—Pyelitis is *mycotic*. The kidneys usually eliminate bacteria without injury to their structure, but when the general vitality or the local resistance of the kidneys is lowered, inflammation of the pelvis or kidney may result. Various bacteria may be found, including the tubercle bacillus. (*a*) *Foreign bodies*, as calculi, less often cancer or tubercle, and rarely parasites, promote infection. (*b*) Goodhart has shown that 50 per cent. of cases with obstruction (from urethral stricture or prostatic hypertrophy) to the flow of urine die of renal suppuration —*ascending infection*. When the bacteria causing cystitis decompose urea, the ammonia evolved is an additional eroding factor; certain streptococci act in this way, but not the tubercle or colon organisms. *Bacteria* usually ascend from cystitis along the ureters, producing ureteritis, then pyelitis and perhaps a pyelonephritis, but usually reach the kidney along the *lymph vessels*, entering through erosions in the vesical mucosa. (*c*) Infection may be *hematogenous*, as in typhoid, etc. (*d*) Less common factors include *traumatism*, extension by *contiguity* from neighboring inflammations, pyelonephritis (usually an ascending process, in which the kidney is secondarily involved, and only rarely a process descending from the kidney to the pelvis), toxic factors, irritating diuretics, such as turpentine; hemorrhagic diseases; cold, venous stasis and diabetes; arteriosclerosis induces a form of pyelitis attended by hemorrhage and fibrin coagula.

Pathology.—Pyelitis may be solitary or combined with cystitis; unilateral or less often bilateral. Acute catarrhal forms present redness, edema, desquamation, mucopus and ecchymoses. Chronic forms show grayish to brown pigmentation. Croupous or diphtheritic inflammation or even gangrene may develop. Urates and triple phosphates may precipitate. Small retention cysts may form in the pelvis or ureter; the small lymph nodes may enlarge and in rare cases the epithelium may hypertrophy, causing cholesteatoma.

Symptoms.—(*a*) In the ascending type early vesical symptoms antedate and often obscure the pyelitis. (*b*) The symptoms are most clear in primary pyelitis from calculus or hematogenous infection.

Pyuria is the chief symptom. The amount of albumin is generally held to parallel the number of pus cells but often it exceeds the latter (the converse being true in cystitis). The pyuria varies; when the ureter is obstructed, pyuria is suspended and when the obstruction is removed the pus escapes in increased amounts. *Polyuria* and a low specific gravity are usual in chronic cases; the writer observed a case in which 6 quarts daily were voided, simulating diabetes insipidus; polyuria results from decreased absorption of water from the medullary portion or from compensatory hypertrophy of the sound kidney. In acute cases the urine is decreased. The often *cloudy* urine is *usually acid* but frequently alkaline from coincident or causal cystitis or from bacteria decomposing

the urea. Large, caudate, club-like epithelial cells, often overlapping like shingles, are present in moderately severe pyelitis, but they occur, though less frequently, in ureteritis and cystitis. When pyelitis extends to the papillæ or higher (pyelonephritis), cylindrical plugs of pus cells, epithelium, but chiefly of cocci, are seen. Red cells (calculus), triple phosphate or uric acid crystals, fibrin (croupous or diphtheritic pyelitis) and various bacteria (the colon bacillus, in 90 per cent., perhaps the tubercle bacillus) may occur. *Pain* over the kidney is usually dull, but in acute cases may suggest stone. The desire—probably reflex—to urinate frequently, may lead to an erroneous diagnosis of cystitis. *Fever and rigors* are common, especially in severe calculous, colon bacillus or tuberculous types or those invading the kidney substance. Temperature is sometimes sudden and high, at other times intermittent from septic absorption.

Complications.—(*a*) There may be extension to the renal substance (*pyelonephritis*) which may be reduced to a pulpy, inspissated and even calcified mass. (*b*) Prolonged inflammation may lead to fibrosis—the "*ascending contracted kidney.*" (*c*) Plugging of the ureter may occasion distention of the pelvis by retained urine (*hydronephrosis*) or pus (*pyonephrosis*), both of which are described below. (*d*) *Anuria*, described by Morgagni, is occasioned by reflex suppression of the sound kidney or by extensive lesions in both kidneys. (*e*) *Amyloidosis* may develop. (*f*) *Intoxication* may occur by bacterial toxins and renal inadequacy resembling uremia in some respects; Senator described a dyspnea like that of diabetic coma. Treitz and v. Jaksch ascribe intoxication to absorption of ammonia (*ammoniemia*) from decomposed urea. (*g*) Perforation outward causes the *paranephritic abscess* (*v. i.*). In some cases a "spinal paraplegia" is described, probably resulting from an ascending neuritis.

Prognosis.—The prognosis is influenced by the basic simple, tuberculous or calculous infection and by the complications. In acute cases the outlook is generally good. Chronic cases without much necrosis may last many years and may even recover.

Diagnosis.—Much depends on careful analysis of the pyuria (*q. v.*). *Cystitis* may be confused or combined with pyelitis; no absolute reliance can be placed on the reaction, acidity or alkalinity, though pyelitic urine is more often acid. The tailed, clubbed or shingle-like epithelia are more common in pyelitis, though not pathognomonic. Thorough vesical irrigation and gentle compression of the suspected kidney to force pus or cells into the bladder and into the catheter is easier but less certain than cystoscopy and ureteral catheterization. In *nephritis* the leukocytes are mononuclear and casts and cardiovascular changes are decisive.

Treatment.—*Prophylaxis* involves care in the use of catheters or irritating diuretics, the expectant treatment of gonorrhea, etc. *Water, milk* and mineral waters should be used freely to flush out the urinary tract. *Pain* is relieved by hot applications to the kidney; opium is seldom indicated. For *pyuria*, phenylis salicylas and hexamethylenamine, āā gr. x, p. c., are most efficient. Methylene blue, gr. j–ij, t. i. d., may

induce digestive disturbances. Colon vaccines are less efficacious than pelvic irrigation. *Surgical interference* may be necessary when ureteral drainage is incomplete. Nephrotomy is the best operation, nephrectomy being seldom indicated.

II. Suppurative Nephritis.—Its *etiology* is partly that of pyelitis. The microörganisms encountered are the staphylo-, strepto- and pneumococcus, gonococcus, proteus, colon, typhoid and tubercle bacilli, actinomyces, etc. (*a*) The *embolic form* from suppurative processes elsewhere represents one phase of septicopyemia; suppurative foci are chiefly *bilateral* and *cortical;* the glomeruli are filled with microörganisms and, as the foci enlarge, they show as yellowish areas through the cortex; some deposits may also occur in the medulla. Clinically, *pyuria,* hematuria and cylindruria are *seldom* seen and a diagnosis seldom can be made; anuria or oliguria may result from multiple glomerular involvement. (*b*) The *ascending type* is essentially a cystopyelonephritis, the so-called "surgical kidney"; infection and urinary obstruction are its usual antecedents; pyuria, polyuria and albuminuria its usual symptoms; and suppuration in the medullary rays its usual anatomical evidence; very rarely necrotic renal tissue is evacuated. (*c*) Suppuration of the kidney may result from its *invasion by contiguity* from adjacent pus foci as appendicular, periuterine or vertebral foci; much pus may be voided at once, with later gradual cessation of the pyuria.

The diagnosis is uncertain, save when sequestra come away. In 100 nephrotomies, 17 died, 27 recovered and 53 were not benefited (Küster).

PERINEPHRIC ABSCESS.

A distinction is made between perinephritis, inflammation of the fibrous capsule and paranephritis, inflammation (suppuration) of the fatty capsule and surrounding tissue.

Etiology.—(*a*) The most common cause is *suppurative* disease of the *kidney,* as pyelitis, pyelonephritis, pyonephrosis and tuberculosis, from which extension occurs by actual rupture or along the blood or lymph vessels. (*b*) *Extension by contiguity* may occur from vertebral caries, appendicular, hepatic, pelvic or pancreatic suppuration, etc. (*c*) It may result from *trauma* plus infection. (*d*) The so-called *idiopathic* abscess is probably *embolic.* Most cases occur between thirty and forty years of age and 70 per cent. occur in males.

Symptoms.—(*a*) The *onset is acute,* with a rigor and sudden fever in the embolic (so-called primary), but it is *insidious* in the secondary group. (*b*) *Fever* of a continuous or remittent type is usual. (*c*) *Pain and tenderness* are experienced over one kidney, more often the right. Sometimes, with coincident involvement of the kidney, pain shoots along the ureter to the pubes or thigh. To relieve tension the patient lies on the back with the *knee flexed* and the thigh rotated outward. Pain may also arise from simple perinephritis, relieved by nephrotomy. (*d*) *Local findings.* In some patients there are lumbar *edema,* redness and *swelling,* as suppuration usually occurs behind the kidney. The surgeon finds a diffuse phlegmon and less often diffuse serous infiltration

or circumscribed abscess; the pus may be odorless; it may smell of urine when the abscess has arisen from perforation outward of a pyelitic lesion; a fecal odor results from infection (without perforation) from the bowel or appendicular abscess. Necrotic fat and sometimes altered blood are voided. (*e*) The *urine is negative,* unless there is a pyelitic origin or rupture of the abscess into the urinary tract. (*f*) The chief *complication* is *rupture,* which occurs in about a quarter of the cases, twice as often upward into the lung and pleura, as downward, chiefly into the intestine. Burrowing occurs along the ureter toward the pelvis, when the abscess is anterior to the renal fascia, and toward the lumbar region or iliac fossa, when the abscess is behind the fascia. *Amyloidosis* from neglected or obscure cases of paranephritis, pleurisy, subcutaneous emphysema, portal compression, icterus and septicemia may result.

Diagnosis.—Diagnosis depends on local pain, edema or swelling, fever and exploratory aspiration. Psoas abscess, appendicular abscess behind the kidney or rupture downward of empyema are more often etiological factors than separate questions for differentiation.

Treatment.—The only treatment is incision and drainage. In Küster's 230 cases 66 per cent. recovered.

HYDRONEPHROSIS.

Definition.—Hydronephrosis is a sac-like dilatation of the pelvis and calices, with accumulation there of a watery fluid; obstruction to the urinary flow is its cause and atrophy of the renal substance is its result. It was first described by Tulp (1674) and named by Rayer.

Etiology and Pathology.—(*a*) The *congenital form* is most often due to anomalies in the ureters; these include atresia, stenosis, valve formations, twists, compression by peritonitic bands, vestiges of Müller's or Gärtner's ducts, abnormal bloodvessels, imperfect insertion into the bladder or insertion too high into the pelvis, in the lower part of which the urine accumulates; of the latter form the author observed 2 cases. When there are double ureters, one may end blindly in the bladder wall and dilating, may compress its fellow of the same side. Hydronephrosis may occasion dystocia and is commonly associated with other congenital malformations; 65 per cent. of congenital cases are bilateral. (*b*) The *acquired form* may be bilateral or unilateral; its cause may, in rare cases, be in the kidney itself, producing partial hydronephrosis. One or both ureters may be compressed by malignant pelvic growths, as uterine cancer, and this explains the large proportion (67 per cent.) of bilateral hydronephrosis in autopsy figures; in clinical cases 62 per cent. are unilateral. Ureteral obstruction may arise from calculus, tumors, tuberculous detritus, ulceration, cicatrization, floating kidney or rarely clots from trauma or parasites. Large sacs are usually caused by affections of the ureter. Other possible types are obstruction from urethral stricture, enlarged prostate, phimosis and atresia of the hymen.

Cohnheim contended that a slow development with intermissions is more potent in the production of large sacs than is acute obstruction.

The writer has seen G. D. Scott's experiments which prove that acute obstruction rapidly produces marked hydronephrosis.

The *results of urinary accumulation* are (*a*) distention of the pelvis, (*b*) flattening of the papillæ, (*c*) renal atrophy and (*d*) the formation of a fibrous sac.

Symptoms.—The chief finding is a fluctuating renal tumor. (*a*) It is located at first in the *renal region* but later assumes an abdominal importance. (*b*) Its *size varies,* being largest when the ureter is obstructed and in slowly developing cases becoming as large as the head and even simulating ascites; Peter Frank's case contained 60 quarts. (*c*) The chief causes of *intermittent hydronephrosis* are valve formation, stenosis or abnormal insertion of the ureter into the renal pelvis. The amount of urine is (i) normal from hypertrophy of the sound kidney, (ii) decreased in bilateral disease or from increased pressure in the diseased kidney, reflexly lessening the secretion from the sound kidney (Israel); or (iii) when the sac evacuates, suddenly very abundant and then the tumor disappears; this point is often volunteered in the patient's history. (*d*) Its *form,* on bimanual palpation, is *irregularly oval;* in moderately severe cases the kidney itself may be palpated, as well as its irregularly protuberant calices and sometimes the dilated ureter. (*e*) The *ballottement rénal* is a repercussion shock obtained by tapping the tumor with one hand while the other is spread broadly over it. (*f*) Small tumors are *covered by the colon,* which imparts a modified tympanitic note on percussion. Distention of the colon by air brings out tympany over the tumor. Very large sacs push the colon aside. The *ren saccatus* may develop in a floating or horseshoe kidney. (*g*) Its *contents* are albumin, paralbumin, mucus, urea and other urinary constituents, which are usually absorbed only after a long time. Diagnostic puncture is not always safe. (*h*) Sometimes pain may arise from torsion of the ureter. The author noted hematuria and renal colic in hydronephrosis due to an insertion of the ureter high up in the renal pelvis. (*i*) *Complications* include rupture, infection (pyonephrosis) or anuria and uremia if both kidneys are implicated.

Diagnosis.—Diagnosis is easy (*a*) by recognizing the cause, if possible, as stone or tumor; (*b*) by careful bimanual palpation and (*c*) by cystoscopic proof that urine does not flow from one ureter. *Differentiation* concerns especially:

1. Other Renal Tumors.—(*a*) *Pyonephrosis,* a dilatation of the pelvis and calices by purulent fluid, is caused by obstruction plus infection, especially from pyelitis with obstruction. It occasions the same kind of tumor as to location and physical examination, but it is generally smaller and more irregular than hydronephrosis; its walls are thicker and more tender from infection; there is more renal necrosis; systemic septic symptoms prevail; its contents are pus, pulpy detritus, triple phosphates, bacteria, disorganized blood, fat, cholesterin, fetid ammonium compounds and even gas. Puncture in doubtful cases is more dangerous than operation. Its prognosis also is clearly more unfavorable. (*b*) *Cystic kidneys,* (*c*) malignant *renal growths* and (*d*) echinococcus (*v. i.*).

2. Other Abdominal Tumors.—(*a*) *Ovarian tumor* is connected with the uterus and grows upward. Ovarian tumor with a long pedicle may deceive, but it pushes the cecum backward. The dulness is greater, though in large renal sacs the colon is pushed aside. Confusion is likely when hydronephrosis develops in a floating kidney. Exploratory puncture is an unsafe procedure; when made, the fluid from ovarian cysts shows a high specific gravity (1.020); much paralbumin is present, also cylindrical epithelium, colloid or chocolate-colored material and no urea. (*b*) *Ascites and pregnancy* are readily differentiated. (*c*) *Encapsulated peritonitis* may be harder to differentiate, but the urinary condition and sac-like distention are usually decisive. (*d*) *Splenic* and *hepatic* tumors are more flat on percussion, encroach more upon the lungs and are above or anterior to the gas-distended colon. In a renal growth a furrow and tympanitic loop of bowel may lie between it and the liver or spleen.

Prognosis.—The prognosis is generally unfavorable, as the cause is often inaccessible or the process well advanced. The prognosis is better in cases of renal stone than in cancer or in the generally inviable congenital forms. The course is often intermittent and protracted.

Treatment.—Nephrotomy is indicated; nephrectomy is poor surgery since Ayrer proved that some sound renal tissue nearly always remains. The mortality resulting from nephrectomy for hydronephrosis is 5.7 per cent., for pyonephrosis 15.3 per cent. (Schmieden); Rosving reports 0 per cent. and 19 per cent. mortality in his cases of nephrotomy for hydro- and pyonephrosis, respectively; 34 per cent. of his cases recovered completely, 27 per cent. recovered, though with fistula, and secondary operation was necessary in 20 per cent. Fenger advised conservative operation in partial hydronephrosis. Israel and Rosving prefer to cut down on both kidneys before extirpating either one, to tests of the renal function.

RENAL CALCULUS (NEPHROLITHIASIS).

Etiology.—Stone in the kidney or its pelvis results from deposition of the solid constituents of the urine. Probably *bacteria,* especially colon and typhoid bacilli, form the nucleus of stones. An albuminous cement substance is found even in slight urinary deposits or renal sand. Renal stone was described by Hippocrates.

The *predisposing factors* are obscure. (*a*) A *sedentary life* and over-indulgence in food and alcohol are apparently predisposing factors. Galen noted the coincidence of gout and renal calculus. Stones are said to be uncommon in very active persons. (*b*) Most cases occur between *thirty and sixty years* of age; in the extremes of life vesical calculus is more common. (*c*) Seventy-five per cent. of cases are in *males.* Stones are passed more easily, and therefore are possibly less often detected in women. (*d*) Renal calculus occurs most frequently in Asia and there are certain foci, where it is particularly frequent, as central Russia, Holland, Italy, Altenberg, Munich, Hungary, Scotland, west France and southeast England. (*e*) Other possible factors are heredity, especially formation of uric acid and cystin calculi; paraplegia (Müller reported 10 cases of

spinal fracture with calculus formation) and foreign bodies in the renal pelvis, as blood clots from trauma and parasites.

Chemistry and Pathology.—According to their *size*, urinary deposits are spoken of as sand, gravel or stones; sand and gravel consist of uric acid or its salts. They may range from the size of a pea to that of a hazelnut. The heaviest recorded stone weighed thirty-six ounces. Though most often round, oval or cylindrical, they may be dendritic or coral-shaped. A number of them may exist, perhaps 15 to 20; 1000 to 2000 may be found. (*a*) The *uric acid* and urate stone constitutes 66 per cent. of renal stones. It is hard, smooth or slightly uneven, sometimes facetted or lamellated and consists of uric acid, ammonium and sodium urates and a small amount of xanthin. Its broken surface is amorphous. Its color is yellow, red or brown, depending on the amount of urinary pigment. It may contain some oxalates and phosphates. Under the microscope needles are seen. Uratic calculi turn carmine-red on adding nitric acid and ammonia. They dissolve in caustic potash. They are deposited in acid urine, but the exact reason is not known; a decrease of the urinary pigments is considered of great importance; disturbed metabolism or indigestion may occasion uratic deposits, though little is known of the "uric diathesis." (*b*) *Oxalate stones* are next in frequency; they are very hard, warty or prickly (the mulberry calculus); for this reason they cause more pain and hematuria than the uratic stones and are more often removed surgically. They are usually single and gray, but may be dark colored from the bleeding they induce. Lamellation is never observed. Uric acid, calcium carbonate or xanthin may also enter into their composition. No effervescence occurs on adding acetic acid unless carbonates are also present. They dissolve in mineral acids and oxalate of lime crystals gradually precipitate on the addition of ammonia. Oxalates precipitate from acid urine. (*c*) *Phosphatic* calculi are formed in alkaline or neutral urine. They are usually calcium salts, but if the urine is ammoniacal triple phosphates may be formed. Admixture of bacteria, calcium carbonate and xanthin may be noted; phosphatic stones are small, soft and clay-colored. They are infrequent and usually only form a coating to calculi of other composition. (*d*) *Other forms* are rare. Calculi of (i) *calcium carbonate* and more rarely ammonium or magnesium carbonate may occur in the aged; they are white and dissolve with effervescence in mineral acids. (ii) *Cystin* calculi are smooth, yellow and waxy, never exceed the size of a pea, are soluble in acids and alkalies and deposit hexagonal crystals when dissolved in acetic acid and ammonia. Only 60 cases are reported. (iii) *Xanthin* stones occur almost exclusively in children; only 10 cases are recorded; they are smooth, yellow or brown, dissolve in nitric acid and on evaporation leave a residue which turns red on adding caustic potash. (iv) Only 3 *indigo* stones are on record. (v) *Urostealiths* probably result from fatty substances deposited in the bladder; they are 85 per cent. fat and are soft, rubber-like and combustible.

Symptoms.—Stones may be first discovered accidentally at autopsy or, very exceptionally, may pass without symptoms. Gravel or renal sand is more often latent than is stone.

1. Pain.—Pain, the most common symptom, is of two kinds: (*a*) The *dull pain* in the loin, often associated with local tenderness over the kidney; localized pain without any urinary changes, may indicate stone in the medullary substance. Pain may be felt over the opposite kidney. (*b*) *The renal colic,* which arises from incarceration of the stone as it attempts to leave the pelvis; it is often preceded by pain in the loin and may be initiated by exercise or indigestion; Eichhorst's patient had renal colic every time he drank white wine. The pain begins over the kidney and *radiates down the ureter* to the testis, labia, bladder or urethra; the testicle is retracted (in the descent of the testis, its envelopes receive a twig from the first lumbar nerve). It may rarely radiate to the chest, heart, shoulder, epigastrium or to the opposite side. During the colic, the *kidney* is *sensitive;* when it is not so, Israel hesitates to diagnosticate stone. The *pain* is *cutting* and very *severe;* it often occasions reflex nausea and vomiting, sometimes chills, sweats and fever or in children convulsions and rarely collapse or even death in most exceptional cases. If the stone passes, the pain is greatest as it enters the bladder. It lasts, with intermissions, a few hours, rarely days; movement may excite recurrence. The patient lies on his back with the leg drawn up and the abdominal wall rigid. After the attack there may be dull pain (*v. s.*), which may be caused by large, numerous or sharp stones.

2. Urine.—The urine is almost invariably altered. (*a*) *Hematuria* occurs independently of pain and colic, as well as after them. Absence of hematuria after a renal colic argues strongly against stone (Israel). It is often macroscopic and red disks are found with the microscope, except in the rare cases when stone is imbedded in the renal tissue or there is permanent occlusion of the ureter. At the time of the colic the scanty, bloody urine is frequently voided with considerable tenesmus. In exceptional cases early profuse hematuria may result from vascular erosion. (*b*) *Anuria* (suppression of urine) is not common. Legeau collected 30 autopsied cases of calculous anuria; there was stone in one kidney and the opposite kidney (α) contained a calculus in 14 cases, (β) was hypoplastic in 3, (γ) sclerotic in 6, (δ) its ureter was stenosed by calculi in 6 and (ϵ) in one case the opposite kidney was absolutely normal (Morgagni's reflex suppression *via* the splanchnic). The urethra may be plugged with a calculus. The resulting symptoms are called "latent" uremia. However, in 41 cases collected by Herter convulsions were present in but 12 per cent., headache in 14 per cent. and vomiting in 29 per cent.; consciousness is usually preserved and the temperature is low; 11 cases of anuria persisted over four days, 18 cases seven to fourteen days and 7 cases over fourteen days. Russel records recovery after twenty-eight days of anuria. With its relief large quantities of urine are voided. (*c*) *Pyuria* results in protracted cases from pyelitis, secondary to the calculus; infrequently a renal intermittent *fever* is observed, resembling the hepatic intermittent fever of gall-stones. Pyelitis may be catarrhal, suppurative, ulcerative or perforative. (*d*) *Crystals* of pointed uric acid and oxalates are considered important in diagnosis. (*e*) *Stones in the urine* are not frequently found.

3. The X-rays.—Oxalate stones throw a shadow in every instance; uric stones often are invisible. Leonard reports but 3 per cent. of errors; he found calculi in the ureter twice as often as in the renal pelvis. Kümmel insists that 90 per cent. of stones cast a shadow.

4. Physical Findings.—The *physical findings* as tenderness (*v. s.*) are less important than the urinary findings and the colic. Palpation of the stone in the ureter is rarely possible, either from without or by the rectum or vagina; once the writer felt a calculus at McBurney's point. Stones have been felt to crepitate in the renal pelvis (Piorry). Cystoscopic examination and catheterization of the ureters are most valuable methods.

Complications.—Ulcerative *pyelitis* may lead to perforation, pyelonephritis and paranephric abscess. Hydronephrosis results from mechanical occlusion of the ureter and pyonephrosis from obstruction with infection. Injury to one kidney may be compensated by hypertrophy of its fellow. Amyloid degeneration, renal tuberculosis or even cancer may develop. The ureter may rupture from ulceration or tension. Troja instances a pregnant woman in whom abortion occurred after each of fourteen attacks of colic.

Diagnosis.—The diagnosis is based on (*a*) the colic, particularly after exertion, (*b*) hematuria, (*c*) pyelitic pyuria, (*d*) *x*-ray findings and (*e*) less often, the anuria and swelling of the kidney. If the *x*-rays are negative stone is not excluded. A diagnosis of stone is found correct in only 60 per cent. of the cases operated on.

1. Pain and Colic.—Pain and colic may rarely occur in renal embolism; also in passage of blood clots, parasites, echinococcus vesicles, malignant tissue or tuberculous detritus; renal aneurysm and floating kidney. In one case pain, colic and hematuria resulted from high insertion of the ureter. Confusion may result with vertebral disease, the crises of tabes or angioneurotic edema, herpes zoster, gall-stones, appendicitis, diaphragmatic pleurisy or hysteria.

2. Hematuria.—Associated with pain, it may cause errors in diagnosis, usually corrected only at operation; thus acute congestion or capsular fibrosis may be found; Israel describes a unilateral nephritis (*a*) with colic, (*b*) with albumin and without casts; (*c*) with casts and without albumin; (*d*) with hemorrhage, simulating tumor; but (*e*) the bleeding never causes the colic. Hematuria from cancer and tuberculosis does not improve with rest in bed, while calculous hematuria usually does. Hematuria and renal colic may develop in ordinary chronic nephritis from acute renal congestion.

Prognosis.—The course is usually chronic and relapsing, yet the author knows of four physicians who passed calculi without symptoms afterward. Rupture of the ureter, collapse during the colic, pyelitis becoming pyelonephritis, anuria and uremia must be considered in the prognosis. Pyuria is more ominous than hematuria.

Treatment.—1. Prophylaxis.—Little is known regarding the pathogenesis of renal calculus. It is usually stated that regulation of the diet concerns moderation in both proteids and carbohydrates; foods rich in nuclein, acid foods and alcohol are interdicted, water given freely

and exercise insisted upon; vegetables may be allowed. In uric acid calculi, the alkaline waters are valuable, as those of Karlsbad, Vichy, Marienbad, Saratoga, Bedford, Poland and Mountain Valley. Sodium phosphate, gr. x, or bicarbonate, ʒ ss, t. i. d., is indicated. Lithium carbonate, piperazin and glycerin possess no proved solvent powers. In localities where calculi develop very frequently, the drinking water must be used with caution. Stones once formed cannot be dissolved by drugs.

If the oxalates are deposited, treatment is the same as in oxaluria (*q. v.*). In alkaline urines from which phosphates are precipitated, the urine must be made acid by phenylis salicylas, gr. x, t. i. d.; acid waters, as Seltzer or Apollinaris, may be employed; meats should be restricted; hexamethylenamine, gr. x, t. i. d., is excellent in bacteriuria.

2. Colic.—The colic is treated as in gall-stones.

3. Surgical.—Intervention is indicated by (*a*) anuria; (*b*) severe pyelitis; (*c*) septic or aseptic obstruction of the ureter; (*d*) exhausting hematuria and (*e*) repeated attacks of colic. The ideal operation is *pyelotomy;* next is *nephrolithotomy* (nephrotomy), closing the renal wound if there is no infection; operation on the aseptic kidney entails a mortality of 3 per cent., and operation in the presence of infection 10 per cent. (Albarran); in these cases the pelvis should be drained (nephrostomy). Tenny collected 134 cases of *ureteral calculus;* 122 were operated on, with a mortality of 19 per cent. or of 47 per cent. if pyelitis was present; anuria is a serious complication, for 52 per cent. of the operated cases die; without pyelitis or anuria the fatalities were 6 per cent.

TUMORS OF THE KIDNEY.

I. Cancer. — **Etiology.** — The etiology is obscure. It constitutes but 2 per cent. of all cancers and but 1 per cent. of primary cancers in adults (Rubinstein); 62 per cent. occur in males and 32 per cent. in the first ten years of life. Its frequency in children was noted by Bright. Weigert observed 1 case in a newborn child. Cancer in the renal pelvis may result from stone. In the Albarran and Imbert series of 380 renal tumors cancer constituted 50 per cent.

Pathology.—The primary form is usually infiltrating and the secondary form consists of nodules. Medullary cancer is more common than scirrhus, colloid and other forms. It usually begins in the convoluted tubules of the cortex, and is limited by the renal capsule. The average kidney weight in children is $8\frac{1}{2}$ pounds and in adults 10 to 15 pounds (or even 50). The opposite kidney and the sound parts of the affected viscus may show compensatory hypertrophy. Hemorrhages into the neoplastic tissue, invasion of the renal veins, ureter, cava, pancreas, adrenals or intestine and metastases (58 per cent.) in distant organs sometimes develop.

Symptoms.—Some, particularly secondary, cancers are latent.

1. Renal Tumor.—Renal tumor occurs in 97 per cent. of cases. It begins in the renal region, but, resisted by the firm lumbar tissues, it grows forward. Bimanual palpation demonstrates its origin. If large, it may fall forward. It is tender and *firm.* The form of the kidney

is usually preserved, though nodules may be palpated. It attains large dimensions (*v. s.*), compressing and dislocating the liver and spleen upward, crowding the abdominal viscera and even pushing the heart up to the clavicle. On tapping, a repercussion wave is sometimes noted (*ballottement rénal*). Percussion gives a dull note, except so far as the *colon overlies the kidney,* running from left to right, from above downward; on the left side this relation is more clearly distinguished; on inflation of the colon with air its tympanitic note in some degree obliterates the renal dulness, in less than 50 per cent. of large renal tumors; in tumors on the right side the colon is often dislocated laterally or even downward. There is but little if any respiratory excursion. In rare instances the tumor may pulsate and by pressure on the aorta produce a systolic murmur, which, as in Richard Bright's case, may simulate aneurysm. If the tumor grows into the ureter hydronephrosis may augment the swelling.

2. Pain.—Dull pain is an early and frequent symptom, from pressure on the lumbar plexus, which also may cause some sensory disturbance in the legs. Only rarely is the pain colic-like, due to passage of clots or bits of tumor (*v. i.*).

3. Hematuria.—According to Guillet it occurs in 50 per cent. and is the first symptom in 25 per cent. of cases; Israel holds that it occurs in 92 per cent. and is the first manifestation in 70 per cent.; it is less frequent in children (16 per cent.). Sometimes worm-like clots are passed, with great colic; they sometimes measure one-half to one inch; clots in a clear urine are almost pathognomonic (Israel). The cystoscope and ureteral catheterization prove that the hematuria is unilateral. The hemorrhage usually originates in the diseased kidney, rarely from the opposite hyperemic kidney. Albarran asserts there is more albumin than the blood accounts for. Very rarely bits of cancer tissue pass. The urea is increased as in all cancers. Pyuria is no part of renal cancer.

Complications.—*Compression* symptoms include paraplegia dolorosa, constipation, intestinal obstruction, edema from compression of the iliac veins and ascites; varicocele, first noted by Guyon, is due to late compression of the spermatic vein by the tumor or contiguous lymph nodes. Metastases, enlarged glands, rarely perforation into the lumbar muscles, skin, peritoneum or intestine with fatal hemorrhage, uremia, fever from cachexia or terminal infection may be observed. According to Israel, tumor of the renal pelvis causes greater hematuria, more hydronephrosis and therefore greater variations in the size of the tumor; it sometimes grows down the ureter and with the cystoscope is seen to project into the bladder; particles of tumor tissue and cells are more often obtained.

Course and Prognosis.—Death is invariable without operation; it usually results from cachexia, in two years in adults and in eight months in children; 6 cases lived over ten and 1 case seventeen years.

Diagnosis.—The following are important: renal tumor, pain, hematuria and cachexia. Imbert speaks of the complete type (tumor and hematuria), the hematuric type (without tumor, 6 per cent.) and the tumor type (without hematuria, 23 per cent.).

Differentiation.—(*a*) *Liver tumor* encroaches much more upon the diaphragm and is characterized by its respiratory excursion, location of the tumor within the edge of the liver, icterus and ascites. There are no suggestive urinary findings. Bimanual palpation clearly differentiates uncomplicated cases. Between a kidney tumor and the liver a furrow and loop of resonant intestine are frequently found. (*b*) A *splenic tumor* maintains the form of the spleen, lies above the colon (whose distention does not modify the splenic dulness), moves on inspiration, shows the characteristic notches and seldom causes confusion except when it so rotates that only its convexity can be felt. (*c*) An *ovarian* tumor projects upward from the pelvis, with which its connection can be demonstrated, save when it has a long pedicle. It is more central and sustains no characteristic relation with the colon. (*d*) *Retroperitoneal* growths are much more difficult to differentiate, especially enlarged lymph glands and lymphosarcoma. They are generally more central and less movable. Of perirenal retroperitoneal lipoma 50 cases are reported. (*e*) Cystic degeneration (*v. i.*) is usually bilateral and runs a most chronic course. Sarcoma cannot be differentiated. In hydro- or pyonephrosis the tumor varies and presents characteristic urinary changes. Tuberculosis is distinguished by pyuria and tubercle bacilli in the urine.

II. Sarcoma.—Sixty-six per cent. occur under ten years and most in the first two years of life. Sarcoma constitutes 20 per cent. of renal growths. It is slightly more common in girls than in boys and in the left than in the right kidney. Cohnheim's theory of embryonal inclusion is supported by the occasional presence of cartilage, bone, striated and unstriated muscle and even carcinomatous tissue. Sarcoma is of the round-, spindle-cell or melanoid type and may weigh 6000 gm. Secondary sarcomata may develop by metastasis from other organs or by invasion by contiguity.

Its *symptoms* are essentially those of carcinoma, from which an *intra vitam* diagnosis is rarely possible, except when sarcomatous tissue is passed in the urine or excised at operation. Hematuria and cachexia are infrequent; age and glandular involvement are of no diagnostic value. A remarkable overgrowth of the pubic and axillary hair and pigmentation of the skin may be observed. Operated cases live on an average sixteen, and non-operated, eight months.

III. Hypernephroma.—This "struma lipomatodes aberrans renis" of Grawitz develops from aberrant (included) particles of adrenal tissue. Lately their origin is considered Wolffian. Small tumors are not a rare autopsy finding. They constitute 20 per cent. of renal tumors and consist of a scanty vascular stroma, containing fatty cells like those of the adrenal bodies. Hemorrhage, cystic degeneration, a tendency to invade the veins and a large proportion of glycogen are considered characteristic. Fatty cells may be found in the urine. Hematuria occurs in 80 per cent. of cases. Bone metastasis is the first symptom in 16 per cent. but may develop as a late symptom only. Fever is noted in 57 per cent.; it occurs in but 1 to 2 per cent. of other tumors of the kidney (Israel).

IV. Other Tumors.—Fibroma, lipoma, myxoma, angioma, endothelioma and rhabdomyoma are very rare; 33 cases of tumor of the capsule are on record.

Treatment of Renal Tumors.—Symptomatic therapy concerns the pain. Surgical treatment alone is of value. Thirty-three recoveries after radical operation are on record, one patient being alive after eighteen years. Israel in 43 cases reported a mortality of 18.6 per cent., of which two-thirds died of heart paralysis; 19 per cent. enjoyed perfect health for three and a half to fourteen years. Schede in 18 operations obtained 22 per cent. apparent recoveries after four to nine years. Recurrence after two years is very exceptional. Advanced cachexia, large or bilateral tumors, metastases and growth into the cava are contra-indications.

CYSTIC DEGENERATION.

Various renal cysts are observed. (*a*) The most frequent occur in chronic interstitial nephritis from snaring-off of the tubules and glomeruli. (*b*) Parasitic cysts are next in frequency (*v. i.*). (*c*) But three dermoid cysts are on record. (*d*) Cysts of the fatty capsule, retroperitoneal cysts and degenerated lymph glands are very rare. (*e*) *Hydrops renum cysticus, degeneratio renum polycystica* was first separated by Rayer from the above-mentioned varieties and constitutes cystic degeneration, as it is generally understood.

Etiology.—Over 50 per cent. are observed in *men* between *forty and fifty years* of age. Cystic kidneys are observed in later fetal life, at birth and in the adult. Probably they are congenital and remain latent until middle life.

Pathogenesis.—Three main theories obtain; (*a*) that they are retention cysts, (*b*) new formations, *i. e.*, neoplastic (cystadenomata), the most acceptable hypothesis, and (*c*) malformations.

Pathology.—Congenital cases are usually bilateral; Lejars found 1 unilateral case in 67 and Luzzatto 41 in 226 cases. The kidneys may contain innumerable small cysts, when the shape of the kidney is preserved, or it contains larger cysts, when its contour is more irregular. The kidneys may be the size of a child's head and weigh twenty-one pounds. On section the kidney is honey-combed. The cyst walls consist of fibrous tissue. The cysts contain thin, yellow, transparent fluid, less often milky, gelatinous or brownish contents (altered blood). They are multilocular, are lined with cells like the urinary tubules or with flat polygonal cells and contain albumin, oxalate of lime, leucin-like crystals, red disks, epithelial detritus, fat granules and rarely urea. According to Engländer, only 23 unilocular cysts are on record. Between the cysts are found vestiges of renal tubules or glomeruli, which may be vicariously hypertrophied. Cysts may occur in the pelvis, ureter, pancreas and liver (*v. i.*). Other congenital deformities are sometimes found, particularly in the genitals, brain and fingers.

Symptoms.—In congenital forms the cystic kidneys may obstruct delivery; if the infant is born alive death from asphyxia is usual, for the diaphragm is crowded upward. Adult forms generally come to autopsy undiagnosticated or with the diagnosis of nephritis. The symptoms may be classified as follows: (*a*) *Renal cysts* are detected clinically in 20 per cent. of cases only. Their general characteristics are those of

solid kidney tumors, but they are *bilateral* (*v. s.*). Fluctuation seldom is elicited. In 2 cases which the author demonstrated, the individual cysts could be clearly palpated. Echinococcus cysts, hydro- and pyonephrosis, sarcoma and cancer of the kidney are only distinguished by their unilateral occurrence. (*b*) The *urine* may be normal, increased or decreased. The urinary findings are typically like those of interstitial nephritis, *i. e.*, the urine is abundant, with few solids, low specific gravity and traces of albumin. Red disks are found in 20 per cent., sometimes in great numbers. In 2 of the author's cases many red cells appeared each time the kidneys were palpated. Leucin-like bodies may appear in the urine or in exploratory punctures; Beckmann first described these small colloid bodies ("rosettes") with structureless centres and one to five concentric rings and clearly radiating striations. (*c*) *Cardiovascular* changes (left ventricular hypertrophy and sclerosed arteries) are common and heighten the resemblance to contracted kidney. (*d*) *Uremia* is the common cause of death. The clinical course is very chronic and is broken by attacks of periodic uremia; digestive disorders are very frequent. Patients may live fifteen and twenty years after the cysts are found.

Diagnosis.—The diagnosis can be made when there are (*a*) bilateral (perhaps palpably cystic) renal swellings; (*b*) the cardiovascular and urinary findings of contracted kidney, with hematuria and colloid "rosettes," obtained in the urine or by puncture; (*c*) uremia and (*d*) perhaps renal pain like calculus, tenderness, or congenital cysts in the liver (in 28 per cent., Lejars); in a case of Dr. W. F. Dickson renal cysts were found with cystic liver, atrophic heart and pain resembling appendicitis. (See ECHINOCOCCUS OF THE LIVER.)

Treatment.—Operation is permissible only in the exceptional, unilateral cases; about one-third of the operated cases die immediately and in few only are any lasting results obtained. The treatment is that of chronic nephritis. Multiple punctures are advocated.

RENAL PARASITES.

Echinococcus Cysts.—Renal cysts occur in 9 per cent. of echinococcus disease (Neisser's 900 cases). In 970 cases (Vegas and Cranwell) the liver was diseased in 64 per cent., the lungs in 7 per cent., the spleen in 3 per cent. and the kidneys in 2 per cent. Baradulin in 1906 collected 142 renal cases (see page 305).

Symptoms.—(*a*) A *renal tumor* is found in 50 per cent. when the cyst attains large dimensions. Without the exploratory puncture and the urinary findings, it is easily confused with other cysts, hydronephrosis, cancer or tuberculosis. *Fluctuation* and the *hydatid thrill* are not often found (see LIVER ECHINOCOCCUS). The cyst begins in the cortex, usually in either pole of the kidney; it is unilateral in 96 per cent. of cases and is left-sided in 62 per cent. If the cyst calcifies it may be seen by the *x*-rays. The opposite kidney may hypertrophy. (*b*) Exploratory *aspiration* may show (i) fluid or pus containing urinary constituents, cholesterin, succinic acid, much sodium chloride and a reducing sub-

stance; (ii) vesicles, hooklets or particles of lamellated membrane. (*c*) The *urine* may contain vesicles, hooklets, membrane, pus from pyelitis and blood from rupture. As many as 100 vesicles have been passed. (*d*) Passage of vesicles is attended by severe *colic*, fever, chill and vomiting.

Complications.—Seventy-five per cent. *rupture*, chiefly into the renal pelvis; perforation may occur into the intestines, lumbar muscles, lung or pleura; the patient may feel the rupture. The toxic substances, liberated by rupture or puncture, may cause urticaria, severe heart symptoms or even fatal syncope. Adhesions to the liver, spleen and intestine may form. Suppuration or gangrene may develop (pyonephrosis or paranephritis). Obstruction of the ureter causes hydronephrosis; vesicles plugging the lower end of the ureter have been seen with the cystoscope.

Prognosis and Treatment.—The course is chronic, covering even twenty to thirty years. Suppuration may cause pyemia. The cyst may shrink or calcify. Bilateral involvement makes the prognosis unfavorable. Spontaneous recovery by rupture into the urinary tract occurs in 32 per cent. Extra-peritoneal nephrotomy is indicated, as there is seldom more than a single cyst. In 30 nephrotomies by Schede there were no fatalities; 87 per cent. completely recovered and 10 per cent. recovered but had fistulæ. Nephrectomy entails a higher mortality (8 per cent.).

SECTION VI.

DISEASES OF THE BLOOD.

Anemia is a reduction in the blood as a whole or more often a reduction of its important constituents, as the red corpuscles, their number, hemoglobin or albumin. Every anemia has a cause, yet in our present ignorance we adhere to the older division of anemia into primary and secondary forms. *Primary anemia* includes chlorosis and pernicious progressive anemia.

CHLOROSIS.

Definition.—Chlorosis is a disease of hemogenesis, occurring in women, chiefly in the developmental years, in which the cardinal symptom is anemia; the red cells are reduced but the hemoglobin is disproportionately reduced; chlorosis responds to therapy by iron. Chlorosis is a disease, while anemia is but a symptom.

Etiology.—Its etiology is uncertain. (*a*) *All cases occur in women,* especially in girls who menstruate before the pubes and mammæ develop (Niemeyer). It is more common in blondes than brunettes. (*b*) Chlorosis occurs largely between the *fourteenth* and *seventeenth years,* rarely after the twenty-fourth year. (*c*) *Heredity,* chlorosis, tuberculosis and hysteria are not infrequently associated in the family history. (*d*) *Malnutrition,* lack of hygiene, working in dark quarters, insufficient or excessive exercise, wearing of corsets and emotional or sexual factors are indirect causes. Various theories are advanced as to the nature of the disease, as *adynamia of the blood-making organs* at puberty, insufficient absorption of iron, auto-intoxication from the internal genitalia and intestines.

General Symptoms.—Among the early frequent symptoms are *rapid tiring, dyspnea* and *palpitation. Pallor* is the earliest and most constant sign, the skin being of a *yellowish-green hue* (chlorosis, as named by Varandel in 1670). Sometimes the face is flushed, probably from peripheral vasomotor irritability (*chlorosis rubra*); hence the mucous membranes should be examined also. The eyes are often brilliant. *Lack* of *mental and muscular energy* is noted, associated with *headache,* which may be dull or sharp, sometimes migraine-like or increased at the time of menstruation, spots before the eyes, tinnitus, vertigo and syncope, especially from standing, travel and exposure to the sun. *Menstrual disturbances* are frequent, the flow being often decreased in younger subjects. Digestion is very often disturbed. The hands are cold, the skin dry and the peripheral circulation sluggish. Circulatory symptoms

appear, as the *bruit de diable* in the neck veins, *anemic heart murmurs* and rapid pulse.

Special Symptoms.—1. The Blood.—The blood is fluid and its coagulability is increased. The red blood cells are decreased (*oligocythemia*) and average *3,000,000* to *4,000,000;* as few as 900,000 have been observed. In rare cases they are not decreased. Formation of rouleaux is poorly marked. The red cells appear *pale* and their average *size is reduced.* Irregularity in contour (*poikilocytosis*), megalocytosis and microcytosis are rare; normo- and megaloblasts are found in the severest cases, or in "blood crises." More suggestive than the oligocythemia is the reduction of hemoglobin (*oligochromemia*), which is reduced more than the red cells (Duncan, 1867); the hemoglobin averages 30 to 40 per cent. This *reduction in the color index* to one-half or less always suggests chlorosis, and is less conspicuous in relapses and in older individuals. The *white cells* are essentially *normal,* therein differing from many other forms of anemia; occasionally lymphocytosis and eosinophilia occur. The iron and nitrogen of the blood and its specific gravity are reduced proportionately to the decrease in hemoglobin.

2. The Vascular System.—(*a*) Rokitansky and Virchow observed hypoplasia of the aorta and vessels, which may coexist with hypoplasia of the genitalia, muscles and bones, but cannot be regarded as causative. (*b*) *Palpitation* is almost constant on exertion; the apex-beat is strong to compensate for the poverty in hemoglobin. (*c*) *Heart murmurs* occur in two-thirds of the cases; they are functional and caused by insufficient tension of the mitral valve, relative mitral insufficiency, weakened heart muscle (producing a muscular murmur in place of the tone) or vibration of the pulmonary artery. The murmurs are largely systolic; undoubted diastolic venous bruits have been heard. (*d*) *Dilatation of the heart* may occur from anemia or myocardial degeneration, though usually it is not real but apparent, retraction of the lungs away from the heart, because of insufficient lung expansion being seen by the *x*-rays. (*e*) The *pulse* may be rapid, throbbing or excitable. Dicrotism and capillary pulsation are frequent and double tones have been noted in the crural vessels from decreased tension and diastolic recoil. (*f*) A venous *bruit de diable* in the neck occurs more frequently than in other anemias (in over 50 per cent. of cases and in two-thirds of the cases over the right jugular vein, because it is more vertical). It is increased by turning the head and deep inspiration. It is referred to change in the tonus of the vessel, waves in the veins above their valves or change of the blood plasma. (*g*) Arterial murmurs may be heard, as over the subclavian artery; pulsation in the peripheral veins, angiospasm and erythromelalgia have been observed. (*h*) *Edema* is seen in 12 per cent. of cases and is usually slight, occurring chiefly about the ankles and eyes.

3. Respiratory Tract.—Dyspnea is common, explained as a hematogenous respiratory insufficiency. The respiration-rate is 26 to 30. Sometimes a paroxysmal cough develops.

4. Digestive Tract.—The digestive tract is involved in one-third of the cases. (*a*) *Pain* is frequent near the ensiform with tenderness, especially in nervous individuals. Vomiting and perversity of appetite

are occasional. (*b*) *Dyspepsia* may be purely nervous. Exceptionally the stomach is dilated and atonic. (*c*) The hydrochloric acid is often increased, probably from nervous irritability. (*d*) Constipation is no more frequent than in the average case; intestinal fermentation is not the cause of chlorosis.

5. Sexual Sphere.—Menstruation is decreased or absent in 80 per cent.; menstrual disturbance, the uterus infantilis or small ovaries are neither the cause nor result of chlorosis, but result from some common factor. Conception is unlikely to occur. Chlorotics are first treated by the gynecologist for leucorrhea in 22 per cent. of cases.

6. Special Senses.—The usual symptoms of anemia, as spots before the eyes and weakness of accommodation, are observed. The retinal vessels may be transparent or pulsating; retinitis and neuroretinitis or "choked disk" exceptionally simulate brain tumor. Retinal hemorrhage is rare.

7. Skin.—The skin is usually anemic; angiospasm, coldness and paresthesia occur. In chlorosis rubra the cheeks are flushed. Seborrhea and acne are very common. Sweats, urticaria, eczema, purpura, chloasma and trophic disturbance of the hair and nails may occur.

8. Nervous System.—Chlorosis often brings out a latent *neurosis*—especially hysteria—and psychosis.

9. Metabolism.—Metabolism is not essentially disturbed. Usually the body weight is maintained, less from decreased oxygenation than from inactivity. The *urine* is clear, of low specific gravity, often alkaline with decrease of urea and uric acid and sometimes albuminuria. Leube found fever frequently in mild chlorosis. When present, we may suspect "tuberculous pseudochlorosis."

Complications.—Chlorosis may favor *acute infections* and render their course severe. Venous *thrombosis* complicates 2 per cent. of cases and is probably inflammatory. In Quensted's series 67 per cent. involved the veins of the leg and 25 per cent. the cerebral sinuses; pulmonary embolism occurred in 19 per cent. Virchow emphasized the frequency of septic endocarditis. Rheumatic endocarditis with organic valvular lesion is frequent, as also are ulcer of the stomach, tuberculosis and Basedow's disease. The *spleen* is enlarged in 16 per cent. and often the thyroid gland. *Hemorrhages* are not infrequent from the nose, stomach or intestine, although less than in severe secondary or pernicious anemia.

Clinical Course and Prognosis.—Chlorosis develops abruptly and runs a *short course* of two to four months. The prognosis is generally good. The tendency to *recurrence* is great, especially in unmarried or childless women; the blood may not return absolutely to normal, shown by persistent pallor or irregular menstruation. *Acute chlorosis* is observed in young girls after their first menstruation; it is attended by severe headache, nervous symptoms, weakness and optic neuritis or "choked disk," which may be confused with brain tumor. The *periodic type* occurs in the higher classes and in those who acquire the disease in their seventeenth to nineteenth year. The disease may last for years with *chronic* chlorotic blood, menstrual, dyspeptic, muscular and nervous symptoms, with alternate exacerbation and amelioration; there is poor development of the bones, muscles,

genitalia, mammæ and adipose tissue; marriage, pregnancy and iron often benefit these cases, but they may last until the menopause.

Diagnosis.—The diagnosis is made by the grouping of symptoms, positive and negative, for no single symptom is pathognomonic. The obtrusive symptom is *anemia*, especially when there is disproportionate *reduction in the hemoglobin*, when the color index is less than one and the white cells are normal. Rhomberg described exceptional cases in which the blood was normal. The age, *rapid development, periodic manifestation* and successful *treatment* with iron are important features. *Secondary anemia* is most difficult to differentiate and is usually attended by some leukocytosis (*v.* page 677). Hemorrhage from latent ulceration of the stomach, early pregnancy in unmarried girls, the anemia of heart disease, etc., must be considered. *Splenic anemia*, especially in children, requires differentiation; cases of chlorosis with enlarged spleen are a transitional form between chlorosis and splenic anemia proper. *Malignant disease* and incipient *tuberculosis* are important.

Treatment.—1. Prophylaxis.—General hygiene and development of adipose tissue to a reasonable extent are prophylactic. More albumin should be given to growing children than to adults, together with fruit and vegetables, for their iron, and butter, cream and oils, rather than animal fats. Physical culture and trips to the country are valuable. Children at this age are confined too much to the school-room; they should be kept children as long as possible. In society or in the struggle for existence young girls too often overtax their strength.

2. Specific Therapy.—Iron exists in the hemoglobin of the blood cells as an oxygen carrier and is a food as well as a medicine; it is found in blood, meat, oatmeal, peas, spinach and beans.

Physiological Action of Iron.—(*a*) The *nervous* tonus is increased; an excess produces frontal headache from constipation; some persons exhibit a peculiar intolerance of iron, as shown by headache, vertigo and palpitation. (*b*) It increases the number of red disks and the hemoglobin. (*c*) Iron stains the teeth because of the acid it contains. Small doses stimulate digestion, improving, by their astringent action, the tone of the tissues. Hydrogen is generated and from it sulphuretted hydrogen, which causes eructations. Small doses constipate but large doses relax the bowels; diarrhea sometimes follows small doses. Sulphide of iron darkens the stools. (*d*) The amount of urine is increased. Iron is found in the urine in very small quantities. The kidneys may be irritated by very large doses. (*e*) It is absorbed from the stomach and intestines as an albuminate, reaches the blood and is deposited first in the spleen and then in the liver, by which, and the intestines chiefly and kidneys, it is eliminated.

Administration.—Soluble would seem to be better than insoluble preparations, but Quevenne has shown that they are precipitated by the gastric juice. Headache is obviated by using laxatives, or ferrous salts, which derange digestion less than ferric salts, and can be reduced; chlorotics cannot reduce the ferric salts. Iron is said to be best absorbed when some gastric catarrh exists, hence Brunton gives large doses at first to improve absorption. There are only 38 to 45 grains of iron in the body; the ordinary

daily diet contains but one-sixth of a grain of iron, but while sufficient as food, does not stimulate the blood-making centres. The doses should not be too small. Statements of the patient that she has previously taken iron should not be regarded, for its administration may have been irregular. In the first week the dose should be gradually increased; for three weeks the maximum dose should be given and for the last two weeks it should be gradually decreased. If iron is not beneficial it should not be repeated but other methods tried. Relapses are less easy to treat than the first attack. Idiosyncrasy, as gastro-intestinal disturbance, is only a relative contra-indication. The administration should then be subcutaneous. At mineral springs hygiene is more important than iron; the water should be taken on an empty stomach, for the gas promotes absorption of iron. When bottled it is precipitated as a carbonate.

Preparations.—Ferrum reductum (reduced iron) is iron gray (if it is black it is impure) and tasteless; it is least astringent, but may cause eructation; gr. j. (See formula on page 377.) *Pilula ferri carbonatis*, a ferrous salt nearly free from astringency; grs. ij–v. *Ferri sulphas* (green vitriol), a ferrous salt which is very astringent, whence its use in chronic diarrhea. It stimulates digestion, but in large quantities it causes vomiting or diarrhea; gr. ij may be given with 1 dram of magnesium sulphate p. c.

℞—Ferri sulphatis exsiccati,
Potassii carbonatis . . . . . āā ʒij
Syrupi . . . . . q. s.
M. et fac pilulas no. 1.
S.—One to three pills after meals (Blaud's pills).

Liquor ferri et ammonii acetatis (*Basham's mixture*) is diuretic and excellent for chlorosis in the obese, ʒj–iv given after meals, well diluted. *Tinctura ferri chloridi* is the chloride of iron plus hydrochloric acid and alcohol, which develop hydrochloric ether. It is yellowish-red, astringent, somewhat corrosive and diuretic, increasing the amount of urine and frequency of micturition. It is used to best advantage when the tongue is white, flabby and indented. In weak, anemic girls, with vomiting, neuralgia and epigastric pains, full doses should be given; when poorly tolerated, one drop in a full glass of water is more beneficial than chalybeate waters. Iron and iodine given separately are usually more beneficial, but the *syrupus ferri iodidi* may be given in doses of 30 to 40 drops in adults and of 5 to 10 in children. *Ferri citras* in doses of gr. v, is efficacious in delicate stomachs; for hypodermic use it is painful; ferri arsenas, gr. $\frac{1}{12}$, or ferri cacodylas, gr. $\frac{1}{4}$ to j, is better. Most *organic* irons are useless. *Arsenic* alone seldom increases the red cells or hemoglobin; ferri phosphas solubilis, gr. v, is almost free from astringency and taste. Arsenic is given as in pernicious anemia (*q. v.*) uninterruptedly, with gradually increasing doses, sustained doses and then decreasing doses for a period of four to six weeks.

High altitude increases the red cells and hemoglobin.

3. Dietetic Treatment.—The amount of *albumin* should be increased and the heavy English breakfast is indicated. Equal parts of milk and cream should be given but with care lest the appetite be spoiled, too much

fat be deposited and the stomach suffer atony. *Alcohol* is indicated in thin rather than in obese individuals. Patients should rest before and after eating and retire early. In thin subjects the diet should be concentrated, consisting of albumin, vegetables, fats, alcohol, and milk at breakfast and at bed time, but without increase of the carbohydrates. Iron produces better results in such cases after limiting the amount of water to about one quart. Sweats may produce the same results. Gastric disturbance disappears after administration of iron, and meals at two-hour intervals—fluid and solid meals being alternated. Hydrochloric acid, strychnine, belladonna, exercise in convalescence (during chlorosis the patient should rest in bed) and cool rubbings early in the morning are advantageous; cold baths produce chills, insomnia and digestive disturbances. Marriage may help mild cases, but is injurious in the severe type.

PERNICIOUS ANEMIA.

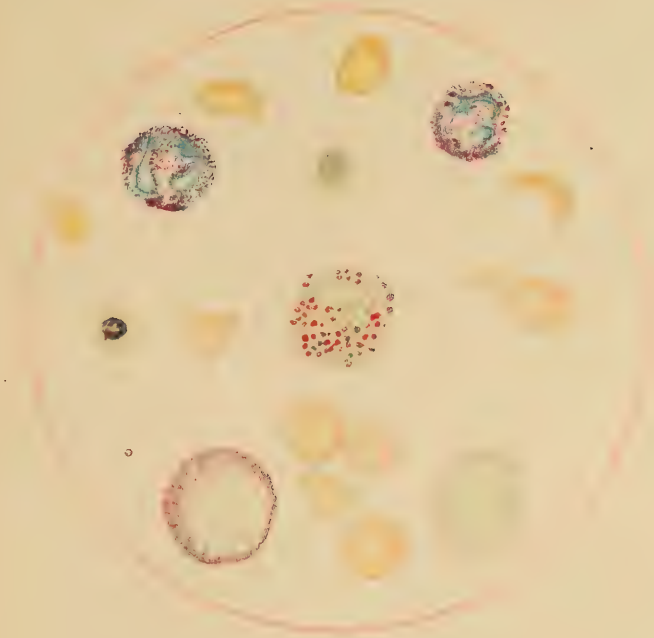
Definition.—A primary anemia with progressive diminution of the red cells and reduction in the hemoglobin; due to hemolysis and defective hemogenesis; characterized by pallor, adynamia, edema and cardiac symptoms and resulting in death from vomiting, diarrhea, hemorrhages and fever. It was described by Combe (1822) and Andral (1826); Addison (1855) gave the best early description and it is sometimes called Addison's anemia. Since Ehrlich drew the blood picture, little has been added to our knowledge of the disease.

Etiology.—The disease occurs in 0.2 per cent. of cases, especially in the *fourth and fifth decades* of life; six-sevenths of the cases occur after the thirty-fifth year (Cabot). Baginsky could collect but 16 cases in children. Sixty per cent. occur in males. The ultimate cause is to be sought in some *hemolysin,* as yet unknown. Although called a primary or essential anemia, some undoubted secondary cases may be included under this head because the symptom-complex is the same. Bothriocephalus and ankylostoma anemias present identical blood findings, symptoms and pathology. Talquist found a lipoid substance in the alimentary mucosa and in tape-worms. Pregnancy, the puerperium, syphilis, typhoid, malaria, and diarrhea, vomiting and oral or gastro-intestinal sepsis have been considered factors.

Symptoms.—1. The Blood Changes (Sorenson, 1874).—The amount is decreased, several punctures often being necessary to obtain enough for the blood count; it is often watery. The specific gravity is low, even 1.027. There is an increase in the albumin in the individual red cell (hyperalbuminemia rubra, von Jaksch). The serum does not contain free hemoglobin. If injected into normal individuals the serum has a hemolytic action. In contrast to chlorosis and secondary anemias, the *coagulability* of the blood is *decreased* and the blood may remain fluid for some time after death. Dropped on filter paper, there is a white zone outside the blood stain (Talquist). The *hemoglobin* is rarely more than 50 per cent. and may be reduced to 10 per cent. In 90 per cent. of cases and in contrast to other anemias, the *hemoglobin per corpuscle* is proportionately *increased, i. e.,* the color index is more than 1 (even 2.25).

PLATE XVII

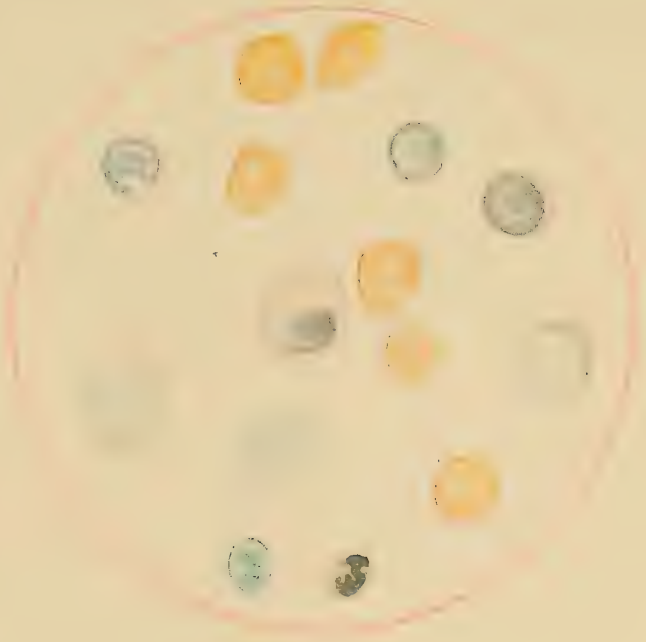
FIG. 1



Myelogenous Leukemia

1, myelocyte; 2, eosinophilic myelocyte; 3, 3, eosinophilic morphinuclear cells; 4, small, and 5, large lymphocytes; 6, nucleated red cell.

FIG. 2



Lymphemia

Large and small lymphocytes with various intensity in stain of nuclear chromatin

FIG. 3

Pernicious Anemia

1, megalocyte; 2, microcyte; 3, 4, 5, malformed erythrocytes; 6, 6, normoblasts; 7, 7, 7, megaloblasts.

Laache's explanation is that certain red cells, especially giant forms, contain a *large compensatory individual amount* of hemoglobin, which is also true of the microcytes, but less so of normal-sized red cells. Free hemoglobin, acting on the white cells, possibly produces a fibrin ferment, which causes fever, hemorrhage and capillary embolism. The *red blood corpuscles* are greatly reduced in number, even to 100,000, the average being 1,200,000. Patients often die when this figure is reached. *Poikilocytosis* is marked, the irregularity in form and size of the corpuscles being great, as Litten's horseshoe or pessary-shaped corpuscles. In 90 per cent. the *red corpuscle* is *above the average size, i. e.*, 11, even 18μ, instead of 5 to 8μ, the highest normal measurement. Polychromatism is frequent. *Megalocytes* may be observed in numbers, with less marked or absent umbilication; they probably indicate megaloblastic degeneration of the bone-marrow. (See Plates XVII and XVIII.) *Megaloblasts* are rarely found in large numbers; in rare cases they may be abundant, as just before death, whence their generally ominous significance; they may be absent for months or weeks at a time. *Normoblasts* occur in nearly all cases, especially in the "*blood crises*" but are not numerous; they are immature cells and indicate activity of the blood-forming organs. Microblasts are frequently absent; their teleological significance lies in the increase of the respiratory surface of cells thus afforded. Cell divisions are found. Granular deposits (staining with basophilic dyes) in the protoplasm are noted; according to Ehrlich, they are not due to polychromatophile degeneration but to nuclear degeneration. The cells may stain only at their margins. Decreased formation of rouleaux is observed as well as ameboid movement of the red cells. The *white corpuscles are decreased* absolutely, the lowest record being 1500; the polymorphonuclears are decreased. Leukocytosis is a distinct complication, is often ominous and indicates inflammation or suppuration. The lymphocytes are relatively increased, even to 62 per cent. of the total white cells. Marked decrease of the eosinophiles is an unfavorable sign. Pernicious anemia due to parasites shows increase in the eosinophiles to 6, 36 and even 50 per cent.

2. Subjective Symptoms.—The most marked are weakness, muscular adynamia, vertigo, palpitation and dyspnea.

3. Skin.—The skin presents a *peculiar pallor*, often of a somewhat characteristic lemon tint. Slight icterus is not infrequent. The skin is usually dry. *Edema is invariable*, especially about the ankles and eyelids. It is obstinate and may produce pronounced general anasarca; it is due to degenerative vascular permeability. Hemorrhages, usually punctate, are frequent in the skin and mucosæ. Large hemorrhages occur in 25 per cent. of cases. Pigmentation is sometimes as marked as in Addison's disease. The author observed herpes zoster and crural herpes.

4. Metabolism.—The *nutrition is maintained* in 61 per cent. of cases because tissue waste is lessened and water is retained in the tissues; in 39 per cent. the weight and strength finally decline with persistent vomiting and diarrhea (Cabot). In the *urine* indican, putrescin, cadaverin, leucin and sometimes tyrosin, acetone and albumose are found. *Albuminuria*, due to tubular degeneration, is frequent. Urobilin is frequently increased and indicates hemolysis. The chlorides are decreased.

The iron in the urine and uric acid are sometimes increased and the phosphates are greatly increased. Irregular fever, present in 80 per cent. of cases, bears no constant relation to the severity of the disease. It may rise considerably; the temperature may fall below normal in the antemortem coma. It is probably due to a ferment from the destroyed red cells or from the leukocytes (*v.* page 673).

5. The Circulation.—*Palpitation, precordial anxiety* and *dyspnea* are observed, sometimes only on exertion. The heart impulse is fallaciously strong to the palpating hand. Its outlines are usually normal, save that dilatation of the right heart may develop. Almost always a blowing systolic murmur is heard, usually with a clear diastolic tone; the murmur is rarely presystolic or diastolic. No parallelism exists between the degree of anemia and the loudness of the murmur. Leube reports cases of chlorosis and pernicious anemia in which there was relative tricuspid insufficiency with a positive venous pulse but no cyanosis. A venous hum in the neck, carotid pulsation and thrills, a loud, clear systolic tone and murmurs or double tones over the peripheral arteries are often present. The *pulse* is collapsing, decreased in tension and *increased* in rate, usually between 90 and 120. *Anatomically,* vestiges of hemorrhage, atrophy and the *fatty,* "tabby-cat heart" are almost constant. Degeneration in the smaller vessels is noted, especially in the brain.

6. Digestive Tract.—Anorexia, distaste especially for meats, pain and sensitiveness throughout the alimentary tract are noted. The tongue and gums often exhibit circumscribed hemorrhages. Pain in the mouth (50 per cent.) and esophagus is due to the anemia or administration of arsenic. The gums may ulcerate; erythema bullosum and other eruptions may occur. *Vomiting* is frequent and often incoercible; the vomitus consists of food, greenish mucus and sometimes traces of blood. The writer has observed profuse hematemesis and cyclic vomiting as the first symptoms. *Crises* of abdominal pain occur in 60 per cent., and the writer observed simulation of gall-stones by the pain and subicterus. Epigastric pulsation, abdominal distention and achylia are observed. The author saw expansile abdominal pulsation, diagnosticated as aneurysm. The glands in the gastro-intestinal tract may atrophy, which probably toxemic condition is coördinate with and not causal of pernicious anemia. The mesenteric glands are not infrequently enlarged. *Paroxysmal diarrhea* occurs in 50 per cent., constipation in 35 per cent. and an alternation of these conditions in 15 per cent. The stools are not characteristic; parasites or their eggs may be noted. Leucin and tyrosin crystals are frequently caused by diarrhea, but are not characteristic of the disease. Fermentation, indicanuria, edema, hemorrhage and follicular swelling in the bowel are frequently observed. Some ascites is found in the terminal stage. A deposit of iron (siderosis) in the liver, in the outer and middle zones of its lobules, especially in acute types, in the spleen, bone-marrow, kidneys and lymphatic glands is usual and results from hemolysis; it is very unusual in other anemias. The liver is slightly swollen and tender in one-third of the cases.

7. Blood-making Organs.—The *lymph glands* are not usually enlarged. In the prevertebral hemolymph glands Warthin found their sinuses dilated and evidences of hemolysis, shown by increased phago-

cytosis and sometimes hyperplasia. The *bones are tender,* especially the sternum and tibiæ, and sometimes painful; the fatty marrow may become lymphoid (leukoblastic degeneration), as in other anemias, cachexia or fevers; in pernicious anemia the red cells of the marrow are abnormally large, abnormally dark from increase of the hemoglobin and are oval rather than round; the megaloblastic degeneration develops at the expense of the other elements in the bone-marrow. These probably secondary bone changes were first described by Wood, Pepper and Tyson (1875). The *spleen* is not enlarged save from accidental causes; in it are found phagocytosis and evidences of hemolysis.

8. The Nervous System.—The psychical functions are depressed, the memory is weak and psychoses, delirium, hallucinations, mania and insomnia may develop temporarily or antecedent to the terminal coma. The pupil reflexes sometimes disappear. Paresis, apoplectiform attacks, cramps, convulsions, paresthesia, aphasia or vertigo may develop. The *cord* is more frequently involved (in 84 per cent. of cases) than the brain. The weakness may amount to paresis, not due simply to muscular adynamia but to spinal changes. The picture may resemble tabes as concerns the pupils, patellar reflexes, ataxia, bladder and rectal changes, crises, pains, anesthesia and paresthesia; or, less often, the symptoms of spastic spinal paralysis prevail, as increased reflexes and spastic paresis. In one group of cases the nervous symptoms are slight, and in a second they are so conspicuous as to overshadow the anemia itself. Early paresthesia occurs in all cases, usually in the hands and feet, sometimes in the mouth; sometimes there is hyperesthesia or disturbance of the temperature sense. The sexual function is decreased in males and amenorrhea is regular in women. There is a sclerosis of the posterior (which rarely wholly escape) and lateral columns, most marked in the lower cervical and upper dorsal segments, oftener diffuse than focal, and involving chiefly the white substance, but not the entire neuron. Small foci of degeneration with secondary connective-tissue growth occur about the bloodvessels, which foci may fuse into small plaques with secondary degeneration, whence the symptom-complex of a system disease.

The discrepancy between the anatomical and clinical findings is remarkable, *i. e.*, there may be many symptoms with few autopsy findings, and conversely; probably the nervous symptoms are less sequences of anemia than coördinate toxic manifestations. The peripheral nerves present few symptoms.

In *the eyes* the general symptoms of anemia occur; hemorrhages in the retina are found by Hasse in 94 per cent. of cases but not in other anemias requiring differentiation. Occurring on a pale background, with tortuous veins and a strong light reflex from the vessels, the punctate hemorrhages form a striking picture (see Plate VI, Fig. 9). Labyrinthine hemorrhage with vertigo is recorded.

Clinical Course and Prognosis.—The initial pallor and weakness increase insidiously and later dyspnea, weak heart action, edema, complete anorexia, languor, spinal and gastro-intestinal disturbance and finally capillary hemorrhages and fever develop; the vomiting and diarrhea may resemble acute poisoning. In other instances remissions may occur

with treatment or independently of it, perhaps after violent gastro-enteric manifestations. In Ehrlich's case, the red cells in seventeen days rose from 1,340,000 to 4,115,000. The disease with remissions may cover a period of years, the longest duration being seventeen. Death usually occurs within half a year. The writer observed recovery for fourteen years after a count of 400,000.

The prognosis is usually poor when many megaloblasts are present or when the color index is over one.

Cabot recognizes *two forms:*

THE SEVERE, RAPIDLY FATAL TYPE—AND THE LESS SEVERE, SLOW TYPE.

(*a*)	Extreme progressive anemia.	Remissions.
(*b*)	Color index high.	Normal or low.
(*c*)	Red cells increased in size.	Normal.
(*d*)	Degenerative changes.	None.
(*e*)	Many megaloblasts.	Many normoblasts, few megaloblasts.
(*f*)	Lymphocytosis.	Normal ratio of the various white cells.

Emerson distinguishes three groups, according to the blood count: (i) With 1,000,000 reds, weakness and shortness of breath on exertion; (ii) with 1,000,000 to 2,000,000 reds, alimentary symptoms; and (iii) in higher counts, various, perhaps severe, but particularly nervous, symptoms.

Death usually occurs in coma. There are only 6 recoveries, according to Cabot, yet he found 27 cases which lived eight or more years. The mortality in the parasitic form is 17 per cent.

Diagnosis.—No single finding is absolutely characteristic, though the pallor, apathy, adynamia, edema, weak heart, retinal hemorrhages, maintained nutrition and the blood findings are highly suggestive. The yellow color is very often mistaken for icterus. Ewing states no importance should be attached to the increased diameter of the red cells unless 33 per cent. of the red disks show it.

DIFFERENTIATION.—(*a*) From *metastatic bone tumors,* in which the white cells are increased (the polymorphonuclear, neutrophile and eosinophile marrow cells); (*b*) from *parasitic anemia,* for which the stools should be examined for parasites and their eggs; (*c*) *fever,* if dominant, may suggest typhoid, meningitis or acute endocarditis; (*d*) from *latent carcinoma;* the gradual cachexia, anorexia, nausea, vomiting, hematemesis and absence of hydrochloric acid may suggest gastric carcinoma, but the blood examination determines the point; (*e*) *spinal disease,* Addison's disease and hemorrhagic pleuritis are usually excluded with ease; and (*f*) *from other anemias.* The table on page 677 is constructed after Cabot's table.

Aplastic anemia, generally regarded as a type of pernicious anemia, is characterized by its low blood count and reduced hemoglobin. It was so named by Ehrlich because the bone-marrow was aplastic; therefore in the blood there are no evidences of regeneration, such as nucleated red cells, the red cells are small and the color index is low. The white cells may number only 200 per c.mm. and 80 to 90 per cent. of them are lymphocytes. Two-thirds of the cases are women. There may be hemorrhages, especially purpura, fever or hyperplasia of the spleen and lymph glands. The outcome is rapidly fatal, 80 per cent. dying within three months.

Fig. I

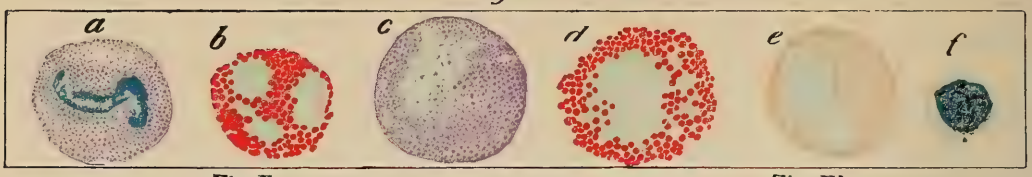


Fig II.

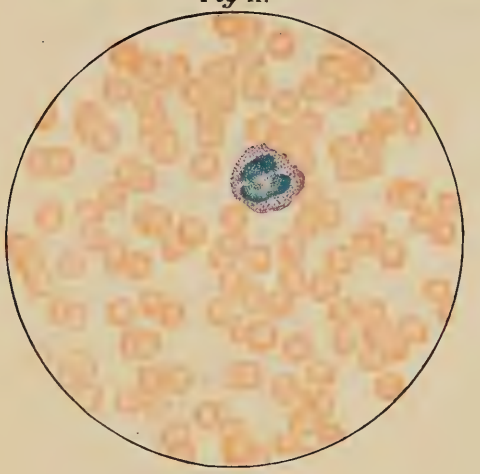


Fig III

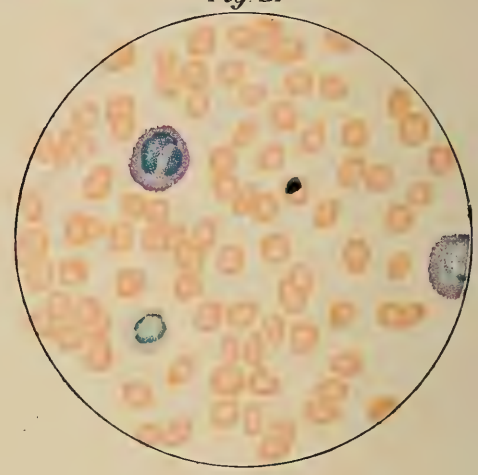


Fig IV.

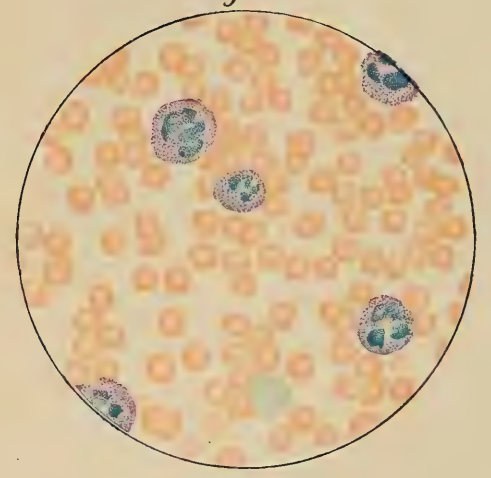


Fig. V.

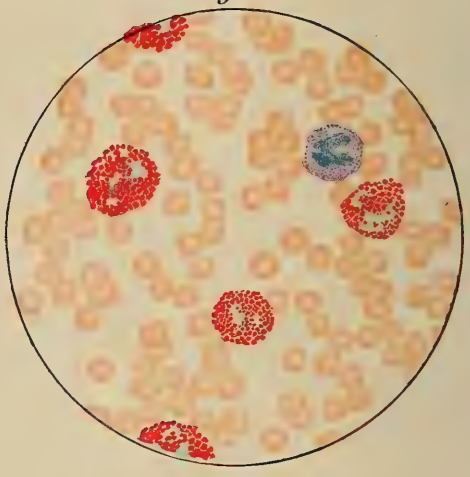


Fig. VI.

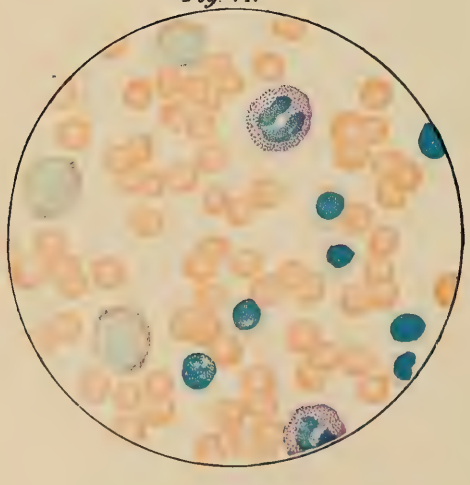


Fig. VII.

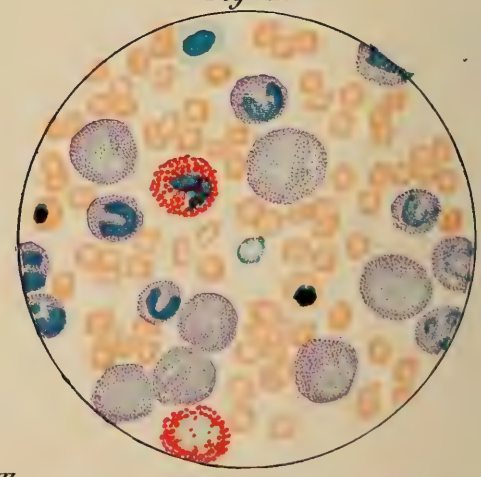
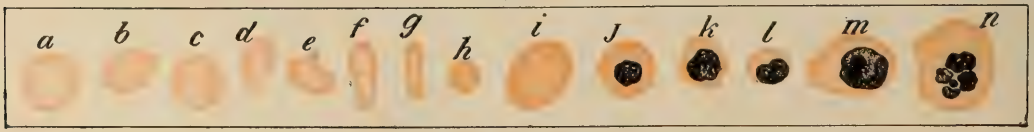


Fig. VIII.



DRAWN BY J.N.Z. CHASE

PLATE XVIII

BLOOD.

(Ehrlich Triple Stain.)

(Prepared by Dr. I. P. Lyon.)

Fig. I.—TYPES OF LEUKOCYTES.

a. Polymorphonuclear Neutrophile. *b*. Polymorphonuclear Eosinophile. *c*. Myelocyte (Neutrophilic). *d*. Eosinophilic Myelocyte. *e*. Large Lymphocyte (large Mononuclear). *f*. Small Lymphocyte (small Mononuclear).

Fig. II.—NORMAL BLOOD.

Field contains one neutrophile. Reds are normal.

Fig. III.—ANEMIA, POSTOPERATIVE (secondary).

The reds are fewer than normal, and are deficient in hemoglobin and somewhat irregular in form. One normoblast is seen in the field, and two neutrophiles and one small lymphocyte, showing a marked posthemorrhagic anemia, with leukocytosis.

Fig. IV.—LEUKOCYTOSIS, INFLAMMATORY.

The reds are normal. A marked leukocytosis is shown, with five neutrophiles and one small lymphocyte. This illustration may also serve the purpose of showing the leukocytosis of malignant tumor.

Fig. V.—TRICHINOSIS.

A marked leukocytosis is shown, consisting of an eosinophilia.

Fig. VI.—LYMPHATIC LEUKEMIA.

Slight anemia. A large relative and absolute increase of the lymphocytes (chiefly the small lymphocytes) is shown.

Fig. VII.—SPLENOMYELOGENOUS LEUKEMIA.

The reds show a secondary anemia. Two normoblasts are shown. The leukocytosis is massive. Twenty leukocytes are shown, consisting of nine neutrophiles, seven myelocytes, two small lymphocytes, one eosinophile (polymorphonuclear) and one eosinophilic myelocyte. Note the polymorphous condition of the leukocytes, *i. e.*, their variations from the typical in size and form.

Fig. VIII.—VARIETIES OF RED CORPUSCLES.

a. Normal Red Corpuscle (normocyte). *b, c*. Anemic Red Corpuscles. *d-g*. Poikilocytes. *h*. Microcyte. *i*. Megalocyte. *j-n*. Nucleated Red Corpuscles. *j, k*. Normoblasts. *l*. Microblast. *m, n*. Megaloblasts.

	Pernicious anemia.	Chlorosis.	Secondary anemia.
Red Cells	Average number 1,000,000.	Rarely under 2,000,000.	Findings as in chlorosis. May be 1,000,000 or less, though rarely.
Shape.	Poikilocytosis great, sausage-shape, etc.	Somewhat deformed.	
Size.	Average increased, some dark.	Diminished, light colored.	Various, not increased.
Hemoglobin.	Increased per corpuscle.	Reduced per corpuscle.	
Staining.	Polychromatophile cells.		
Nuclei.	Micro-, megalo-, normoblasts. Megaloblasts constitute majority of nucleated red blood cells.	Megaloblasts rare. Normoblasts more than megaloblasts.	Normoblasts far more than megaloblasts in malignant diseases. Normoblasts common (more than in chlorosis).
Rouleaux.	Not present, or decreased.		
Color Index.	High color index (not in chronic or mild cases).	Index less than 1; lower than in secondary anemias.	Relatively low (this rule does not hold positively).
Leukocytes.	White blood cells decreased, except in blood crises, hemorrhage, etc.	Usually normal.	Polymorphonuclear leukocytosis in malignant disease; when red blood cells are low, *e. g.*, 1,000,000, speaks for secondary anemia.
Lymphocytes.	Lymphocytosis (relative).	Lymphocytosis.	Rarely increased (no decrease).
Myelocytes.	Small number only.	Rare; less than in pernicious anemia.	Myelocytes very rare.
Adult Leukocytes.	Usually decreased.	Decreased.	Usually increased.
Coagulation.	Slow.	Rapid.	Often rapid.

Treatment.—1. Hygienic.—The patient should have absolute rest in bed, if possible in a warm climate. A full, nitrogenous diet increases tissue waste and produces diarrhea. Patients display a distaste to nitrogenous foods. A vegetable diet is better tolerated, with bread, zwieback, beef-tea, peptones, milk, honey or porridge. Nutrient enemata are indicated in the gastric crises. In pregnant women labor should not be induced.

2. Hematinic.—(*a*) Transfusion of human blood is seldom beneficial. (*b*) Lépine recommends the physiological *salt solution*, based on Maragliano's findings that the serum in pernicious anemia has a globulicidal action on the red blood cells (the blood of a patient clears in a test-tube on standing). (Following Talquist's experiments, Vetlesen employed glycerin with success to neutralize the hemolytic oleic acid in the blood.) (*c*) Phosphorus is without value and iron is often injurious. (*d*) *Arsenic*, recommended by Bramwell, is said to have cured the disease, and is the best remedy known, probably acting upon the blood-forming centres.

Physiological Action of Arsenic.—(*a*) The smallest dose increases the appetite. (*b*) Small doses stimulate the appetite by gastric irritation, increasing secretion and stimulating peristalsis; tissue waste is lessened. It is an alterative, increases the muscular strength and endurance, improves the quality of the blood, the complexion, sexual powers and lung capacity. (*c*) Continuous administration produces constitutional effects, as puffiness of the eyes, first seen in the morning (Wier Mitchell);

this may be followed by general edema, due to vascular and sometimes renal alteration. (*d*) Large doses result in nausea, diarrhea, abdominal colic, sympathetic headache, and neuritis. Arsenic is absorbed by the bloodvessels. The circulation, respiration and digestion are stimulated by small and depressed by large doses, although some deny the stimulant action of any dose. Elimination is very rapid and mostly by the kidneys and intestinal mucosa, but also by the skin, bile, lungs, saliva and mother's milk.

Administration.—Children tolerate large doses, while the aged stand the drug poorly and their digestion suffers; after the fifth year adult doses are given; girls tolerate the drug better than boys. When susceptibility is marked a few drops of laudanum may be added, but its administration should always be stopped when the eyes become puffy or itch, the tongue becomes furred or colic develops. It may be given in the following forms: (i) Arseni trioxidum, gr. $\frac{1}{30}$ p. c., with ext. glycyrrhizæ and piper (black pepper) āā gr. iv ("Asiatic pill"). (See formula on page 377.) (ii) *Liquor potassii arsenitis* (Fowler's solution, 1786); for hypodermic use it must be sterilized, because fungi are likely to grow in it. Even with great care abscess may develop. Minims v equal gr $\frac{1}{30}$ of arseni trioxidum. Treatment should commence with ♏iij and each day the dose should be increased a drop until ♏xij are given; rarely can larger doses (♏xv–xxv) be given. (iii) The cacodylate of soda pill (gr. $\frac{1}{6}$) may be given one to five times daily; it is seldom toxic.

3. *Symptomatic Treatment.*—Opiates for diarrhea, cardiants for weak heart, dilute HCl for gastric achylia, colonic flushings, salvarsan and splenectomy are recommended.

SECONDARY ANEMIA.

I. Acute Posthemorrhagic Anemia.—Etiology.—It may result (*a*) from trauma of large vessels: (*b*) from postpartum hemorrhage, abortion or tubal pregnancy; (*c*) from hemoptysis, aneurysm or cardiac disease; (*d*) from carcinomatous ulcerations in the uterus, gastro-intestinal tract, etc.; (*e*) from peptic ulcer; (*f*) from varicosities, as in the leg, stomach, esophagus and liver cirrhosis; (*g*) from blood conditions, hemorrhagic states or constitutional diseases.

Symptoms.—1. *The blood* is (*a*) *hydremic*, the loss of fluid being supplied by the tissues, and its specific gravity is lowered. (*b*) The *red disks* are reduced to 50 or even 20 per cent., are often irregular in contour, pale and poor in hemoglobin; generally there are some normoblasts. (*c*) The *hemoglobin* is lessened even more than the red cells. (*d*) The leukocytes show a moderate increase; the polymorphonuclears are decreased and the mononuclears increased. In anemia with rapidly resulting death the red cells are seldom nucleated and there is no leukocytosis, because the bone-marrow makes no effort to regenerate the blood. (See Plate XVIII, Fig. 3.)

2. There is *marked anemia* of the *skin and mucous membranes.* Some edema results from changes in the blood or increased permeability of the

vessel walls. The vasomotor system accommodates itself to a loss of one-quarter of the blood volume; a greater loss produces cardiac weakness. Sometimes the heart action seems more violent and respiration deeper and more rapid than normal.

3. *Albuminuria* is due to permeability of the renal vessels.

4. *Amblyopia* and amaurosis, known to Hippocrates, are at first functional, but usually later degeneration of the retina develops; as the blood-pressure rises, retinal hemorrhages may follow. The patient sees spots before the eyes. Occasional nervous manifestations include aphasia or vertigo; delirium and hallucinations are chiefly terminal events; sometimes lasting psychoses develop.

5. The *autopsy findings* are fatty degeneration, particularly of the heart, endothelium of the vessels, liver, brain, stomach and kidney cells, which is usually caused by imperfect oxygenation. Hemorrhages in the uterus, gums and retina are due to impaired nutrition of the vessel walls. The bone-marrow changes from a yellow to a red color and abounds in normoblasts.

Prognosis.—The patient may survive the loss of one-half of the blood; Hayem reports recovery after the number of red cells fell to 550,000. Men stand hemorrhage better than women and young children; the loss of a pint may cause death in arteriosclerotics. Recovery is more rapid in acute than chronic hemorrhage; water and albumin are readily yielded to the blood by the tissues; the red cells regenerate more slowly and the hemoglobin is the last element to reach normal. Increase in the coagulability of the blood is a favorable prognostic. According to Bierfreund, a loss of 10 to 15 per cent. of the blood is restored in eight days; of 16 to 20 per cent., in twenty days; of 21 to 25 per cent., in twenty-two days and of more than 25 per cent., in thirty days.

Treatment.—This has been considered under gastric ulcer and intestinal hemorrhage in typhoid, viz., absolute rest, morphine to insure quiet and salt infusions only in extreme cardiac failure.

II. Chronic Secondary Anemia.—Etiology.—Its possible causes are numerous; the chief types are (*a*) *hemorrhages* like those listed under Acute Posthemorrhagic Anemia, but frequent and persistent, as chronic hemorrhoidal bleeding. (*b*) *Poor hygiene;* overwork, long hours in poorly ventilated, sunless offices or factories; insufficient food or food poor in iron. (*c*) *Exhausting discharges or loss of albumin,* as frequent pregnancies with prolonged lactation, albuminuria, respiratory or alimentary catarrh or protracted suppuration. (*d*) *Acute infections,* as typhoid, diphtheria or malaria, in which the activity of blood-making centres is diminished and food is poorly assimilated; chronic infections, as syphilis and tuberculosis. (*e*) Gastro-intestinal and possibly other *intoxications.* (*f*) *Neoplasms* acting as mechanical obstructions (as those of the esophagus or pylorus), affecting sometimes the bone-marrow by metastases deposited there, sometimes causing repeated losses of blood or elaborating toxins, which break down the tissue albumins; in neoplasms the red cells and hemoglobin average 55 to 66 per cent. of normal. (*g*) *Parasites* (uncinaria, etc.); and (*h*) poisons (alcohol, lead, arsenic or phosphorus).

Symptoms.—The *blood is pale* and its specific gravity is lowered. The red disks and hemoglobin are reduced in rather close parallelism. The hemoglobin may fall even to 14 per cent. of normal and the red cells may, in rare instances, approach the count of pernicious anemia. The red cells stain unequally and their umbilication is clearer, so that the hemoglobin may appear only as a slight peripheral ring; many of them are undersized; they show less poikilocytosis and less of nucleated erythrocytes than in the acute posthemorrhagic form. The white cells vary with the causal conditions; the polymorphonuclear neutrophiles are increased and the eosinophiles decreased. The latter are often increased in parasitic anemias, up to 10 or even 50 per cent. In some chronic anemias the leukocytes are diminished.

It may be doubtful whether the *general symptoms* result from the anemia or from its cause. An anemic skin, muscular adynamia, irritable weakness, edema and hemorrhage into the skin, mucosæ and serosæ may be noted. There may be anorexia, tenderness over the stomach, nausea and decrease or increase of the HCl. The intestinal functions are normal. The aromatic sulphates are frequently increased. Exceptionally pernicious symptoms may develop, as alteration of the bone-marrow, fatty heart, cerebrospinal degeneration, atrophy of the gastro-intestinal cells and retinal hemorrhage. For *differentiation* see page 677.

Treatment.—The treatment depends upon the cause, for which thorough and repeated search should be made. The hygiene of the home and place of work should receive consideration. Fresh air, sunlight and proper food are often difficult to obtain for the lower classes. Iron and arsenic are valuable in simple types (*v.* pages 670 and 677).

LEUKEMIA.

Definition.—Leukemia (leukocythemia) is a disease of hemogenesis, characterized by an increase of the circulating leukocytes, hyperplasia of the leukoblastic tissues (bone-marrow, lymph glands and spleen) and by secondary infiltration into various tissues. It was discovered by Bennett and Virchow (1841).

Classification.—The older classification embraced (*a*) the splenic, (*b*) glandular and (*c*) medullary form (and also a gastro-intestinal and a dermal variety). This classification is clinical, but that of Ehrlich is based on the blood findings:

I. Lymphatic leukemia. (i) Acute lymphemia. (ii) Chronic lymphemia.

II. Myelogenic leukemia with great polymorphism in the blood.

I. Acute Lymphatic Leukemia.—*Acute lymphemia* includes nearly all acute leukemias. This infrequent disease occurs at any age, but especially in *children*, and 66 per cent. in males. The bacteriological findings are inconstant. The lymph glands are not necessarily the cause; there is evidence that the bone-marrow may be the seat of the causal change and that it may contain a parent cell for both leukocyte and lymphocyte. The chief pathological finding is *hyperplasia of the lymphadenoid tissue.* The course is that of a *rapid infective disease,* usually with an acute onset,

lymphemic blood findings, asthenia and often fever; it is fatal in a few days to a few weeks.

Symptoms.—The prodromal symptoms may be headache, pain in the neck, spleen, joints or head, epistaxis, mental obscuration, dyspnea or stomatitis, after which four cardinal findings develop:

1. Lymphadenoid Changes.—*Lymph gland* intumescence is very frequent and usually moderate. Reed published a case without glandular enlargement. *Splenic enlargement* occurs in 66 per cent. and is of slight degree; in children it may be enormous; *tonsillar* hypertrophy occurs in 50 per cent., sometimes with hemorrhage and necrosis. The *bone-marrow* is *constantly involved,* especially in the long bones; "the lymphoid marrow" is red and jelly-like, more rarely reddish-gray or hemorrhagic. The typical granular neutrophile myelocytes are few, while large and small lymphocytes abound as in the circulating blood. Opinions vary as to whether the lymphatic or the medullary lesions are the primary change.

2. Hemorrhages.—Hemorrhages are most characteristic and frequent (*in areas of lymphemic infiltration*), (*a*) in the skin, as petechiæ or larger effusions, with necrosis or ulceration; (*b*) in the mouth, gums, palate and pharynx, as petechiæ, erosions or extensive necrosis; (*c*) in the retina in 100 per cent.; (*d*) in the viscera, brain, cerebral nerves, labyrinth and spleen; (*e*) mucous surfaces, the vagina, urinary passages and intestines; and (*f*) the serous membranes.

3. Necrosis—Necrosis in the infiltrated foci of the mouth occurs in 70 per cent. and is *per se* highly suggestive of acute lymphemia.

4. Lymphemia.—*Lymphemia is the crucial finding.* The lymphocytes may reach 918,000 to the cubic mm.; in general, the increase is less than in the splenomedullary form and averages 350,000. The polymorphonuclears are decreased, the eosinophiles constitute about 1 per cent. of the white cells, a few myelocytes may be found, but the larger lymphocytes may constitute 90 or even 99 per cent. of the leukocytes. Very large primordial cells are found, from which the red and white cells are probably derived. The red cells vary between 1,000,000 and 3,000,000 and exhibit some nucleation. The hemoglobin falls below 50 per cent., or much lower in peracute cases. (See Plate XVIII, Fig. 1, Plate XVII, Fig. 2, and Plate XVIII, Fig. 6.)

Other findings are pallor, frequent pulse, priapism, exophthalmos, swollen, infiltrated liver, increased flow of urine, with increased urea and uric acid (increased nuclein destruction); albuminuria and nephritis are sometimes observed. Lymphomatous masses may cause dyspnea. The fever is irregular, both in type and occurrence. Intercurrent sepsis reduces the splenic and lymphatic enlargements, the white cells decrease even to normal and sometimes an ordinary polymorphonuclear leukocytosis replaces the lymphemia. Lymphatic deposits occur in the mouth, stomach, intestines (simulating typhoid ulcers), muscles, liver, kidneys and thymus. In a personal case the teeth were hidden by the infiltrated gums. The patient emaciates. Fatty and degenerative changes in the viscera are observed.

Diagnosis.—Though the splenic and lymphatic enlargements, stomatitis, fever, ulceration and hemorrhages are most suggestive, the blood

findings alone differentiate from typhoid, purpura, scurvy, trichinosis, diphtheria, septicemia, severe acute anemias, etc.

II. Chronic Lymphatic Leukemia.—This type is rarer than acute lymphemia and runs a clinical course of years, with lymphadenoid hyperplasia and lymphemia.

Etiology.—No constant bacteriological cause has been proved.

Symptoms.—The symptoms begin insidiously and the disease may be first detected by (1) *swelling of the lymph glands,* usually in the neck, which may reach enormous dimensions. The axillary glands are more massive than the inguinal; glandular swelling, as a rule, extends from above downward. Sometimes the internal glands alone are swollen and in rare cases the lymphatics are not enlarged. The glands are discrete, without periadenitis, oval and softer than in pseudoleukemia. Sometimes moderate pain is experienced and there may be compression of vital structures, as the vagus, trachea and portal vein. The glands grow until death, unless an intercurrent affection, as erysipelas, causes temporary shrinkage.

2. *Splenic hyperplasia* and intumescence are practically constant, though not extreme. The spleen protrudes beyond the costal arch an inch or so; it is usually proportionate to the duration of the disease. Intercurrent sepsis, cholera, erysipelas, etc., may cause the splenic tumor, lymphemia and the lymph nodes to disappear temporarily, but these findings may persist in such infections. Diarrhea and arsenic may likewise effect splenic reduction. Perisplenic adhesions and thickening are common and the organ is hard, in cases of long standing. The tonsils are often swollen, in which hemorrhages, with or without necrosis, are frequent.

3. The changes in the *bone-marrow* seldom cause pain. The essential character of the marrow alteration is undetermined.

4. The *blood* reveals an *absolute and a relative lymphemia.* The red and white cells may have a ratio of 100 to 1, yet the small lymphocytes constitute 90 to 99 per cent. of all the white cells, whereas the larger forms occur in the acute lymphemia. Many of them are degenerated or swollen. The polymorphonuclears are reduced to 10 or even 1 per cent. Myelocytes are usually absent. The red cells average 4,000,000, and seldom fall lower than 2,000,000. The lymphocytosis may temporarily disappear during infection and in one instance this was permanent. (See Plate XVII, Fig. 1.)

5. The *skin* may be the seat of papular eruptions, or lymphomata, sometimes of considerable size; they may precede the lymphemic blood findings. Edema occurs, while hemorrhages are rarer. Facial growths are observed similar to chloroma (*v. i.*) or to Mikulicz's disease.

6. The heart is degenerated, as are the vessels, from which hemorrhages may occur. Intraperitoneal hemorrhage from ruptured adrenals is reported. The nerve trunks may degenerate with bulbar, spinal and nerve-trunk symptoms. Retinitis leukemica or optic-nerve involvement may impair vision. The liver is swollen from degeneration and lymphatic infiltration. Dyspnea is most common, from anemia, mechanical com-

pression of the air tubes, vessels or nerves or from leukemic changes in the follicles of the throat and air passages. Renal infiltration is frequent.

Course.—The disease develops gradually and passes slowly into cachexia, though now and then extension in the glands is "explosive." Remissions of weeks or months are not uncommon. Intercurrent disease may be fatal, and mechanical stasis, suffocation and acute exacerbations of the disease itself may hasten the course.

Diagnosis.—The diagnosis, suggested, perhaps by the skin lesions, lymphomata, splenic enlargement or hemorrhagic tendency, is positively made only by the blood examination.

III. Myeloid Leukemia.—*Myelemia or myelogenic leukemia* is the most common form and occurs for the most part between the twentieth and fiftieth years; 67 per cent. of cases occur in males. The disease is frequent among the Polish Jews and the poorer classes. A history may be obtained of malaria, syphilis, typhoid, stomatitis, tonsillitis, digestive disturbance, pregnancy, the climacteric and trauma, though they are only accidental occurrences. The disease has been noted in three generations. Leukemia also occurs in animals.

Symptoms.—1. Blood.—The specific gravity is decreased from 1.055 to 1.045–36; it is watery or sometimes chocolate-colored; it coagulates slowly. Large white clots (leukemia) are observed at autopsy and Virchow thought he had incised an abscess when he opened the left ventricle in his first autopsy. The white cells are enormously increased, even as much as 2.5 whites to 1 red cell. In the highest count the author has seen the whites and reds numbered 1,200,000 each. Ameboid movement is greatly decreased (Plate XVII).

The *characteristics of the white cells* are: (*a*) The *large mononuclear neutrophiles* ("mark cells," myelocytes) amount to 30 to 50 per cent. of the leukocytes. They are also found (in small numbers) in pneumonia, uremia, chlorosis, skin diseases, syphilis and normal blood. (*b*) *Mononuclear eosinophiles* occur in other diseases, but in myeloid leukemia are absolutely increased and are necessary to the diagnosis. (*c*) The "*mast cells*" are constantly increased; they are polymorphonuclears with basophile granulations which do not show with the tri-acid stain. Often they are relatively increased from 0.28, the normal percentage, to 1 or indeed 18 per cent. (*d*) The *polymorphonuclear* neutrophiles and eosinophiles constitute 30 to 60 per cent. of the leukocytes. (*e*) The *lymphocytes* are increased. (*f*) Atypical leukocytes, dwarf forms and white cells containing mitoses, fat globules or red cells may also be found. (See Plate XVIII, Fig. 2.)

The *red cells* are decreased to 3,000,000 or 2,000,000 and toward the end may decrease to even 300,000. The reds may exhibit degeneration necrosis, polychromatism, ameboid movement, mitoses and nucleation (more frequently than in any other anemia, normoblasts being found regularly). The color index is 1 or less than 1. (See Plate XVII, Fig. 1, and Plate XVIII, Fig. 7.)

2. Splenic Enlargement. — This is usually the first symptom to attract attention and appears with the blood findings; spleniculi, if present, are enlarged. Instead of a weight of 5 to 7 ounces, 18 to 28

pounds are reported; from the normal measurements, 3 to 5 inches, dimensions of 15 inches are frequently observed. The form of the organ is preserved, the notches are palpable and the edge plump, rounded and hard. It may extend into the pelvis, displacing other organs. The size varies during intermissions, from administration of arsenic and from diarrhea or hemorrhages. A systolic murmur or pulsation is sometimes detected. Crackling under the fingers and tenderness indicate soft adhesions (perisplenitis). Firm adhesions may develop and the capsule may thicken. Though at first soft, it later becomes very firm; the pulp and trabeculæ hypertrophy and new lymphoid deposits are clearly marked against the reddish pulp (myeloid metaplasia). The author has seen 3 cases of leukemic floating spleen, 2 of which had been operated on as uterine tumors. Rupture is a rare complication.

3. Glands.—Enlargement occurs in 33 per cent. The glands are heteroplastic, due to new-formed myeloid tissue, "metastases" from the bone-marrow. Soft at first, they later become hard. Caseation and suppuration are most infrequent. As in the other types of leukemia, recurrent compression of the vagus, trachea, portal vein and bronchi and retrosternal dulness have been observed.

4. Bones.—In the spongy and long bones (ribs, tibiæ, sternum and vertebræ), the bone is mostly rarefied and there may be tender or yielding areas. The histological changes in the marrow are (*a*) the *lymphoid* (Neumann), in which the reddish marrow resembles currant jelly and consists of small lymphocytes; it is sometimes hemorrhagic; the normal fatty appearance disappears. (*b*) The *pyoid*, which is seen mostly in the myelogenic type; the marrow is not transparent, but gray, its cells are rich in protoplasm with one or many nuclei. In both forms nucleated red cells, eosinophile cells and large cells with neutrophile granules and large nuclei abound.

5. Vascular System.—The heart is distended with white clots and the muscle is pale, fatty and displays nodules of leukemic infiltration and foci of hemorrhage. Cardiac dislocation from pressure by enlarged glands and spleen, anemic bruits, venous pulsation, *bruit de diable*, palpitation, systolic murmurs from pressure by the glands on the vessels and centripetal venous pulse are observed. The radial pulse is rapid. Dyspnea results from weak heart, vagus pressure, nodules in the lung, bronchitis, hydrothorax, pressure of the spleen on the heart and compression by the glands.

6. Respiratory Tract.—Nodules are frequently seen in the larynx, trachea, thyroid and thymus, often with hemorrhage and sometimes with tracheal, bronchial or laryngeal stenosis. In the lungs lymphoid nodules may necrose and form cavities like those of caseous tuberculosis; small myeloid foci of leukocytes may resemble miliary tuberculosis. Bronchitis with many eosinophile cells in the sputum and pleural hemorrhage or exudation are observed.

7. Fever.—Fever occurs in the majority of cases. It is rarely high; it may resemble recurrent fever, typhoid, sepsis or malaria.

8. Digestive Canal.—Stomatitis or pharyngitis with ulceration of the lymphoid deposits, dysphagia from glandular pressure on the

esophagus, eructations and vomiting are occasional symptoms. *Diarrhea* is very common, with leucin and tyrosin in the stools. In the enteric type the bowel symptoms are conspicuous, the lymphadenoid structures in the ileum prominent and Friedreich once confused this form with typhoid on account of the ulceration, enlargement of the spleen and hemorrhage from the bowel. The abdominal lymph glands may be enlarged alone. The *liver* is much enlarged and may weigh 13 to 25 pounds. Its surface is smooth, its consistence increased and its edges plump. Myeloid deposits occur in the liver and deform the rows of liver cells. The portal lymph nodes may be enlarged. Icterus is infrequent and ascites is common from cachexia or leukemic peritonitis. The fluid contains many myelocytes.

9. Kidneys.—Frequently there is myeloid infiltration and cloudy, fatty and amyloid alteration. The urine is decreased, pale and strongly acid. The *uric acid* may amount to 20 or 105 grains. Normally uric acid is as 1:50–80 of urea, but in leukemia it is 1:16. The xanthin bodies are increased. Albuminuria is usual only in the last stages. The amount of urea depends on the degree of cachexia. Priapism occurs from nervous lesions or thrombosis of the dorsal vein. Amenorrhea is frequent.

10. Nervous Symptoms.—These complications include delirium, neuralgia, headache, vertigo or mania or rarely bulbar palsy, facial paralysis, apoplexy or symptoms like those of brain tumor.

The *Eye.*—Lymphoid nodules may develop in the orbit, possibly with exophthalmos. The *retinitis leukemica,* purely a leukemic infiltration, occurs in 33 per cent.; the retina is pale, its arteries small and veins large; the papilla is "washed" and yellow spots with red borders and hemorrhages appear (Plate VI, Fig. 9).

The *Ear.*—Vertigo, deafness, tinnitus and an atactic gait may result from growths in the labyrinth; the author saw within one year 3 cases of sudden deafness from hemorrhage.

11. Skin.—There may be acne, pigmentation, edema, pruritus, lymphoderma perniciosa (diffuse or tumor-like infiltrations) or sweats.

12. Hemorrhages.—Cabot found them in 80 per cent. Epistaxis is the most common and may be fatal. Stomach and bowel hemorrhages rank next and those from the respiratory and genito-urinary tracts, the skin, joints, muscles, or brain are rarer. The writer has seen three massive hemorrhages under the skin of the thorax. Death has followed the extraction of teeth in a leukemic subject. The writer saw Kolisko autopsy a slow ascending paralysis following hemorrhage between the spinal pia mater and arachnoid.

Complications.—Complications are nephritis, pneumonia, endocarditis, amyloid degeneration, diabetes and gangrene. Dock could collect but 27 cases of complicating tuberculosis; the author saw a case in which tuberculous pleurisy developed; tubercle bacilli were recovered from the blood-stained fluid.

Diagnosis.—Formerly the difference between leukemia and leukocytosis was made one of degree only and various limits were placed, beyond which leukocytosis was said to cease and leukemia begin. The mere number of white cells is no criterion, because a ratio of 1 to 20 may be

leukocytosis (as in gastric cancer), and on the other hand, 1 to 200 may be leukemia. Leukocytosis from digestion, fasting, marasmus, pregnancy, acute infections or in the death agony is of the ordinary polymorphonuclear neutrophilic type. (Plate XVIII, Fig. 4.) Leukocytosis is a symptom, but leukemia is a disease. The *polymorphism* of the blood is striking; there must be (*a*) mononuclear leukocytes with granulations (myelocytes); (*b*) increased mono- and polynuclear eosinophiles; (*c*) absolute increase in the "mast cells," and (*d*) nucleated red cells. The gross splenic, medullary or lymphatic lesions are of presumptive value in diagnosis but the polymorphic blood picture alone differentiates from pseudoleukemia, typhoid, purpura, anemia, sepsis, etc.

Caution should be exercised (*a*) during remission; in one of the author's cases the leukocyte count dropped from 1,250,000 to 9000 in a week, though the myelocytes still constituted 33 per cent. of the white cells. (*b*) The leukemic characteristics may disappear during typhoid, acute miliary tuberculosis, influenza or sepsis. (*c*) The blood findings may become atypical before death.

Similar Conditions.—1. Chloroma is considered a malignant type of leukemia or a "neoplastic hyperplasia of the red bone-marrow," by Dock and Warthin; 80 cases are on record. Lymphomata form, causing orbital pain, tinnitus, deafness, temporal and orbital swellings and exophthalmos, strabismus, disturbed vision and retinal infiltration and hemorrhages. The lymphomata are greenish, whence the name chloroma. Other findings are ataxia, pallor, sternal tenderness, hemorrhages, greenish urine and greenish lymphomatous infiltration of the bone-marrow, periosteum, liver, lungs and glands. The hemoglobin and red cells are reduced, even to 15 per cent. and 500,000 respectively. The leukocytes number about 50,000, even 500,000; they are chiefly lymphocytes. This form is called lymphoid, to differentiate from the rarer myeloid type, in which the leukocytes are myelocytes; Jacobæus collected 10 myeloid cases (1909). Chloroma is always fatal, usually within six months.

2. Leukanemia is a severe blood disease in which the myelogenous formation of both the red and white cells is disordered, *i. e.*, it combines some features of both leukemia and pernicious anemia, *e. g.* (*a*) a progressive anemia, waxy color and asthenia; (*b*) numbers of normoblasts, but especially megaloblasts; (*c*) no abnormal pigments in the urine, no adenopathy and no siderosis of the tissues; (*d*) no true leukemic blood findings, but considerable myelocytosis and lymphocytosis; (*e*) firm connective tissue in the bone-marrow and (*f*) enlargement of the spleen and prevertebral hemolymph glands.

3. Secondary Cancer in Bone.—There are instances in the literature in which cancerous bone metastases have produced blood findings rather like those of pernicious anemia and increase of the leukocytes (of which the myelocytes constituted 4 to 17 per cent.).

Prognosis.—Leukemia may last five or even ten years, but a little over one year is the average *course*. The *beginning is insidious* and enlargement of the abdomen, dyspnea, anemia and adenopathy are the cause for seeking medical treatment. *Sudden death* may occur from hemorrhage

of the brain, hematemesis, marasmus, anasarca, diarrhea, pleuritis, peritonitis, deep coma or pneumonia. *Remissions* occur, but the blood findings persist. Twelve *acute cases* were collected by Gordinier (1904); they resemble an infection with hemorrhages, moderate splenic and lymphatic hyerplasia, severe anemia, necrosis of the throat and jaw and low fever. A permanent cure after erysipelas was reported by Richter.

Treatment.—Fats and carbohydrates cause dyspepsia, whence we give eggs, milk, raw, chopped meat, koumyss, oysters, zwieback, spinach, etc.; an ice-bag to the spleen may mitigate pain. *Benzol* causes an initial rise in the leukocytes, especially after small doses but a decrease after two or three weeks, *e. g.*, from 988,000 to 1720; an aplasia of the bone-marrow, spleen and lymph glands ensues. Given with olive oil, āā ♏vij, the dose is pushed to ♏xv (℥j daily); larger doses produce burning in the stomach, eructations, bronchitis, vertigo or necroses in the liver and kidneys. Acute exacerbations may follow its exhibition. The drug is discontinued when the white cells reach 20,000. *Splenectomy* is irrational, for leukemia is a primary disease of the bone-marrow; 94 per cent. of Bissel-Hagen's group of 35 died, chiefly from hemorrhage. Caution must be observed in using *purges*, which, as in Addison's disease, may cause sudden collapse and death. Inhalations of oxygen, blood infusions, spleno- or organotherapy and iron are inferior to *arsenic*, which decreases the number of white blood cells and the size of the spleen, and inhibits hemorrhage. In tapping an ascites, peritonitis is prone to develop. The *x*-rays may prove beneficial; in 1 case already alluded to, the patient lived nineteen months after the day he seemed to be dying. The *x*-rays produce a leukolytic substance. Wendell thinks 90 per cent. are benefited. The exposure to the *x*-rays should average fifteen minutes daily, carefully avoiding any toxic reaction. In some cases the glandular and splenic enlargements and the blood findings wholly disappear.

PSEUDOLEUKEMIA.

Definition.—A disease resulting in chronic anemia or cachexia, with enlargement of the spleen or lymph structures. The definition of the disease as "leukemic appearance without leukemic blood findings" no longer holds, as it would include glandular tuberculosis, lymphosarcomatosis, etc. It is also called Hodgkin's (1832) disease, pseudoleukemia (Cohnheim), adenia (Trousseau), malignant lymphoma (Billroth) and anemia lienalis or lymphatica (Wilkes).

Etiology.—The etiology is obscure. Like chloroma and leukemia, 75 per cent. of cases occur in males between twenty and thirty years of age. The disease often occurs in the lower animals. Local lymphatic disturbances in the neck (following otorrhea, coryza or tonsillitis), malaria, syphilis, scrofulosis, alcoholism, rhachitis, typhoid, measles and scarlatina are often noted in the patient's history, but their relation, if any, is obscure. Bunting and Yates cultivated a pleomorphic diphtheroid organism. The disease has no established relation to leukemia, yet transitions from pseudoleukemia to leukemia have been observed. Neumann held that the leukemic blood changes depended solely on the hyperplastic

marrow changes and that as long as the hyperplasia involves only the spleen and glands pseudoleukemia results. Sternberg's contention that it is tuberculosis, is disproved.

Symptoms.—Its types are (*a*) lymphatic, (*b*) lienal, pseudoleukemia splenica, splenomegalia, (*c*) mixed and (*d*) myelogenic.

1. Lymph Glands.—Swelling of the lymph glands is generally the earliest symptom; the cervical glands enlarge first, then the axillary, later the subpectoral, inguinal and other groups. They are soft and elastic; at first the glands are oval, discrete and smooth; later with increasing size, they fuse into harder, irregular masses as large as the fist or a child's head, particularly those in the neck. They may be painful from periadenitis, but never transcend the gland capsule and seldom caseate or suppurate. From the neck, where the jugular veins may be compressed, the glandular enlargement often passes to the mediastinal glands, which are more often enlarged than the other internal nodes; this often occasions dyspnea, compression of the superior cava (whence the development of a large collateral circulation on the chest wall), dysphagia, bronchi compression and recurrent laryngeal or vagus paralysis. The veins of the arm may be compressed, causing edema. In the abdomen the retroperitoneal, mesenteric, periportal, iliac and other groups may enlarge greatly; according to their location, abdominal pain, edema of the legs, paraplegia, hydronephrosis, ascites and jaundice may ensue. *Histologically*, the lymphadenoid changes are (*a*) increase in the endothelial and reticular cells; (*b*) hyperplasia of the lymphoid cells; (*c*) formation of peculiar giant cells; (*d*) fibrosis and chronic inflammation and (*e*) great increase of the eosinophile cells (Dorothy Reed).

2. Spleen.—The spleen, enlarged in 75 per cent. of cases, reaches dimensions rarely attained in leukemia, but maintains its form, is hard and tender and presents the same histological appearance as in leukemia, viz., nodes of lymphoid cells in a fibrous reticulum. The greatest splenomegaly is observed when the lymphatic glands are large and hard; solitary splenomegaly with relative increase in the lymphocytes, is classed as Hodgkin's disease by Pinkus.

3. Bone-marrow.—The bone-marrow is not frequently involved. A myelogenous pseudoleukemia has been recorded, but its status is doubtful. The eosinophiles abound in the bone-marrow.

4. The Blood.—The hemoglobin is decreased and the color index is usually 1. There is moderate anemia, especially in advanced cases. Poikilocytes and microcytes often occur, megalocytes rarely and normoblasts sometimes, but the eosinophiles are not increased. The blood plates are increased. There is a relative increase of lymphocytes but no total increase in the leukocytes. The polymorphonuclear neutrophiles are decreased. Leukocytosis generally occurs in the death agony or in a terminal infection.

5. Other Organs.—(*a*) *Lymphatic growths in the skin* occur more frequently than in leukemia; the lymphoderma perniciosa of Kaposi may be considered pseudoleukemic in cases with an absolute lymphocytosis. Erythema, purpura, furuncles, pemphigus, prurigo from irritation of the lymphatic deposits around the sweat glands and pig-

mentation have been observed; severe *sweats* are frequent. (*b*) Symptoms of *anemia* occur, as vertigo, syncope, palpitation, dyspnea, edema, hemorrhage, dyspepsia and diarrhea. (*c*) *Local pain* over the *liver* or *spleen* indicates perihepatitis or perisplenitis. Swelling of the liver is due to lymphoid deposits. Severe muscular pain has suggested trichinosis. (*d*) Gangrenous stomatitis or pharyngitis may occur in degenerated lymph nodes in the mouth, tonsils, tongue and throat. Gastric and intestinal ulcerations and sometimes perforation are noted less frequently than in lymphosarcoma. Neusser noticed gastric crises like those of tabes. (*e*) Ross and Osler described lancinating pains in the feet and edema from involvement of the spine and cava by the lymph nodes. (*f*) Amyloidosis, bronchial catarrh and pulmonary phthisis have been noted. (*g*) The uric acid in the urine is not increased. (*h*) The lymphatic hyperplasia may also involve the tonsils, circumvallate glands, mammæ, eyelids, thyroid, thymus, lungs, heart, pleura, brain, genitalia and pelvis.

6. Fever.—Fever is common. Gowers and Murchison observed a chronic recurrent type, later described by Pel and Ebstein, but also observed in chronic malaria, lymphosarcoma, cancer and glandular and splenic tuberculosis. The urine may show the diazo reaction. When the disease involves the internal glands only, the fever may simulate typhoid. Fever is rather indicative of glandular tuberculosis or of intercurrent (terminal) infections.

Course.—The clinical course is long. The glandular swelling sometimes decreases with or without therapeutic measures. The extension from the cervical to other lymphatic glands is generally gradual, but may occur explosively, as in Trousseau's case. An acute form of Hodgkin's disease is not well established; many are sepsis, tuberculosis or acute sarcomatosis. *Death* most frequently results from chronic cachexia, but also from obstruction of the air passages, twisting of the pedicle of the spleen or from hemorrhages from the nose, uterus, stomach, intestines or larynx, which sometimes resemble purpura hemorrhagica. It is a question whether well-developed types ever recover. Pleural effusions, diarrhea, dysentery and vomiting from pressure of the splenic tumor are unfavorable complications.

Differentiation.—There are two criteria; (*a*) the lymphadenoid hyperplasia and chronic inflammation observed when an excised node is examined histologically; and (*b*) an absolute lymphocytosis, with a practically normal number of white blood cells.

1. Glandular Tuberculosis.—Hodgkin's disease may be confused with tuberculosis; in pseudoleukemia the typical structure of the tubercle is lacking, but in tuberculous adenitis there may be an accumulation of large endothelioid cells between which lie tubercle bacilli, without caseation. Of late the reports of glandular tuberculosis simulating Hodgkin's disease have increased enormously. The author examined two supposed pseudoleukemias in which tubercle bacilli (without anatomical tubercles) were found in the spleen and lymph nodes. In the neck tuberculous adenitis is more often in the submaxillary than in the anterior and posterior glands, in the latter of which pseudoleukemia is more common. Tuber-

culosis is more frequently attended by secondary suppuration. Fever and glandular tenderness indicate tuberculosis and node-like swellings of the lymph vessels in the mouth are symptomatic of pseudoleukemia.

2. Anemia Infantum Pseudoleukemica.—This disease was first fully described by von Jaksch; it is known in Italy as *anemia splenica infettiva dei bambini;* it is called "pseudopernicious anemia of children" by Ehrlich. It is a profound anemia in which the blood findings suggest pernicious anemia; there are normoblasts, megaloblasts, decrease of the red blood cells (1,500,000 to 3,000,000) and sometimes poikilocytes or polychromatophilia; the leukocytes rarely exceed 50,000. It occurs in infants from nine to eighteen months of age; the convalescence is very slow or death may result. Its other symptoms are firm enlargement of the spleen, swelling of the liver and hemorrhages into the skin, nose, mouth stomach or intestines. The status of the disease is not yet determined. The diagnosis is especially difficult when it occurs in syphilitic children; syphilis, rickets and dyspepsia are common antecedents.

3. Anemia Splenica.—It is doubtful whether it should be classed as an essential anemia, liver cirrhosis, chronic splenitis or as a splenic form of Hodgkin's disease. (*a*) The *spleen is enlarged,* its capsule thick, its stroma increased and the endothelium of the lymph sinuses sometimes proliferated (endothelioma). The cause is possibly toxic. (*b*) There is an *anemia of the secondary* type; the red cells average 3,500,000, the hemoglobin is even more reduced and the whites are decreased to 3000 or 4000. (*c*) *Hemorrhages,* particularly from the stomach, occur in one-third to one-half the cases. (*d*) In many cases there is a terminal *liver cirrhosis,* with icterus and ascites (Banti's disease). (*e*) Its course is *very chronic.* In some cases there are profound alterations of nutrition, stunted growth, clubbed fingers or pigmentation of the skin. In several cases Dock and Warthin found portal thrombophlebitis and hyperplasia of the hemolymph glands. Of Armstrong's cases 23 recovered and 9 died.

4. Lymphosarcomatosis.—According to Kundrat, lymphosarcoma is largely a local glandular involvement, especially of the cervical glands, leading to infiltration of the throat by contiguity; the disease may begin in the tonsils, air passages, portal glands, etc. Lymphosarcoma ulcerates and transgresses the gland capsule; this never occurs in leukemia or in pseudoleukemia; though the glands may compress neighboring structures they never actually invade them. Unless lymphosarcoma extends beyond the capsule of the glands it cannot be distinguished by physical examination unless it lies in parts which can be easily inspected, as the mouth. The spleen may enlarge enormously. According to Pinkus, the leukocytes are increased in pseudoleukemia, while in lymphosarcoma they are decreased.

5. Other Splenic Enlargements.—The most common are the spleen of cirrhosis, malarial spleen, chlorosis with enlarged spleen, chronic polycythemic cyanosis and rarer forms, as miliary tuberculosis of the spleen, actinomycosis, syphilis, etc.

6. Multiple Myeloma.—This is characterized pathologically by lymphoid hyperplasia of the bone-marrow, in the ribs and body ends of

the long bones chiefly and more often in the proximal than distal bones; no mestastases occur. Clinically, it occurs in males over forty-five years and is characterized by severe, *deep-seated pain* in the body, chest, spine, neck and more rarely in the extremities. Prominences in the bones, *spontaneous fractures,* especially of the sternum and ribs, and kyphosis, from softening of the vertebræ, may result. The growths may pulsate or rupture externally. The *x-rays* may clearly define them. Severe anemia without leukocytosis; motor and sensory paralyses, which develop late and involve the opticus, hypoglossus, facialis, cord (paraplegia dolorosa) and sciatic nerve; fever and *Bence-Jones's albumosuria* are observed. To call this disease a myelogenic form of pseudoleukemia or myelogadenia there must, according to Pinkus, be a lymphocytosis.

7. Other Affections.—Syphilitic lymphatic glands; Mikulicz's lymphoma of the lachrymal and salivary glands and secondary carcinoma or sarcoma must be differentiated. Difficulties of diagnosis are met when the enlarged glands in internal parts produce topical symptoms, thus simulating hypertrophic cirrhosis, etc.

Treatment.—1. Local Applications.—These are useless, excepting the *x-rays;* two cases, seen by the author eleven years ago, wholly recovered. In some cases the rays have no influence on the spleen and glands and in others the cachexia increases, though there may be improvement in the local findings. Inunctions of green soap, once or twice daily, are sometimes beneficial.

2. Drugs.—*Arsenic* is most efficacious (*v.* page 677); it acts on the glands more than on the spleen. Some believe that the fever which it sometimes produces, reduces the adenopathy. Winnewarter and Billroth recommended its parenchymatous injection into the glands, but the results are not very encouraging. Iodides are injurious.

3. Surgical Treatment.—Extirpation of the glands is seldom beneficial. The removal of the spleen is attended by constantly decreasing mortality; the average mortality in splenomegaly was 61 per cent. before 1891 and since then 13 per cent. Carstens, 1905, collected 739 cases of splenectomy; 72 per cent. recovered.

ERYTHREMIA.

Vaquez's disease (1892) is a syndrome including polycythemia, splenomegaly and cyanosis. Lucas collected 189 cases (1912). The etiology is obscure; lienal tuberculosis is reported in the earlier cases, but the cause is probably excessive erythroblastic activity of the bone-marrow. (*a*) The *red cells are increased,* even to 9,000,000 or 15,000,000 (polycythemia rubra or polyglobulism); the hemoglobin may exceed 200 per cent.; the white cells number from 4000 to 31,000, with increased polymorphonuclears. The viscosity, specific gravity and volume of the blood (plethora) are increased. (*b*) There is *chronic cyanosis,* or intense redness of the face, in 75 per cent. and (*c*) *splenomegaly.* The retinæ exhibit congestion, even choking. There may be vertigo, headache, dyspnea, enlarged liver, mental and physical weakness, albuminuria, constipation, vomiting,

pigmentation and a hemorrhagic diathesis. The arterial tension is increased and the heart hypertrophies.

The prognosis is unfavorable and therapy uncertain—arsenic, benzol, *x*-rays, iodides, nitrites, phlebotomy and splenectomy.

THE HEMORRHAGIC DISEASES.

The hemorrhagic diseases may be classified as follows:

(A) Acquired types. { 1. Purpura. 2. Scurvy.

(B) Congenital (hereditary) type. 3. Hemophilia.

Purpura. — Purpura includes a number of affections, more or less similar and differing chiefly in degree, some symptomatic, others independent.

I. Symptomatic Purpura.—Purpura may be symptomatic of: (*a*) Various *infections*, as endocarditis, scarlatina, measles, smallpox, rheumatism, sepsis, syphilis, etc. (*b*) *Toxemias*, as nephritis, cholemia, snake-bites and drug poisoning (copaiba, ergot, belladonna, quinine, mercury, iodides, bromides or arsenic). (*c*) *Cachexia;* old age, malnutrition, carcinoma, sarcoma, leukemia, pseudoleukemia or pernicious anemia. (*d*) *Nervous* states; tabes, neuroses (bloody stigmata of hysteria) or neuralgias. (*e*) *Mechanical* conditions, as trauma, embolism or stasis.

II. Purpura Simplex.—In purpura simplex (Zeller, 1684) cutaneous hemorrhages are the chief symptoms. Its *etiology* is obscure. It develops most often in middle-aged males. As in the other forms, whose description follows, there is no constant or characteristic *pathology*, though vessel thickening, hyaline and fatty degeneration, thrombosis, and pigmentation sometimes occur. In the blood itself no change is observed other than its tardy coagulation, in 10 to 30 minutes instead of 3 to 5, which is the normal time. The blood platelets are decreased. The *eruption* begins as small red spots, which gradually become green and black; they occur largely on the extensor surfaces of the limbs and more often on the legs than on the arms or trunk. They do not fade upon pressure. The initial eruption may be macular, papular or urticarial (purpura maculosa, papulosa or urticans), but soon becomes blood-tinged. Constitutional symptoms are usually absent; there may be slight fever, gastric disorder or diarrhea. The *course*, sometimes with relapses, is 1 to 2 weeks, the *prognosis* good and the *treatment* hygienic.

III. Purpura (or Peliosis) Rheumatica.—Purpura rheumatica, described by Schönlein, is somewhat arbitrarily listed by itself because of its articular symptoms, although joint symptoms also occur in Werlhof's disease (*v. i.*), scurvy and hemophilia. Most cases develop in males between fifteen and thirty years of age. It has no relation to rheumatism, being a pseudorheumatism. Its pathology is obscure.

Symptoms.—(*a*) As *prodromes*, indisposition, muscular pains, colic and, particularly, sore throat may be noted. (*b*) The *purpura* may occur as simple hemorrhages of the skin, which vary from red to greenish-black, according to their age, or as urticaria, erythema, pemphigus or edema,

which becomes stained with blood; the legs are most often involved, largely on the extensor surfaces. (*c*) *Articular symptoms* are most common in the knees and ankles, less frequent in the elbows and rare in other joints; there may be only stiffness and pain, or actual swelling; if the joints are first involved their symptoms frequently abate with the appearance of the purpura. (*d*) *Other symptoms* are inconstant, as fever, which may rise to 102° or 103°, splenic tumor, albuminuria or nephritis. The author had 3 cases at one time in the Cook County Hospital, in which there was severe sloughing of the tonsils. Osler (1904) described 29 cases in which *visceral lesions* occurred with purpuric, urticarious and erythematous symptoms, chiefly in children and often with relapses; (i) digestive complications occurred in 25 cases; vomiting in 15; colic in 8 (resembling renal stone or appendicitis); blood in the stools in 8 and diarrhea in 15; (ii) acute nephritis occurred 14 times and 5 cases died of uremia; (iii) arthritis occurred in 17 cases; (iv) swelling of the fauces, edema of the larynx, aphasia, transient hemiplegia and least often endocarditis may occur.

Diagnosis.—The diagnosis chiefly concerns affections characterized by hemorrhages of the skin, articular symptoms and fever. Acute leukemia, septicopyemia and endocarditis must be excluded. Genuine rheumatic polyarthritis is differentiated with ease by the earlier migrating, inflammatory arthritis, the fever, sweating, characteristic cardiac complications and the later, accidental and wholly secondary purpura.

Prognosis.—The prognosis is generally good, relapses are common, perhaps periodic and its course rarely exceeds two weeks.

Treatment.—The joint symptoms yield somewhat to acetanilide, though the salicylates exert no specific influence.

IV. Purpura Hemorrhagica.—*Morbus maculosus Werlhofii* (Werlhof, 1775) closely resembles the above types, but differs in that not only are the skin and joints the seat of hemorrhage, but also the mucous membranes, serous membranes and perhaps the viscera.

Etiology.—Of this little is known; staphylo-, strepto- and pneumococci and Bacillus pyocyaneus and coli have been found. It occurs oftener in young, weakly women than in robust adults. Exposure ot cold or dampness and malnutrition seem predisposing causes. It usually develops as an apparently primary affection.

Symptoms.—(*a*) *Hemorrhages* appear on the *extremities;* the face is not often involved; they occur as spontaneous petechiæ, blood-stained bullæ or vibices or may result from slight trauma, as by pinching the skin. (*b*) Of the *mucous membranes* the nose and mouth bleed most often; less frequently there is hemoptysis, hematemesis, enterorrhagia, hematuria or metrorrhagia in various combinations, sequences and degrees. The conjunctivæ may be blood-stained. The writer saw in one rapidly fatal case hemorrhage from the middle ear. (*c*) The joints, particularly the knees, ankles and elbows, are less often implicated. Ecchymoses in the endo- and pericardium, pleura, peritoneum and meninges are common autopsy findings. (*d*) Hemorrhage into the retina, brain, cord or testis is far less common. The connective tissues, fasciæ and bones are practically exempt.

General Symptoms and *Complications.* As to the blood, there is delayed coagulation and moderate anemia; some nucleated red cells may be found, as well as leukocytosis, reduction in the blood plates and methemoglobinemia. Fever is inconstant and atypical. The pulse-rate may be raised; palpitation, delirium, vertigo, albuminuria, hepatic enlargement, splenic tumor, subicterus, intestinal ulceration or perforation and nephritis occasionally occur.

Diagnosis.—Most mistakes result from confusion with purpura symptomatic of sepsis, hemorrhagic exanthemata or blood affections. The difficulties in diagnosis are often great; in an anemic old man, who suddenly vomited blood and passed bloody urine, the diagnosis of purpura was made; the autopsy revealed a round ulcer of the stomach and a papilloma of the bladder. *Scurvy* is distinguished by its gingivitis and muscular hemorrhages, and *hemophilia* by reason of its hereditary facts. *Henoch's purpura* (1874) occurs chiefly in children and is characterized by (*a*) cutaneous lesions, which resemble erythema nodosum rather than purpura; (*b*) hemorrhages from the mucous membranes; (*c*) gastro-intestinal crises of vomiting, diarrhea and abdominal pain; (*d*) pains in the joints and (*e*) relapses, often extending over a number of years. Of *chronic purpura*, Bensaude and Rivet collected 34 cases (1905).

Prognosis.—The prognosis is uncertain; 50 per cent. die. Unfavorable factors are high temperature, nephritis and cerebral hemorrhages. Recurrence is not uncommon. *Purpura fulminans*, of which C. A. Elliott (1909) collected 56 cases, may be fatal in 24 to 48 hours; it was described by Guelliot, 1884.

Treatment.—Werlhof's therapy was dilute sulphuric acid, ♏x, and quinine, gr. v, after meals. Everything of a supportive nature, as sufficient nourishment, rest in bed, iron and arsenic, is helpful. The hemorrhages are arrested with difficulty and the author doubts the value of drugs in their control. Adrenalin solution to the nose and mouth, gelatin by mouth, calcium lactate, gr. xv, t. i. d., to increase the coagulability of the blood and antitoxin or simple, fresh serum are indicated (*v.* Hemophilia).

Hemorrhagic Diseases of the Newborn.—Syphilis Hemorrhagica Neonatorum.—It appears soon after birth. In some instances the syphilitic child may be born with hemorrhages in the skin, mucosæ, internal organs and from the navel. Occasionally deep jaundice develops. Mrâcek found a causative endartertitis, especially of the capillaries, in 14 per cent. In other cases an added sepsis apparently coöperates.

Winckel's Disease.—Winckel's disease, epidemic hemoglobinuria, is (*a*) an affection of the newborn in the first four days of life; (*b*) it appears epidemically in hospitals; (*c*) it is characterized by fever, gastro-intestinal symptoms, icterus, dyspnea, cyanosis, albuminuria, methemoglobinuria and large mortality, and (*d*) pathologically by internal hemorrhages, fatty degeneration (Buhl's disease) and splenic tumor; it is doubtless a hemolytic sepsis.

Morbus Maculosus Neonatorum.—It is no clearer etiologically than Werlhof's disease in adults; some cases are congenital syphilis; others with fever, jaundice and epidemic occurrence are infections (pyogenic cocci,

pneumococci, pyocyaneus and colon bacilli); and still others seem embolic, thrombi forming in the auricles when the heart is weak. Injuries to the head, causing hemorrhages in the fourth ventricle and cerebral peduncles, and other vasomotor disturbances are questionable causes. It occurs once in 500 to 1000 births.

Symptoms.—In the first week of life hemorrhages occur from the navel and mucous membranes; in Townsend's 50 cases they occurred in the following frequency: from the bowel (melena), 40 per cent.; navel, 36 per cent.; mouth, 28 per cent.; stomach, 28 per cent.; and nose, 24 per cent. Death occurs within a week in 63 per cent. Dusser, in 78 cases, found the mortality was 55 per cent.

Treatment.—Iron, ergot and adrenalin are useless. Diffuse stimulants should be given when there is a weak, rapid pulse. Injections of gelatin are dangerous, for it possibly contains some ptomaine; fresh serum or direct transfusion is most reliable.

Scurvy.—Definition.—A general disease, characterized by gingivitis, hemorrhages, inflammations, cachexia and chronicity of course.

Etiology.—It was first observed in the Crusades and was described most fully by Vasco de Gama; in his voyage around the Cape in 1498, 33 per cent. of his sailors were stricken with scurvy. It has developed among prisoners, Canadian lumbermen, Pennsylvania miners and among workers on the Chicago Drainage Canal, where in 1893–95 the author saw 100 cases. Insufficient, possibly spoiled food causes the majority of cases, sometimes in conjunction with lack of fresh meat, impure water, damp quarters and overexertion; a lack of fresh meat, vegetables (potassium salts, malic and citric acids) and fats predispose to scorbutus, though no single group is solely responsible. The Drainage Canal cases were Poles who lived on coffee, bread and preserved and fresh meats. In 1849 there were 260,444 cases in Russia and 60,598 deaths; it is still endemic there.

Symptoms.—Scorbutus begins insidiously and progresses slowly; its earliest symptom is (1) *anemia* (cachexia scorbutica). The skin is scaly, pale, lustreless and perhaps pigmented. The mucous membranes are livid. The blood coagulation is slow, the red cells and hemoglobin are proportionately reduced and leukocytosis is absent, except with intercurrent inflammation or severe hemorrhage. Emaciation, mental depression, muscular weakness, anorexia, hypochlorhydria, intestinal fermentation, constipation, palpitation, weak and irregular heart action, a systolic functional murmur and edema about the ankles accompany the anemia. Splenic enlargement, albuminuria, peptonuria and oliguria are common.

2. *Gingivitis* begins with turgescence of the gums, which is due to a serohemorrhagic infiltration, noticed first about the canine teeth; the gums are spongy, painful, particularly on eating, bleed readily and may become so swollen as to hide the teeth. There is less gingivitis when there are no teeth or roots. The breath is fetid. In severe cases the gums necrose and bleed profusely; deep ulcers, on healing, leave cicatrices; the teeth may fall out and the salivary and cervical lymph glands and tongue may swell.

3. *Hemorrhages* occur (*a*) in the *skin*, especially on the extensor surfaces of the legs and first about the hair follicles; they are at first small petechiæ, but later enlarge and stretch the skin. Less frequently the first eruption may resemble acne, herpes or pemphigus, hemorrhages appearing later in these exanthems. The face is usually exempt. As the hemorrhages increase, the legs are diffusely mottled with red, green and black and are swollen and tense. Similar stains may run upward in streaks along the lymph vessels. Some of these changes are due to (*b*) muscular hemorrhages (*scorbutic sclerosis*), mostly in the calf and thigh, less in the buttocks or abdominal wall. The legs are large, extremely hard, blood-stained and later pigmented. As the disease regresses the muscles usually recover their function, but in some instances scorbutic ulcers, suppuration, muscular cicatrization or shortening of the tendons leave permanent deformity and contractures. (*c*) *Mucous membrane hemorrhages* are less common than in purpura or hemophilia, though epistaxis is fairly frequent. Sometimes hemorrhages occur in the conjunctiva, air passages, stomach, bowels, perhaps with intestinal ulceration. (*d*) Of serous membrane hemorrhages, articular hemorrhage (hemarthrosis) is the most frequent. Next in order of frequency are those in the pleura, pericardium, peritoneum and meninges. They are usually sudden in onset, extreme in degree and unfavorable in outcome. (*e*) Parenchymatous hemorrhages are least common, as in the eye, brain and other viscera. Subperiosteal and epiphyseal hemorrhages are rare, except in Barlow's disease (*v. i.*).

4. Scorbutics are subject to *various inflammations.* (*a*) Painful *joint* swellings are the most frequent (in 35 per cent. of the author's cases, chiefly in the knees); they may be serous or hemorrhagic or in severe cases the cartilages or bones may ulcerate or suppurate; deformity and ankylosis are possible sequences. (*b*) Hemorrhagic pericarditis and pleuritis are very dangerous. (*c*) Paronychia, choroiditis and panophthalmitis are occasional incidents. Fever is uncommon.

Course and Prognosis.—The onset is insidious, the course chronic and convalescence protracted. Most cases recover but death may occur from (*a*) cachexia, the autopsy showing fatty degeneration of the heart, liver and bloodvessels; (*b*) profuse hemorrhages, into the pleura, pericardium, gastro-intestinal tract or rarely into the brain; (*c*) infections, as dysentery, pleurisy, pneumonia or septicopyemia of the terminal type, promoted by reduced physiological resistance.

Diagnosis.—The diagnosis is made without difficulty by the cachexia, gingivitis, tendency to hemorrhage and inflammation, the chronic course, and the temporary nature of the condition (as contrasted with the permanent tendency to hemorrhage in hemophilia).

Treatment.—(*a*) *Hygienic;* avoidance of cured or smoked foods is both prophylactic and curative. In Nansen's polar expedition, which lasted three years, not one case of scurvy developed; cooked meats, berries, sauerkraut, potatoes, radishes, cress, apples, oranges and lemons were used. A monotonous dietary is particularly to be avoided. (*b*) *Anemia* should be treated by strychnine, iron and arsenic. Yeast ʒij, three times daily, is recommended. (*c*) The *gingivitis* is treated by local appli-

cations of silver nitrate (10 per cent.). (*d*) *Symptomatic treatment;* hemorrhage is benefited by rest and serum. Stimulation is required in intercurrent infections. Drastic cathartics are to be avoided. Complicating arthritis necessitates rest and cold, rarely aspiration or drainage.

Infantile Scurvy.—Barlow's Disease.—Möller (1852–62) described the affection as acute rhachitis, and Ingerslev, Jalland, Cheadle and Gee, but particularly Barlow (1883), described an infantile cachexia with hemorrhages. In 1898, 379 cases were collected in the United States.

Etiology.—Unlike scurvy in adults, the hygienic conditions are good in 80 per cent. of cases, and it occurs largely in the better classes. (*a*) The obvious *cause is feeding* with malted, condensed or cooked milk or baby foods; these foods lack some unknown substance essential to health. In 713 cases only 2.5 per cent. occurred in infants at the breast (S. Weiss, 1905). (*b*) Ninety per cent. of cases occur between the sixth and twelfth months (Heubner and Neumann).

Symptoms.—There may be anemia without hemorrhages, hemorrhages without anemia, or typically both together.

1. *Subperiosteal hemorrhage* is the leading symptom. The primary vague symmetrical swelling over the epiphyses later becomes more obvious, though no edema is noted. There is pain and tenderness usually in the thighs or legs, which are drawn up, flexed or everted and immobile (pseudoparalysis). The anatomical findings are subperiosteal hemorrhages; a colloid and fibrillar alteration in the marrow of the diaphyses; arrest of ossification; decrease of the osteoblasts; rarefaction of the bones, sometimes leading to fractures (spontaneously or after slight trauma) or to epiphyseolysis (usually at either end of the femur or the upper end of the tibia); the joints are normal. Seventy per cent. of the lesions are in the thigh, 24 per cent. in the leg and 6 per cent. in the orbit; the arms, ribs, spine, skull, sternum and scapulæ are seldom involved. Rib involvement results in depression of the sternum.

2. The *cachexia* is evidenced by the moderate anemia (sometimes attended by slight lymphocytosis), asthenia, anorexia, constipation, irregular temperature (in 66 per cent.) and severe sweats.

3. *Gingivitis* is noticed when the teeth have already erupted or are about to erupt; the gums are normal in but 4 per cent.

4. Other symptoms embrace hemorrhages in the skin in 50 per cent., from the bowels (10 per cent.), nose (9 per cent.), kidneys (9 per cent.) and rarely in the conjunctivæ, muscles or meninges.

Diagnosis.—The etiology, typical subperiosteal hemorrhages, gingivitis and anemia prevent confusion with genuine paralysis, for the immobility is voluntary to escape the pain of movement. Syphilitic bone disease (Parrot's disease) is similar as to the crepitus, epiphyseolysis and pain, but other syphilitic or scorbutic stigmata are decisive.

Prognosis and Treatment.—Proper treatment secures immediate improvement in the majority of cases. Death is uncommon and occurs in the third or fourth month of the disease. The diet consists of raw cow's milk, potato purée, beef-juice (not broth or extracts), orange-juice, apple sauce, pears, cooked berries and spinach. Treatment by drugs is superfluous.

Hemophilia.—Definition.—A congenital and hereditary affection, characterized by deficient coagulability of the blood and profuse hemorrhages, which are most difficult to arrest. It was named by Schönlein.

Etiology.—(*a*) *Heredity* is the only clear etiological fact (Fordyce 1784); as Grandidier (1877) remarked, hemophilia is "the most hereditary of all hereditary diseases." Brown traced hemophilia through seven generations in 200 years. Sometimes a generation may be exempt. (*b*) Broadly speaking, the *males* of the "bleeder families" are the "bleeders" and the females—the "conductors"—transmit the disease to their male offspring without manifesting the disease themselves. In 200 families of "bleeders," Grandidier found 609 cases in males (93 per cent.) and 48 in females (7 per cent.). In the Mampel family, first reported by von Chelius in 1827, Lossen (1905) found 212 members, of which 111 were males; of these exactly one-third were bleeders and 48.7 per cent. died of hemorrhage; none of the females were bleeders and all transmission of the disease occurred through the women. (*c*) The disease occurs in Germany, England, France and North America. Consanguinity, family gout, rheumatism and tuberculosis are suggested as causes. Bleeders are generally healthy individuals with blond, delicate skins and blue eyes. The factors involved in clotting are calcium and activation of prothrombin in the presence of some unknown substance (thrombokinase).

Symptoms.—In 75 per cent. of cases, *hemorrhages develop* in the *first two years of life;* rarely do they first appear after puberty, in the third decade or after the seventieth year; only 46 congenital cases are known. The hemorrhages are spontaneous or traumatic; they most often occur as capillary oozing from a mucous surface; deep interstitial bleeding is most unusual. They are copious and hard to still. Epistaxis is the most common (50 per cent.); hemorrhages from the gums (12 per cent.), intestine (12 per cent.), lungs, kidneys and stomach (each 6 per cent.) and uterus are next in frequency. Arthropathies are the rule. According to König, the joint findings are (*a*) an hemarthros, with pain, swelling and fluctuation; (*b*) then panarthritis, whose gross pathology resembles tuberculous arthritis, with flexion of the joint; (*c*) regression, or ankylosis and deformity, which rather resembles arthritis deformans. The knees and elbows are most often affected. Subcutaneous hemorrhages may occur. The first bleeding may occur from the navel or may result from vaccination, extraction of teeth, circumcision, from a trifling prick or cut, from delivery or from rupture of the hymen on the wedding night. In various attacks, one part after another may bleed or the hemophilic disposition may be confined to one part, as the kidney (renal hemophilia) or nose; in Agnew's celebrated case, the patient bled from cuts on the head but never from lesions lower. Hemophilia differs from purpura, in that bleeding seldom occurs into a serous membrane (excepting the joints) or into the viscera. The amount of blood lost may be enormous and in Litten's case amounted to 24 pounds in eleven days. Hemophilics tolerate loss of blood better than other individuals, and recover from it more readily. The coagulation time of the blood is delayed to sixty, even eighty-four minutes; the clot, when formed., is porous, but retracts

normally. The white cells are normal or slightly decreased, with relative lymphocytosis; the percentage of eosinophiles and mast cells is high.

Diagnosis.—The diagnosis may be for a time uncertain, when hemorrhage develops after an operation or occurs from one organ only, as the kidney. The family history clearly stamps the affection and readily differentiates it from scurvy, purpura and hemorrhages in the newborn. It is peculiar that patients often deny that they are bleeders. The 16 cases of multiple hereditary telangiectases with recurring hemorrhages, described by Osler, may be hemophilic.

Prognosis.—The prognosis is grave. Any hemorrhage may prove fatal. Sixty per cent. of cases die before the eighth year and only 11 per cent. reach the twenty-second year; if middle life is reached the danger gradually lessens. It is more favorable in girls, despite the apparent menstrual dangers. De Bovis (1905) found 69 excessive hemorrhages among 150 deliveries in hemophilic women; he believes pregnancy has a rather favorable effect on the disease. De Lee reported premature detachment of the placenta in hemophilia. Fränkel and Bohm found 24 mortalities in 104 hemophilics with genital hemorrhages. The rudimentary or local forms are more favorable than general hemophilia.

Treatment.—1. Prophylaxis.—Grandidier lays down the following rules: (*a*) Women, with a hemophilic family tendency, whether bleeders or not, should not marry. (*b*) Men, if not bleeders themselves, may marry, unless their family history indicates that male bleeders have begotten hemophilic children. General measures to elevate the physiological resistance and avoidance of trauma or any but life-saving operations, are indicated. Vaccination is rarely contra-indicated.

2. Control of Actual Hemorrhage.—This is almost impossible. Wounds should be cauterized or enlarged, as Fordyce discovered that in hemophilics large bleed less than small wounds. Bruising the bleeding part and dusting with thyroid extract are helpful. The Esmarch constrictor controls some hemorrhages of the extremities. Styptics as Monsell's solution or tannic preparations are useless. Adrenalin, locally and internally, has no apparent effect. Calcium lactate is more beneficial; gr. xv, t. i. d., should be given intermittently, because protracted administration decreases the coagulability of the blood. Gelatin may be given by mouth. The work of Wirth and Weil indicates that normal serum or antitoxin is of much value. Human serum or the entire blood is excellent. Saline cathartics are sometimes efficacious, possibly by diverting blood from the seat of hemorrhage. Very acute anemia necessitates injection of salines or 5 per cent. grape-sugar solution intravenously or under the skin; when the hemorrhage is over, regeneration of the blood is remarkably rapid. Joint involvement is treated by rest, immobilization and rarely aspiration.

SECTION VII.

DISEASES OF THE DUCTLESS GLANDS.

CERTAIN organs elaborate secretions, which are voided externally as it were, as the stomach, duodenum, liver, kidneys, pancreas, testes, ovaries, etc. Each of them also secretes substances which are absorbed into the blood, by which they reach other organs, as secretin (duodenum), products from the islands of Langerhans (pancreas), etc.—an internal secretion. Although all the cells or glands of the body probably produce some internal secretion, either in the katabolic process or as stimulant to the activity of other cells or organs, yet internal secretion, in its narrow acceptation, relates particularly to those ductless glands, whose sole secretion is internal, *e. g.*, the thyroid, suprarenals, parathyroids, pituitary gland and perhaps still others, as the spleen, thymus or lymph nodes. The correlation of the ductless glands is effected by hormones, acting through the circulation. In various combinations, these internal secretions supplement or antagonize each other. The subject, aside from certain well-defined clinical syndromes, is still in its infancy.

DISEASES OF THE SUPRARENAL GLANDS.

ADDISON'S DISEASE. OTHER AFFECTIONS OF THE SUPRARENAL GLANDS.

Definition.—A hypoepinephry characterized pathologically by lesions (usually tuberculous) in the adrenal glands or chromaffin system elsewhere, and clinically by muscular, vascular and mental adynamia, digestive disorders, and pigmentation of the skin.

Etiology.—Suprarenal disease was first associated pathologically with melanoderma by Addison in 1855. By far the most common lesion is (*a*) chronic fibrocaseous *tuberculosis* of the adrenal glands; tubercles of the adrenal glands are usually bilateral, destroy most of their structure and frequently involve the semilunar ganglia and solar plexus by the attendant formation of connective tissue; there is a primary focus in the lungs in over 50 per cent., in the genito-urinary, alimentary or glandular system; in rare cases only is the adrenal tuberculosis primary. (*b*) Less common are tumors, atrophy, fibrous induration (usually tuberculous), gumma, amyloid degeneration, acute inflammation, hemorrhage,

embolism, echinococcus, adenoma or other very rare lesions. Addison's *melasma suprarenale* is a disease in which the chromaffin cells in the medulla of the adrenals or the sympathetic ganglia waste (Weisel). The adrenal cortex contains epithelial structures, and the medulla consists of non-medullated nerve fibers and large mononuclear cells; these latter chromaffin cells are also found in the sympathetic ganglia, along the aorta in Zuckerkandl's bodies, the carotid glands, and perhaps also in the coccygeal gland, etc. Epinephrin is the active glandular substance of the chromaffin system; it stimulates the skeletal muscles, controls pigmentation, maintains vascular tonus and stimulates the vasomotor centres, the vagus and respiration; when secreted in excess, as in hypernephroma, the blood tension may be much elevated (Fränkel, Neusser, Kolisko) and this may induce cerebral hemorrhage. The cortex is concerned in sexual activity and hypertrophies in pregnancy; it is hyperplastic in sexual precocity (hirsutism; virilism) and hypoplastic in infantilism. The glands elaborate an internal secretion necessary to life which renders less toxic substances produced by other tissues, *e. g.*, the "fatigue stuffs" of muscle metabolism.

In Lewin's statistics from 500 cases the adrenals were diseased in 88 and sound in 12 per cent.; on the other hand, disease of the adrenals is attended by pigmentation of the skin in 72 per cent. and unattended by it in 28 per cent.; in adrenal tuberculosis without Addison's disease, there is no general disturbance of the chromaffin system and in Addison's disease with no adrenal tuberculosis, the chromaffin cells elsewhere are affected.

Most cases occur between the fifteenth and fortieth years, 66 per cent. in males and 90 per cent. in the laboring classes.

Symptoms.—1. Adynamia.—Marked *physical and mental adynamia* is usually the initial symptom and persists till death. The insidious languor, weakness, apathy, lack of initiative and muscular relaxation contrast sharply with the healthy appearance of the patient in the early stages. These symptoms are toxemic, partly from hypoepinephry and partly from the tuberculous lesion. Obstinate early insomnia and headache and later attacks of vertigo, tinnitus, spots before the eyes and syncope are frequent. The intellectual functions are weakened, periodic irritability is frequent and the deep reflexes are somewhat reduced.

2. Digestive Symptoms.—Digestive symptoms generally appear next. The tongue is coated; there are a sense of abdominal fulness, eructations and sensitiveness over the abdomen. *Paroxysmal pains* in the epigastrium or back radiate widely, especially into the chest, like severe tabetic crises. *Vomiting* is the most distressing, ominous and intractable alimentary symptom; it may occur independently of eating. At first intermittent, it later hastens the fatal issue. Early constipation gives way to terminal diarrhea.

3. Pigmentation.—Melanoderma, bronzed skin, *melasma suprarenale* develops after the adynamia and alimentary disturbance. It is found in the deep layers of the rete Malpighii, to which it is apparently brought from the blood by leukocytes. At first the *skin* has a dirty, yellowish-gray color and small areas appear which gradually fuse and then

slowly become darker and more diffuse; extensive pigmentation may occur with focal areas of deeper discoloration. Pigmentation develops particularly (*a*) where it is normally deepest, as about the nipples, anus and genitalia; (*b*) where pressure or friction is greatest, as over folds of the skin, over the clavicles, spine, ischia, scapulæ or waist (from belts or corsets); (*c*) where there is exposure to the sun, as in the face, neck or arms; (*d*) where accidental irritation occurs, as by blistering, eczema or parasitic affections. The soles, palms and nail-beds are seldom stained. Previous scars may be surrounded by a zone of discoloration. The skin is dry, yet sometimes bathed with profuse perspiration and may have a fish-like odor. Scattered spots of pigment may atrophy (vitiligo) and impart a strange appearance. Drawing a sharp object across the skin may elicit a "white line," which runs parallel with the cardiac weakness (Sargent). The *mucous membrane* of the lips, gums and palate shows pigmentation later than the skin. The horizontal ink-like stains on the cheek where the teeth touch it are particularly diagnostic. The conjunctivæ are pearly white, though frequently small aggregations of pigment can be seen on careful examination. Staining of the larynx, nymphæ, vagina, glans penis, serous membranes and internal organs is infrequent.

4. Other Findings: (*a*) The *heart* shares in the general adynamia; its tones and apex are weak; it frequently shows brown atrophy at autopsy. Pain over the heart is reflected from the abdomen; palpitation and dyspnea are common, but functional murmurs are rare. The pulse is compressible and somewhat accelerated. The blood-pressure is generally low. The peripheral vessels and abdominal aorta pulsate actively. Atheroma is common, even in young subjects. (*b*) *Anemia* was considered characteristic by Addison, though Nothnagel demonstrated its infrequency; in one instance the writer observed a red-cell count of 2,000,000. Increase in the red cells (polycythemia rubra) and in the hemoglobin results from concentration of the blood, due to vomiting and diarrhea. Lymphocytosis is ominous. (*c*) The *lymph nodes,* Peyer's patches, spleen, thyroid, thymus and pineal glands are often enlarged, probably vicariously. (*d*) *Emaciation* and *cachexia* are terminal conditions. Edema of the ankles is infrequent even toward the end. Characteristic metabolic changes are not found; acetonuria is occasional and terminal albuminuria may develop. The temperature is most often normal or subnormal and a subjective chilliness is experienced.

Clinical Course.—The cardinal symptoms progress slowly, with occasional intermissions, and in nearly all cases death results within two years; the terminal symptoms are often violent, viz., delirium, convulsions, incoercible vomiting and diarrhea, small pupils, pseudomeningitic or peritonitic symptoms and finally collapse and coma. Variations include: (*a*) *Acute cases* resembling typhoid, or acute poisoning; (*b*) *chronic cases* lasting ten to thirteen years; (*c*) *sudden death* before pigmentation develops; (*d*) the course modified by *pulmonary* or intestinal *tuberculosis.*

Diagnosis.—Diagnosis is based on the prostration, cardiac, psychical and physical; digestive disorders, vomiting, diarrhea and neuralgia; pigmentation; and the chronic lethal course.

A tuberculin reaction may occur from tuberculosis in other tissues. Adrenal tumors may cause metastases in the brain or (like tumors of the thyroid, prostate and mammæ) in the long bones, skull and spine. According to Grünbaum adrenal extract, gr. iij, t. i. d., for three days raises the blood-pressure 10 mm. if adrenal insufficiency exists.

DIFFERENTIATION.—*Pigmentation* also occurs in (*a*) poisoning by silver (*argyria*), in which the reduced metal forms a gray deposit in the extracellular tissue of the skin, sweat glands, conjunctivæ and nail-beds; by *arsenic*, which seldom stains the mucosæ; and in *pellagra*, which is exempt from mucous membrane pigmentation. (*b*) Chronic *icterus*, *cyanosis* (sometimes pigmenting the oral mucosa), various physiological pigmentations, dirt and parasites, as in tramps (vagabondism). (*c*) *Abdominal diseases;* dyspepsia, gastric ulcer, tumor (cancer, lymphomata and Hodgkin's disease), hypertrophic cirrhosis and hemochromatosis (hepatic and pancreatic cirrhosis, diabetes and skin pigmentation, *diabéte bronzé*). (*d*) *Pelvic conditions;* pregnancy, uterine and ovarian tumors (after the removal of which pigmentation may regress). (*e*) Phthisis, malaria, melanosarcoma of the skin, or exophthalmic goitre. In these conditions, pigmentation of the oral mucosa is rare.

Treatment.—1. SUPPORTING.—Absolute rest is imperative because of exhaustion and proneness to lethal syncope. Alcoholic stimulation is poorly tolerated. Careful feeding is necessary because of the sensitiveness of the stomach and bowels.

2. SYMPTOMATIC.—Narcotics for pain, gastric sedatives for vomiting (*v.* GASTRITIS), opiates for diarrhea (*v.* ENTERITIS) should be administered; strong cathartics are always contra-indicated; one case in the Cook County Hospital died suddenly after taking a dram of compound jalap powder.

3. ORGANOTHERAPY.—Of Adam's 105 cases treated by adrenal extract by mouth, one-third were benefited and one-sixth recovered. The medullary portion of the gland is best. It may increase the vascular and muscular vigor or even lessen pigmentation. Some advocate adrenalin chloride, ℥j of 1 to 1000 hypodermically on alternate days. Some advanced cases are unfavorably affected.

Other affections of the suprarenal glands are of slight clinical interest. (*a*) *Developmentally* the glands are sometimes absent or hypoplastic, chiefly with other defects, as encephalocele. (*b*) *Hypertrophy* in one gland may compensate for hypoplasia in the other. *Supernumerary* glands are not uncommon. (*c*) *Atrophy* is usual in advanced age. (*d*) *Degenerations* embrace the parenchymatous, fatty, hyaline and amyloid types. (*e*) *Circulatory disorders* are embolism, thrombosis and hemorrhage which latter may occur in trauma in the newborn, tumors, inflammation and the hemorrhagic diathesis. (*f*) *Inflammation*, acute and chronic, is uncommon. (*g*) *Granulomata*, chiefly syphilitic and tuberculous, are rarely primary. (*h*) *Tumors, e. g.*, carcinoma and sarcoma, are chiefly of anatomical importance. In 23 operations on hypernephroma the mortality was 50 per cent., recurrence developed in 21 per cent. and 29 per cent. recovered. (*i*) Echinococcus is extremely infrequent.

DISEASES OF THE SPLEEN.

STRUCTURALLY and physiologically, the spleen resembles the lymph nodes, which vicariously hypertrophy after splenectomy. It is not proved indispensable to life nor productive of an internal secretion. During fetal and perhaps adult life it is concerned in erythrocytogenesis. In health it destroys old red blood cells and in certain diseases its hemolytic activity is pronounced. In immunization, phagocytosis and perhaps some chemical factors are conspicuous.

ACUTE SPLENIC TUMOR.

This occurs (*a*) in *acute infections,* due to the local action of micro-organisms and their toxins or to toxemic paralysis of the splanchnic nerve; it is most common in typhoid, malaria, recurrent fever and septic conditions; (*b*) in *acute,* usually portal *stasis;* and (*c*) in *embolism* (*v. i.*). *Symptoms* are infrequent; pain usually indicates perisplenitis. On objective examination: (*a*) The spleen may sometimes be seen during deep inspiration. (*b*) Palpation alone is reliable; by the bimanual method and with the examiner sitting at the right side of the patient the edge of the spleen comes in contact with the fingers and slips sharply down below them; splenic crenations are seldom palpable. A palpable spleen indicates enlargement or less often, downward dislocation by pleurisy, pneumothorax or thoracic tumor. (*c*) Percussion, valuable in outlining the upper splenic level, is unreliable in determining the lower border.

CHRONIC SPLENIC TUMOR.

Chronic enlargement is symptomatic of (*a*) *chronic infections,* as malaria, tuberculosis and syphilis, (*b*) *continued portal stasis,* (*c*) *repeated embolism,* (*d*) *blood affections,* as leukemia, pseudoleukemia or splenic anemia, and (*e*) amyloidosis, neoplasms, cysts, polycythemia, arthritis deformans, chronic acetanilide poisoning, etc.

EMBOLISM AND ABSCESS.

Embolism of the spleen ranks second in frequency to kidney embolism because of its wide arteries and slow current. It usually develops in the course of acute endocarditis or chronic valvular lesions and seldom follows local lesions, as splenic vein thrombosis. The *symptoms* vary as the embolus is simple or infective; sudden chill, fever, pain in the side, acute splenic tumor and perisplenitic friction may develop, which with a cause or embolic symptoms in other organs, justify a diagnosis of embolism. Frequently no distinctive symptoms occur. Treatment is symptomatic.

Abscess from embolism, trauma or invasion by contiguity, occurred in 9 per cent. of 430 cases of pyemia (Paget). It is largest in non-embolic cases; embolic abscesses are small and multiple. Symptoms are: (*a*) those of the cause, which may mask the lesion; (*b*) of embolism; (*c*)

of splenic tumor, perisplenitis, friction or pain; (*d*) of rupture into the pleura, kidney or peritoneum; (*e*) aspiration determines the diagnosis and treatment is surgical evacuation.

PERISPLENITIS.

This affection may be primary or secondary, simple or suppurative, an isolated lesion or part of a chronic peritonitis (see "Icing Liver," Chronic Perihepatitis). It usually accompanies the various splenic affections enumerated. Without local peritonitic friction, pain or tenderness its diagnosis is impossible. There is no therapy.

AMYLOID SPLEEN.

Its etiology is considered under Amyloid Liver and Kidney. Pathologically, its two types are (*a*) the sago spleen, in which there are amyloid deposits in the vessels of the Malpighian tufts, and (*b*) the more important, diffuse amyloid infiltration, diagnosticated by (i) its etiology, (ii) large size, hardness, smoothness and thick edge, and (iii) coincident amyloidosis of the liver and kidneys.

RUPTURE OF THE SPLEEN.

Rupture may be traumatic or spontaneous; the latter variety has been observed in embolism, malaria, typhoid, leukemia and pregnancy. It is more common in tropical than temperate climates. Aspiration and even palpation have occasioned rupture. The treatment is surgical; 104 splenectomies for rupture are reported, with 29 per cent. mortality (Hörtz).

MOVABLE OR FLOATING SPLEEN.

Lien mobile is due (*a*) to the same factors observed in enteroptosis, with which it may be associated, and (*b*) enlargement and increased weight of the spleen.

Symptoms.—Symptoms are often absent. Pain or a dragging sensation is frequent, which is either local or radiates to the thorax, left shoulder or legs, similar to that observed in neurotics or women with pelvic disease. There may be headache, psychical alteration, dyspepsia, menstrual anomalies and paresthesias. *Objectively:* (*a*) The most important sign is *palpatory detection of the spleen in an abnormal location*, most often in the left ilio-inguinal region but also below the left costal arch, near the navel, in a hernial sac, in the pelvis or above the pubes. Its hilum usually points upward. The form, notches and possibly the pulsating vessels are those of the spleen. It is usually enlarged, from causal hypertrophy or later congestion; torsion of its pedicle may lead to atrophy, softening, gangrene or even liberation of the spleen as a free body. As a rule it can be replaced and its position changes with posture; perisplenitis, causing a friction-rub, may result in firm adhesions to the colon, bladder or uterus, thereby causing insuperable diagnostic difficulty;

in such cases the spleen ceases to "wander." (*b*) The *spleen is absent from its normal location*, the splenic area being replaced by tympany from the colon (which changes to dulness when water is introduced into the colon). (*c*) *Pressure symptoms* include intestinal, pyloric, vesical and rectal compression.

Diagnosis.—The author knows of three laparotomies performed for supposed uterine or renal disease in which abundant adhesions rendered differentiation impossible. On the other hand, a hard, immovable suprapubic tumor was correctly diagnosticated as leukemic spleen by the adenopathy and blood findings.

Treatment.—(*a*) *Bandages* are difficult to apply, though sometimes of benefit. (*b*) The spleen may be replaced by operation and well packed around with gauze; the resulting adhesions often retain the viscus. (*c*) *Splenopexis* (Rydygier) may be performed, the spleen being sewed in place or deposited in a peritoneal pocket. (*d*) *Splenectomy* may be performed as a last resort.

PRIMARY SPLENOMEGALY.

Reference has been made to Banti's disease and splenic anemia (pp. 571 and 690), splenomegaly with acholuric jaundice (p. 590), pernicious anemia, hepatic cirrhosis, etc.

Gaucher's splenomegaly (*primary endothelioma*), of which 16 cases are reported, is characterized anatomically by peculiar large, round and multinucleated cells in the hemopoietic system (marrow, spleen, lymphadenoid structures, liver). Its cause is disputed (malignancy, endogenous, splenic toxemia, etc.). Besides the splenic tumor, the clinical symptoms are a yellowish-brown discoloration of the skin, conjunctival thickening, persistent leukopenia, mild anemia, hemorrhagic tendencies, development early in life, chronic course and familial incidence. As in Banti's disease, splenectomy is indicated.

Tumors and Cysts.—New growths are very infrequent. They are more often secondary than primary. They include carcinoma, sarcoma, enchondroma, fibroma and lymphangioma. They are rarely differentiated from splenic anemia and like affections.

Echinococcus cysts (*q. v.*) are usually found with concomitant cysts in the liver. One dermoid cyst is reported (Andral, 1829). Only 38 non-parasitic cysts are found in the literature.

Granulomata (see Tuberculosis and Syphilis).

DISEASES OF THE THYROID GLAND.

GOITRE.

Definition.—Hypertrophy of the thyroid gland. Sporadic cases are common in Europe and America. Goitre occurs endemically in Switzerland and Italy, from the drinking water. It is usually acquired and is seldom congenital.

There are *three main forms:* (*a*) The *parenchymatous* type, with formation of colloid material in the hyperplastic follicles; (*b*) the *cystic;* and (*c*) *vascular* type, with dilatation of the bloodvessels, which form borders closely on those physiological variations observed at puberty, in defloration, pregnancy, chlorosis and psychical trauma.

Symptoms.—1. The *thyroid enlargement* may be diffuse, as in the soft, symmetrical, vascular form, or partial or nodose, implicating but one lobe or the isthmus. In most cases there are only local findings.

2. *Compression* symptoms are not common. The carotid artery and jugular vein are pushed to one side and the sternomastoids become thin. The tracheal lumen may be shaped like a sword scabbard; dyspnea, bronchiectasis and dilatation of the right ventricle follow stenosis of the air passages. Laryngeal paralysis occurs in over 10 per cent., and in 25 per cent. of cases in which there is tracheal stenosis. Goitres may grow in a retrosternal or retroclavicular direction (5 to 10 per cent.), compressing the vagus, sympathetic nerves and vena cava. A goitre may disappear into the chest with each inspiration (*goitre en dedans*), or become incarcerated there.

3. An *aberrant or accessory thyroid* may occasionally form a tumor anywhere from the tongue to the aorta, from the spine to the anterior chest wall, in the retropharyngeal or retro-esophageal tissues (causing dysphagia or dysarthria), in the trachea, larynx, root of the tongue, pleura or mediastinum; it may compress an entire lung; 75 benign, 16 malignant and 3 cystic and calcarious growths are recorded in retrosternal accessory thyroids; there are in the literature 14 intratracheal accessory thyroids.

4. *Sudden death* may occur from pressure on the vagus or trachea, spasm or edema of the glottis, sudden hyperemia of the gland, hemorrhage dissecting into the chest or status lymphaticus.

Diagnosis.—Confusion is possible with *tumors,* as carcinoma, adenoma, echinococcus cysts (25 reported cases) and sarcoma (100 reports). In 83 per cent. of cases *cancer* develops from a preëxisting goitre; early metastases are especially common and characteristic; only 10 per cent. recovered among 110 operations (Madelung). In 20 cases recorded, *metastases* throughout the body occurred *from benign tumors* of the thyroid (chondroma or myxoma). Thyroid inflammation (*strumitis*) is usually metastatic or traumatic, rarely primary; it may occur in sound or goitrous glands; it is most often due to the streptococcus but may be caused by the colon, typhoid, pneumonia or other organisms.

Treatment.—In regions where goitre is prevalent the drinking water should be boiled. *Medical treatment* is of uncertain value. Iodine may be used by injection (Maunoir, 1825). Potassium iodide (Coindet, 1820) is beneficial in some subacute cases, according to Bruns, in 75 per cent.; Kraus found it particularly beneficial in the vascular type, with moderate tachycardia and full arteries. The *x*-rays and thyroid extract are occasionally beneficial. *Surgical treatment* is indicated in large or growing goitres; in Kocher's 5000 cases, 1 death occurred in each 600 operations.

EXOPHTHALMIC GOITRE (HYPERTHYROIDISM).

Definition.—A malady due to excessive activity of the thyroid gland and characterized by exophthalmos, enlarged thyroid, tachycardia and tremor. It was first described in England by Parry (1786) and Graves (1835) and later in Germany by Basedow (1840).

Etiology.—1. The cause is *overactivity or perversion of thyroid secretion* (Möbius) which contrasts sharply with myxedema:

	Exophthalmic Goitre. ——*vs.*——	Myxedema.
(*a*)	Hypertrophy of gland; excessive internal secretion (hyperthyrea).	Atrophy; deficient (hypothyrea) or absent secretion (athyrea).
(*b*)	Increased nervous excitability.	Dulness, apathy, cretinism.
(*c*)	Vascular erythrism; flushed, moist, warm skin; rapid pulse.	Skin dry, thick and cool, and pulse slow.
(*d*)	Increased diuresis and active metabolism.	Decreased.
(*e*)	Thyroid extract increases symptoms; it may produce goitre, tachycardia, tremor, even exophthalmos.	Thyroid extract improves or cures.
(*f*)	Surgical (partial) excision relieves, and often cures.	Some cases result from removal of thyroid. Transplantation of new gland improves or cures.

(*g*) Exophthalmic goitre (glandular hypertrophy with hyperthyroidism) may occasionally become myxedema (hypothyroidism from glandular atrophy).

2. *Other etiological explanations* are less plausible, as vasomotor neurosis or lesions in the corpus restiforme of the medulla.

3. *Exciting factors* are: (*a*) *Sex;* 80–95 per cent. are women; fibroids, pregnancy and puberty are sometimes promoting factors. (*b*) *Age;* most cases occur between the twentieth and thirtieth years. Barrett (1904) could collect only 42 cases in persons under fifteen years. (*c*) *Psychical* trauma, as fear, and concussion, anemia, phthisis, neuropathic family tendencies, other neuroses or psychoses favor its development. (*d*) After thyroidectomy postoperative hyperthyroidism is not rare.

Symptoms.—1. Cardinal Symptoms.—(*a*) *Tachycardia* is the earliest symptom. The pulse ranges from 100 to 120 (140 or even 200); it varies from time to time. It is associated with other cardiovascular symptoms, as (i) increased cardiac action, manifested by a diffuse cardiac impulse, sharp tones sometimes heard at a distance of several feet and hypertrophy of the left ventricle eventuating in dilatation; (ii) a functional systolic murmur over the apex; (iii) palpitation, usually antedating tachycardia; (iv) vascular symptoms; a pulse weaker than the apex-beat; active arterial pulsation in the carotids and abdominal aorta; capillary pulsation in the fingers, forehead or even in the retina, palate, liver and spleen; even the peripheral veins may pulsate; and (v) irregular failing heart action occurs late and in severe cases. Hemorrhages are not uncommon.

(*b*) *Exophthalmos,* usually a later symptom than tachycardia, is present in 90 per cent. of cases and results from dilatation of the orbital vessels. The protrusion of the eyeballs and the staring, fixed expression are very

characteristic (exophthalmos may also occur in extreme myopia, tumors of the orbit, nephritis, and sympathetic irritation). The condition is generally bilateral, though often unequal; the author saw unilateral exophthalmos with homolateral goitre. The eyelids are unable to cover the eye-ball entirely and a rim of the white sclera shows about the iris. The protrusion may even amount to dislocation of the bulb, perhaps with corneal ulceration and loss of the eye by panophthalmitis. The *associated eye-symptoms* are: (i) *von Graefe's sign*, the failure of the upper lid to follow the eye-ball when it is rolled downward, is present in at least 33 per cent. and is of considerable diagnostic value, though present also in Thomsen's disease. (ii) *Stellwag's sign*, which is rarely absent, is widening of the interpalpebral fissure (Dalrymple) with infrequent winking (Stellwag); it also occurs in mania and in old age. (iii) *Möbius's sign* is weakness of the internal recti, when the eyes converge; it occurs also in other conditions, as paretic dementia. (iv) *Becker's sign* consists of pulsation of the retinal vessels. (v) In *Joffroy's sign* the patient's forehead remains smooth (instead of becoming wrinkled) when he glances upward with the head bent somewhat downward. (vi) *Gifford's sign* is the difficulty in everting the upper lid; (vii) Jellinek and Rosin describe edema and pigmentation of the upper lid. Kocher noted a spasm of the lid in eliciting v. Graefe's sign. Eye-muscle paralyses and retinal or pupillary changes are infrequent.

(*c*) *The goitre* is rarely an initial symptom and usually develops later and remittently. It is present in 100 per cent. (i) It typically is *vascular*. Acute thyroidism may develop in thyroiditis, chronic cystic goitre or thyroid malignancy. The goitre is *smaller* than the ordinary bronchocele; it is diffuse or oftener more developed on the right side (an accentuation of physiological proportions), shows a vascular, rough surface and is dry, gray and granular on section (instead of the normal amber-red color); microscopically the alveoli are not rounded, full of colloid matter or lined with cubical epithelium, but are extremely irregular in size and form, ramifying and encroached upon by epithelial proliferations and the epithelium becomes columnar; in extreme cases there is epithelial desquamation; the scanty colloid matter stains faintly and lymphoid tissue develops; in the later stages some fibrous tissue may develop. (ii) It is usually *soft, elastic, variable* in size, *pulsating* on inspection, *thrilling* on palpation and revealing on auscultation a loud, systolic bruit or a double murmur, which Guttmann considers pathognomonic. The goitre is retrosternal in 4 per cent.

(*d*) *Tremor* is present to some degree in most cases and is conspicuous in 50 per cent. It is characteristically fine, regular and rapid, numbering 8 or 9 vibrations to the second. It affects the wrist more than the fingers; sometimes it affects the trunk.

2. Accessory Symptoms.—(*a*) *Nervous* symptoms. *Cerebral symptoms* are usual, as headache, impaired memory, insomnia, irritability or depression. Mania is sometimes observed in the fatal cases. "Giving way of the legs" and slight inspiratory increase in the chest measurement (Bryson's sign) are expressions of *weakness of the muscles*, whose strength is reduced to one-third or one-fifth of normal (Müller). The reflexes are

often increased. Rheumatic pains may be noted. *Vasomotor symptoms* are common, as reddening of the face, subjective warmth, dry mouth, sweats of a peculiar, penetrating odor, or intermittent swelling of the joints. (*b*) *Respiratory* symptoms include dyspnea and dry cough, perhaps simulating phthisis. (*c*) *Digestive* symptoms: Severe nervous vomiting, abdominal crises and diarrhea may simulate organic disease; their persistence is ominous. (*d*) *Cutaneous* symptoms: Pigmentation may be extreme. Vasomotor edema, urticaria, itching and falling of the hair may also occur. A peculiar fulness above the clavicles may develop, sometimes with pain in the neck. The electrical resistance of the skin is decreased, perhaps to one-fifth of the normal; it is probably occasioned by profuse sweating and has no pathognomonic importance. (*e*) *Constitutional* symptoms: Emaciation and anemia are common, especially in blondes. Metabolism is increased, evidenced by the nitrogenous excretion, increased diuresis and elevation of temperature; these symptoms may be intermittent. Thompson describes fever and symptoms resembling ulcerative endocarditis. Amenorrhea, albuminuria, glycosuria, persistence of the thymus gland (in 80 per cent.) and the occasional hyperplasia of the spleen are difficult to explain. The lymphocytes and eosinophiles are increased, leukopenia is frequent and coagulation is slowed.

Diagnosis.—The diagnosis is unequivocal when the cardinal symptoms are present, reinforced by the secondary symptoms. Atypical forms (*formes frustes*) may cause confusion. The affection is often incorrectly diagnosticated in young girls at puberty, in whom thyroid turgescence, moderate tachycardia and vascular excitability occur without particular significance. Incipient tuberculosis, carcinosis and neurasthenia may be simulated.

Course and Prognosis.—(*a*) The onset is usually insidious and the course chronic and remittent. Chronic cases sometimes begin rather acutely. Acute cases are unusual but usually fatal; they occur chiefly in men; Lloyd records a case which was fatal in three days. The author has seen 3 cases in which death occurred in two weeks. (*b*) *Death* occurs in 12 per cent. of cases, commonly due to cardiac failure. (*c*) The outlook is best in the less typical (rudimentary) forms. (*d*) Complete recovery is rare, though 20 to 50 per cent. of cases improve. (*e*) Unfavorable prognostics are its occurrence in the very young and in men, emaciation, extreme tachycardia, persistent vomiting or diarrhea, fever, lymphatism and intercurrent myxedema.

Treatment.—1. General.—*Rest* and a quiet life at a moderate elevation or at the sea-side are very beneficial. Travel, sojourn at resorts, gymnastics and excitement are injurious. A simple diet and interdiction of alcohol, coffee, tea and tobacco are essential.

2. Drugs.—Drugs are disappointing; digitalis, strophanthus and spartein fail to slow the heart. Belladonna, in large physiological doses, is recommended by Gowers; the annoying palpitation and abdominal throbbing are most relieved by it. Ergotin, quinine, salicylates, arsenic and strychnine are of uncertain value, but the latter seems rather more beneficial; iron is useful only in chlorotic girls but in men it aggravates

digestive and vasomotor disturbances. Constant application of an ice-bag to the heart relieves palpitation and somewhat slows the heart rate.

3. Electricity.—Galvanization is recommended by Erb, who uses the anode over the cervical spine and the kathode over various peripheral areas. X-rays are beneficial but render operation difficult by the adhesions produced.

4. Organotherapy.—Thyroid extract and iodides aggravate the symptoms. Thymus extract is uncertain. Adrenalin clysters may check diarrhea. Baumann demonstrated that the thyroid gland contained an iodine body which Ross proved was the main functional constituent of the organ. The active element neutralizes toxins (failing in which myxedema results), and if not itself neutralized, Basedow's disease results; to neutralize hyperthyroidism serum of thyroidectomized animals was suggested.

5. Thyroidectomy.—In Kocher's 371 operations, only 3.5 per cent. died; 2 cases died in the last 153 operations; 83 per cent. were cured. Kocher considers (*a*) the blood-pressure, size of the heart and tachycardia; (*b*) he hesitates to operate if there is great nervousness, intoxication or absence of lymphocytosis, and (*c*) if the gland is very large, pulsating or vascular. Sudden operative death has been ascribed to the narcosis, flooding of the blood with thyroid products or enlarged thymus (which Capelle found in four-fifths of 60 autopsies). The mortality is much lower when Graves's disease develops from an old goitre (secondary form). The Mayos prefer local anesthesia. Kocher prefers trying the *x*-rays first and then ligating the thyroid arteries (after which tetany may occur) and later performing partial thyroidectomy; the danger of a "heart" death is avoided by early operation. The mortality of operation, for any indication, in an exophthalmic subject, is increased 15 per cent.

MYXEDEMA.

Definition.—A chronic disease, characterized by (*a*) loss of thyroid function (athyrea), (*b*) myxedema (an edematous change in the subcutaneous tissue), (*c*) cachexia or (*d*) cretinism (mental failure). It was described by Gull (1873), Ord (1877), Charcot, Bourneville and d'Olier.

Etiology.—The thyroid may be aplastic, hypoplastic, cirrhotic from an infective sclerosing thyroiditis (syphilis, tuberculosis, rheumatism or erysipelas), hypertrophied (goitre) or removed by operation, but in all cases the thyroid function (internal secretion or antitoxic action) is suspended or lost. The symptoms vary as to cause and combination, whence (i) cretinism, (ii) adult myxedema and (iii) operative myxedema will be considered separately.

CRETINISM.

Cretinism may be congenital, in which instance life is not long protracted; it usually develops near the time of weaning (infantile form); it may develop between the fourth year and puberty (juvenile form). The endemic form occurs in regions where goitre is prevalent, in Switzer-

land, Italy, France, Great Britain, Spain and Sweden; when both parents are goitrous, the child is invariably a cretin, is myxedematous and may also have goitre. The sporadic form may develop from thyroid aplasia, sclerosing thyroiditis or from goitre. In 1905, 100 cases were collected in America (Howard).

Symptoms.—In the last part of the first or in the second year of life, retardation of mental and physical development is apparent. The head is disproportionately large, the fontanelles persist, the forehead is narrow and the base of the skull is shortened. The face is turgid, pale and imbecile, the lids are swollen, as are the nose, the protruding tongue and pouting lips. Dentition is retarded and caries is frequent. The skin loses its tone and is dry, and the hair is thin. The neck is short, which condition is accentuated by supraclavicular deposits of fat. The abdomen is large and pendulous; umbilical hernia is very frequent. The body is dwarfed and the extremities are short, stumpy and weak or helpless. Metabolism is slow, the gaseous interchange being reduced to 60 to 50 per cent. of the normal.

Diagnosis.—(*a*) The mental alteration is marked, though mild compared with the grimaces, grinding of the teeth and compulsory movements caused by *gross lesions of the brain.* (*b*) In hyperplasia of the bone cartilages (*chondrodystrophia fœtalis*) there are great enlargement of the head and joints, dwarfing of the body and shortness of the extremities (micromelia), but no essential mental change; court fools were probably of this type. (*c*) *Infantilism* (*v. i.*).

MYXEDEMA OF ADULTS.

Symptoms.—Myxedema spontaneum adultorum, or *cachexie pachydermique* (Charcot) is characterized by (1) thyroid atrophy, (2) myxedema (pachydermia), (3) intellectual and physical weakness and (4) certain accessory symptoms. It is most frequent in England and France. Most cases occur between thirty and fifty years of age.

1. *Thyroid Atrophy.*—This is less a clinical than a histological condition, as evidenced by the lack of function sometimes observed in gross enlargement of the gland (one-seventh of the cases). Eighty-six per cent. occur in women and it is possibly related to the uterine functions or to the physiological congestive, thyroid enlargement observed in women. It may be hereditary or familial, perhaps associated with exophthalmic goitre.

2. Myxedema.—Mucin fibrils and nuclei—like granulation tissue—have been found in the skin and subcutaneous tissues. These structures are inelastic, semigelatinous and semiedematous, but do not pit upon pressure; the skin is pale, waxy, dry, rough, firm and its electrical resistance is increased; the hair and teeth may fall out and the nails thicken and break. The loss of the outer half of the eyebrow is described by French writers. The facies is somnolent, coarse and swollen, and the features are individually changed, rather resembling those of a cretin; the cheeks are patchy red, pendulous and tremble like jelly and the

tongue is big. The supraclavicular tissue is enlarged in lumps and rolls, though this is also observed in healthy persons (Verneuil's pseudolipomata). S. Kuh observed a painful swelling over the clavicle alone, which rather resembled keloid. The hands and feet are infiltrated. The swellings may change rapidly. The oral mucous membrane is dry and thick, sometimes also the pharynx, larynx and even the rectum.

3. Mental Weakness.—Intellectual and physical weakness is apparent. Charcot compares the somnolence to hibernation. Headache is common. The patient is apathetic, irritable, weak in memory and slow in thought, speech and movement; sometimes delirium and mania precede the ultimate dementia. The gait is clumsy ("hippopotamus gait") and muscular fatigue follows moderate exertion.

4. Other Symptoms.—The accessory symptoms are variable, as arteriosclerosis, palpitation; small, sluggish pupils, tremor, vertigo, night terrors, sensitiveness to cold; indigestion and constipation; anemia, leukopenia, lymphocytosis and rapid coagulation; amenorrhea; scanty urine with little urea and low specific gravity, sometimes albuminuria and less often glycosuria; hemorrhages, especially from the uterus; and subnormal temperature. The thymus is usually enlarged, sometimes the hypophysis and adrenals.

Course.—The course is chronically progressive and covers ten to twenty years. Remissions may occur in warm weather, by a change to a milder climate or during pregnancy. In the last stage, the myxedema often disappears. Transient cases of acute myxedema have been observed. Osler reported an acute case with melena, mania, glycosuria, tachycardia and death in six months. Sudden death is occasional, but most patients die of intercurrent tuberculosis or acute infections.

CACHEXIA THYREOPRIVA OR OPERATIVE MYXEDEMA.

Horsley reported myxedema, apathy and coma in monkeys after complete thyroidectomy; when kept warm the monkeys did not manifest myxedema, but a species of cretinism. Cachexia follows extirpation in man and herbivora; convulsions develop in carnivora, since the antitoxic elements of the thyroid are not present to neutralize the nuclein of ingested meat. The toxemia strumipriva is accompanied by anemia, leukocytosis and degeneration in the bloodvessels. Kocher and Reverdin observed cachexia strumipriva in 70 per cent. of cases, more often after the complete than after the partial operation, and von Eiselsberg reported it in about 23 per cent. of complete excisions. Accessory thyroids (*v.* Goitre) may avert this operative complication. The author knows of two instances in which removal of an accessory lingual thyroid (under a mistaken diagnosis) caused myxedema. Schiff, von Eiselsberg, and Bircher cured operative myxedema by transplantation of a thyroid. Unfortunately, the function of the transplanted gland is seldom maintained.

Treatment of Myxedema, Cretinism and Cachexia Thyreopriva.—For these allied conditions we possess in thyroid extract (Glandulæ

thyroideæ siccæ) one of the few actual specifics of medicine; and most cases can be cured—in a sense we seldom employ this term. Murray first used thyroid extract hypodermically and his first case is alive, after twenty-five years; the equally successful administration by mouth was advocated by Horwitz, Mackenzie and E. L. Fox. The powdered extract should be prepared from the glands of young sheep, for the thyroid atrophies in older animals. Beginning with gr. ij, t. i. d., the dose may be increased to gr. v. In a short time the evidences of increased metabolism appear, as loss of weight, increased diuresis, increased urea and elevation of bodily temperature; normal growth is stimulated, myxedema and cretinism disappear, the pulse becomes full and lively, the skin moistens and regains its lustre and in every regard the results are magical. In some instances toxic effects (hyperthyroidism) are apparent, as tachycardia, nervousness, delirium, dyspnea, palpitation, flushing, sweating, tremor, less often tonic spasms, severe pains and rarely exophthalmos, softening of the bones, or even death; these symptoms are seemingly more frequent in myocarditic and arteriosclerotic subjects; arsenic lessens the liability of hyperthyroidism, which after all is an infrequent result. Larger doses may be given in winter than in summer. At first large doses are indicated and later, after relief is obtained, smaller doses should be given, averaging gr. j or whatever size experience in the individual case may determine. Relapses are common after withdrawal of the extract and in most cases it becomes rather a food than medication. Change of climate, warm baths and milk or a salt-free diet are of decided benefit.

DISEASES OF THE PARATHYROID GLANDS.

TETANY.

Definition.—Tetany, first described by Steinheim (1830) and Dance (1831) and named by Corvisart (1852) consists chiefly of (*a*) peculiar tonic, intermittent, bilateral, painful spasms, mostly in the extremities, without involvement of the sensorium and (*b*) increased mechanical and electrical excitability of the nerves. The only known diseases of the parathyroids are hemorrhage, tuberculosis and surgical ablation. The parathyroid hormone normally restrains the excitability of the ganglion cells, possibly by promoting calcium assimilation; it antagonizes the thyroid gland.

Etiology.—(*a*) Tetany occurs *endemically*, especially in Vienna, Heidelberg, Berlin and Syria, and during March and April. It is rare in America (115 cases, Howard), England and Italy; though once frequent (1830–1860), it is now uncommon in Paris. In the Vienna outbreaks 46 per cent. of cases occurred in shoemakers and 24 per cent. in tailors; 83 per cent. occur between sixteen and twenty-five years of age. It may develop in an entire family. (*b*) *Digestive affections* may produce it, as

dilatation (Kussmaul, 1871), ulcer or cancer of the stomach or duodenum, often with pyloric obstruction; auto-intoxication plus parathyroid insufficiency affects the nerve cells. Only 80 cases of gastric tetany are on record. (*c*) *Rickets* is causal in 90 to 96 per cent. (Cassel) of the infantile types; Heubner classifies them as spasmophilia, tetany, laryngospasm and infantile convulsions. (*d*) *Tetania parathyreopriva* is a type which follows thyroid operations. Gley (1831) was the first to connect tetany with extirpation of the parathyroid glands, first specifically described by Sandström (1880). Vassali and Generali maintained that absence or ablation of the parathyroid glands causes tetany (without cachexia). Thyroidectomy should be carefully done in order to spare the parathyroids located alongside the lateral lobes. (*e*) Tetany occurs also in the *acute infections,* sepsis, nephritis, intoxications with ergot, morphine or lead. There are recorded only 32 cases of the puerperal form. Of the 6 cases which the author has seen in this country 2 occurred with florid, secondary syphilis, 1 after an adenoid operation and 3 with gartrectasis. It may develop with other nervous diseases. In a few cases there has been cloudiness with swelling of the anterior horns.

Symptoms.—1. Muscular Contracture.—The earliest and chief symptom is the *muscular contracture,* which usually commences in the hand, with stiffness, numbness and pain, and culminates in a tonic, symmetrical, painful contracture. The muscles of the hand are hard; the attitude is that of the accoucheur's hand, the hand in writing or that observed in paralysis agitans; it is produced by contraction of the ulnar flexors; less often it resembles the clenched hand of old hemiplegic contracture, and the nails even enter the palm; rarely the fingers are tonically spread apart. Except in light forms, the spasm cannot be overcome by force. The foot is affected less often than the hand and the spasm draws the foot into the equinovarus position, with the toes flexed and the sole hollow. The spasm is usually limited to these parts, lasts from minutes to hours or even days, disappears and then recurs. In rarer and more severe cases the parts above the elbows and knees may participate, with adduction of the arms and thighs, cramping of the face (risus sardonicus and trismus), neck, chest, tongue, diaphragm and ocular muscles, with diplopia, spasticity of the sphincters, dysuria, etc.

Laryngismus stridulus (Clark, 1815) is common in infantile rickets, in which clonic contractures also occur. Pineles describes laryngospasm following parathyroid extirpation.

2. Trousseau's Sign.—This consists in producing the spasm by pressure over the nerves, for example, just above the elbow or over the peroneal nerve, for a few seconds or minutes. It *is pathognomonic.* Schlesinger's sign; pressure on the sciatic nerve or flexion of the thigh with extension of the knee elicits a talipes equinovarus.

3. Erb's Sign.—There is *increased electrical irritability* of the motor nerves to the galvanic current with early anodal closing and cathodal opening tetanus. Anodal opening tetanus is found in this disease alone. There is also increased electrical irritability of the sensory nerves (Hoffmann) and of the nerves of special sense (Chvostek, Jr.), to faradic and galvanic currents.

4. Chvostek's Sign.—*Increased mechanical irritability* of the motor nerves is observed on tapping or pressing on the facial, median, ulnar or other nerves, which produces contraction of the muscles supplied by them. It is quite constant but may be absent in children and is not pathognomonic, having been found in normal persons, bulbar paralysis, neuroses and facial paralysis. The muscles themselves are not over-excitable.

5. Other Symptoms.—The sensorium is usually clear, except in rare cases with pyloric stenosis. The special senses and reflexes are normal; optic neuritis is extremely rare. Headache and malaise are frequent. In contrast to the pain and paresthesia, objective sensory findings are absent. In rare cases epileptiform seizures are seen. The temperature is elevated in 15 per cent. of cases. In rare cases, there may be disturbances of the intermediary metabolism of albumin; hypothyroidism, trophic, vasomotor and secretory disturbances may occur, as edema, polyuria, glycosuria or falling out of the hair (visceral tetany).

Diagnosis.—The diagnosis in typical cases is most easy from the cardinal signs—the peculiar spasms, Trousseau's, Erb's and Chvostek's signs. In epilepsy these signs are absent and the aura, the cry, falling, tonic and clonic convulsions, biting of tongue and involuntary urination are characteristic. Hysteria mimics everything, but the cardinal symptoms, except the spasms, cannot be reproduced; certain reported epidemics of tetany, as at Gentilly, were clearly hysterical. The other cardinal signs easily separate tetany from the occupation neuroses and acroparesthesia (*v. i.*); meningitis and brain tumor are distinguished on careful examination.

Prognosis.—Death may occur from dilated stomach or laryngospasm. Fränkl-Hochwart concludes that the prognosis is worse than is usually stated; 20 per cent. of his cases died, 20 remained well and the balance had recurrences and serious disabilities.

Treatment.—The treatment varies with the cause. (*a*) *Rest and quiet* are indicated on general principles, although tetany is little influenced by excitement. Massage, strychnine and electricity should be avoided. Change of occupation and location is advisable in the endemic form. (*b*) In the *digestive form,* washing out the stomach and bowels may induce severe spasms. These cases are obstinate and the spontaneous mortality in gastric tetany is 75 per cent.; with operation this figure is reduced (see page 507). (*c*) In the *rhachitic form,* carbohydrates, phosphorus and substitution of human for cow's milk are efficient. Inhalations of chloroform in some cases irritate the sensitive larynx. (*d*) In *tetania parathyreopriva* the mortality is 80 per cent.; parathyroid grafting or feeding is helpful. (*e*) Cases following *acute infections* usually run a spontaneously short course; those in pregnancy rarely indicate abortion; in nursing women, weaning gives prompt relief; for intoxications there are obvious indications. Drugs are of little value except calcium salts, gr. xv; chloral influences the motor nerves, but is inferior to the bromides, as is also morphine.

DISEASES OF THE THYMUS GLAND.

THE **thymic functions** are unknown. It is thought that the thymus is concerned in neutralizing infection, and in certain vegetative functions, as the growth of bone. There is an intimate, though obscure, relation between the thymus, thyroid, pituitary body, parathyroids, testes, ovaries, brain and bone-marrow. The thymus wastes as the testicles and ovaries develop. Thymic extract causes a fall in blood-pressure, cardiac acceleration and, in large doses, fatal collapse. Some class it as a ductless gland. The gland is epithelial in origin, but becomes lymphoid in structure. Its secretion in the first two years of life is milky and contains iodine, cells and a molecular substance. It weighs 12 gm. at birth; after puberty it wastes.

(*a*) *Hemorrhages* into the thymus may be found in congenital syphilis and asphyxia of the newborn. (*b*) Thymic *cysts* are in 50 per cent. merely accentuation of the normal findings, in which the lymphoid elements overgrow into the convolutions of the gland. (*c*) *Abscess* of the thymus, described by Dubois (1850), is often mistaken for softened gummata, cysts and postmortem softening. (*d*) The most frequent *tumors* are sarcoma and lymphosarcoma. Less frequent are dermoids and granulomata. (*e*) *Persistence of the gland after puberty* is described; the term is ill-chosen, as the gland normally persists into adult life. (*f*) Some cases of "persistent thymus" are **hypertrophy** or **hyperplasia,** which is the most common thymic lesion. Microscopically, we find lymphoid hyperplasia, Hassal's concentric epithelial corpuscles, eosinophiles and even myelocytes. (i) *Physical signs.* Dulness of more than 1 cm. to each side of the sternum is pathological; it is greater to the left and shifts on retraction of the head. The gland may be palpated in the jugulum or seen by the fluoroscope like a cap on the heart. The gland may weigh 150 gm. (ii) Plater (1614) associated enlargement of the thymus with *sudden death.* Grawitz and Jacobi described thymic hyperplasia which caused sudden dyspnea, usually in infants, followed by early death; the gland presses on the vagus, cava or trachea or causes laryngospasm. These cases are thought to be lymphatism (*v. i.*). (iii) In *thymic asthma* (Kopp, 1830), also known as Millar's asthma, the stridor, caused by thymic compression of the trachea, is pronounced, progressive and usually fatal from asphyxia. The enlarged gland may actually flatten the trachea (Summa and Benecke), as the distance between the sternum and spine is but little more than an inch and compression in this region is critical. It may be seen by bronchoscopy. In 48 operations, about two-thirds recovered (Veau and Olivier). (iv) Hyperplasia is frequently found in Graves's disease, status lymphaticus, myxedema, syphilis, acromegaly, epilepsy, myasthenia gravis and atrophy of the splenic, lymphatic or myeloid structures. Besides operation, the *x*-rays are excellent.

LYMPHATISM (STATUS LYMPHATICUS).

The *constitutio lymphatica* or *status thymicus* is a condition in which there is hyperplasia of the various lymphoid and kindred tissues and a tendency to sudden death.

The affection is rare and occurs most often in women and children. Felix Plates (1614) observed it as a familial affection.

Symptoms.—I. There is *lymphoid hyperplasia.* The *lymph glands* of the chest and abdomen are more often involved than the external glands. The *tonsils,* nasopharyngeal, lingual and intestinal lymphadenoid tissues are frequently hyperplastic. Moderate *splenic enlargement* is usual, the *thymus* is swollen and the *bone-marrow* often reddish. Leukopenia, lymphocytosis and eosinophilia may be found.

II. *Associated findings* may include thyroid enlargement, tetany and laryngismus. Siess and Stoerk give the following symptomatology: (1) Atypical arrangement of the hair; (2) abnormal length of the extremities; (3) scaphoid scapulæ; (4) a wide pelvis in males, narrow in women; (5) adiposity of the reverse type, in the lower half of the body in males, upper half in females; (6) poor development of the breasts; (7) general glandular hyperplasia; (8) small but elongated heart associated with a strong apex-beat and an accentuated aortic second sound; (9) low blood-pressure; (10) psychoneurotic manifestations; (11) vagotonia; (12) infantile type of epiglottis.

III. *Sudden death* is often the first evidence of the condition and may occur without seemingly adequate cause. It has occurred during or after *anesthesia,* especially with chloroform. In one case a patient with enlarged glands died suddenly after receiving but 20 drops of chloroform. Operations with *local anesthesia* have also been fatal. Administration of *antitoxin* has caused death, as in the well-known case in Professor Langerhans's family. It may occur while *bathing* or after *falling in the water.* Sudden deaths during convalescence from *acute infections* or some collapses during hydrotherapeutic treatment may be attributed to the lymphatic constitution (Escherich). Children with eczema may die suddenly; lymphatism causes 75 per cent. of these deaths. It is said to be due to cardiac excitability (Kundrat), to lessened physiological resistance with a tendency to cardiac paralysis (Paltauf), respiratory paralysis (Meltzer) or lymphotoxism (Blumer).

The *x*-rays may induce regression in the hyperplastic tissues.

DISEASES OF THE HYPOPHYSIS.

The pituitary body consists of two lobes; the anterior, the larger, is of epithelial buccal origin and probably discharges its hormone into the blood, influencing growth and sexual development; the smaller posterior lobe, partly of epithelial and partly of nervous origin, seemingly empties its secretion into the cerebrospinal fluid, increasing blood-pressure

(pituitarin) and influencing diuresis and carbohydrate and fat metabolism.

Hyperpituitarism, analogous to Graves's disease—as in hypophyseal tumors or hyperplasia—is an overfunctioning, perhaps due to chromophile activity. In early life, before the epiphyses ossify, gigantism results; if later, acromegaly. Emaciation, vascular degeneration, calcium retention, thyroid activity (stimulated by the anterior lobe) or adrenal stimulation (from posterior lobe) and lowering of the assimilation limit of carbohydrates may be observed.

Hypopituitarism or deficient internal secretion is analogous to myxedema; when it operates in life, it induces dwarfism or infantilism, obesity, hypoplastic sexual organs and secondary sexual characters (Fröhlich's type), diabetes insipidus and great tolerance of carbohydrates; deficient secretion in later life induces reversive sexual infantilism; often there are dry skin, subnormal temperature and pulse, and drowsiness.

Dyspituitarism combines the above types, to some degree, just as symptoms of Graves's disease and myxedema may occur simultaneously or successively.

ACROMEGALY.

Acromegaly (large extremities) described by Marie (1886), is a dystrophy marked clinically by overgrowth of the bones, tissues of the face, hands and feet and by other symptoms of hyperpituitarism.

Etiology.—In 1902, Woods-Hutchinson collected 262 cases; in 77 autopsies the hypophysis was obviously diseased in 73. The process may be hyperplasia or adenoma, less commonly sarcoma, colloid degeneration, fibrosis, softening or hemorrhage. Benda holds that acromegaly is due to excessive activity of the chromophile cells in the anterior lobe. According to Sternberg, the sexes are about equally affected, and in 50 per cent. the affection begins between twenty and thirty, in 75 per cent. between twenty and forty and in 14 per cent. under twenty years of age.

Symptoms.—1. ACROMEGALY.—Enlargement of the extremities is usually the first clear symptom, but is preceded by paresthesiæ and pains in the extremities, often extreme and due to expansion. (*a*) A remarkable change occurs in the *face;* the superciliary ridges protrude, the maxillæ enlarge and the nose, ears, lips, tongue and chin attain massive proportions. The general contour of the face is hexagonal. The teeth separate and the tongue sometimes protrudes beyond them. The eyes by contrast appear small. Pathologically, the change in the bone is simple hypertrophy, with increase in the vascular furrows, widening of some foramina (with narrowing of others), some roughening and localized protuberances which are not osteophytes; the skull is thicker and larger, especially the sphenoid, superciliary ridges, mastoid and occipital protuberances; the orbit may be narrowed by ethmoid bulging and the sinuses and sella turcica are increased as shown by the *x*-rays. (*b*) *The hands and feet* are greatly but symmetrically *hypertrophied.* The fingers and toes show either the long or the thick type; they are square at their ends—the "spade-like fingers." The palms and soles become flattened. The *x*-rays show simple hypertrophy of the peri-

osteal, subperiosteal and soft tissues; in some cases the soft parts are more hyperplastic than the bones. A common statement in the history is that the patient "must each year buy larger-sized hats, gloves or shoes." The nails are thin and small. The antithesis of acromegaly is micromelia. (*c*) The *changes* in the trunk are less conspicuous. The sternum is increased anteroposteriorly and the ribs and clavicles are thickened. The anteroposterior diameter of the thorax is increased, the head is held backward and the dorsal kyphosis is rather characteristic. The muscular weakness and lean arms and legs contrast sharply with the first impression of strength imparted by the massive face and extremities. The breathing is largely abdominal. (*d*) The *skin* is hyperplastic and also the mucous membrane of the nose, tongue, pharynx and larynx. The enlarged larynx may explain the deep, rough, monotonous voice. Hypertrichosis is common.

2. Nervous Manifestations.—(*a*) Symptoms of *brain tumor* (hypophysis) are common, as headache, vertigo, mental alteration, vomiting, periodic loss of consciousness and eye symptoms in 52.5 per cent., hemianopsia, optic atrophy (40 per cent.), paralysis, anosmia, auditory disturbance and exophthalmos. (*b*) *Changes* in *character,* anxiety, apathy and insomnia which deepens into terminal somnolence, are common; severe pains, paresthesia, acroparesthesia and subjective warmth are frequent.

3. Constitutional Symptoms.—(*a*) *Glycosuria* occurs in 40 per cent. Sometimes polyuria occurs without glycosuria. (*b*) *Profuse sweats* are common, even with glycosuria; the skin is sometimes pigmented; *obesity* is common. (*c*) *Sexual disturbance* is the rule, as amenorrhea often preceded by dysmenorrhea; in men impotence is not unusual; the external genitalia are often hypertrophied, the internal infantile. (*d*) The *heart* is often *dilated* and *hypertrophied* from the concomitant *arteriosclerosis,* deformed chest and rarely from actual overgrowth (cardiomegaly). The vessels are wide and thick. Splanchnomegaly may be observed (intestines, tonsils, spleen, thymus and thyroid). In most cases the lymph nodes, particularly the cervical, are palpably enlarged.

Course and Prognosis.—(*a*) Acromegalic subjects seek advice for the enlargement of the extremities or tonsils, sweats, pain, eye symptoms or polyuria; (*b*) remissions are common, the constitutional symptoms and, to a slight extent, the enlargements subside; on the other hand, a fitful advance may be noted. (*c*) The average course is *chronic,* covering ten to twenty years, though relatively benign cases occur, lasting thirty or even fifty years, and acute malignant cases are known, which are due to malignant adenomata. (*d*) *Death is inevitable;* it may result suddenly or from gradual marasmus, diabetic coma, intercurrent infection, cardiac insufficiency or pyelonephritis.

Diagnosis.—Charcot held that physicians in general see and diagnosticate only what they have seen and learned to diagnosticate; the diagnosis is made at a glance by one who has seen a single case; undue regard to a single symptom, as the skin changes, amenorrhea or glycosuria, may cause error.

Differentiation.—1. In the *cranium progenium,* the hyperplastic lower jaw protrudes and its teeth are directed forward; the upper jaw

is hypoplastic; it may occur in acromegaly, cretinism, healthy individuals, degeneracy and idiocy.

2. *Gigantism* occurs in over 20 per cent. of acromegaly, particularly when acromegaly develops early in life. Forty per cent. of giants are acromegalic (Sternberg). Gigantism implies symmetry of overgrowth; there are normal "giants" and giants with acromegaly, leontiasis (*v. i.*), facial hemihypertrophy, etc.

3. *Diffuse hyperostosis* (Starr, Putnam, Prince) occurs in the young, involves all the skull bones, but seldom the extremities or spine; exophthalmos, blindness, deafness, bilateral facial palsy, dysphagia, dyspnea and other basilar symptoms are noted, which culminate fatally in stupor, idiocy, epileptiform convulsions and paralysis of the extremities. A subtype (leontiasis ossea, Dana) occurs in the form of tumor-like protuberances on the skull and other bones. Prince (1902) collected 20 cases from the literature. He maintains that it is a form of osteitis deformans.

4. *Osteitis deformans* (Paget's disease) is a combined rarefying and hypertrophic osteitis. It begins slowly in subjects over forty years old, with pains and deformity of the thighs, legs and spine; the legs bow widely outward and somewhat forward, the cervical and upper dorsal spine becomes kyphotic, the thorax narrows transversely and widens anteroposteriorly, the abdomen is diamond-shaped and crossed by a sulcus and the head hangs forward. The soft parts do not hypertrophy as in acromegaly, the face is rarely involved, the head is larger above than below and the arms are little altered. Clopton (1906) found 75 cases on record.

5. *Hypertrophic osteo-arthropathy,* known since the time of Hippocrates, was especially described by Bamberger and Marie, in 1889. The ends of the fingers and perhaps the toes become clubbed or "drumstick" in appearance. While the nails are thin and small in acromegaly, in hypertrophic osteo-arthropathy they are large, thick, wide and curved downward. The alteration typically involves the terminal phalanges, but also in severe cases the ends of the ulna, radius, tibia and fibula. The pathology is a toxemic, chronic periostitis, with hypertrophy and osteophytic growth. The most common cause is chronic lung disease (chronic phthisis, bronchiectasis, emphysema or fibrosis), whence Marie's name of *osteo-arthropathie hypertrophiante pneumonique;* it may develop in congenital heart disease, aneurysm or, less frequently, in hepatic cirrhosis, gastric dilatation, syphilis, cystitis or various other infections.

Treatment.—The treatment of acromegaly is symptomatic. Extract of the hypophysis has been given and in a few cases with temporary success. Operation seldom helps.

INFANTILISM.

The persistence beyond puberty of the physical and mental characters of childhood.

I. The Fröhlich Type, dystrophia adiposogenitalis, is hypopituitarism —obesity, persistence of, or reversion to, the secondary sexual characters

of childhood, hypotrichosis, poor mammary or testicular development, gracile figure, lack of the masculine voice and hypoplasia of the sexual organs.

II. The Brissaud type, referable to hypopituitarism (or hypothyroidism, Brissaud), presents a "round face, lips projecting and plump, small nose, smooth face, clear skin, fine hair, eyebrows and eyelashes scant, eyes prominent, large, round cheeks, infantile face and head, skeleton underdeveloped, neck short and chubby, body long and cylindrical, abdomen prominent, limbs, round and large, a layer of fat over the whole body, infantile pelvis, lumbar lordosis, rudimentary sexual organs, no hair except on head, high-pitched voice, thyroid small, mind slow, retardation of ossification, and absence or retardation of second dentition."

III. The Lorain type is as follows: "Small stature, delicately formed, slender skeleton, skin soft and pale, large shoulders, lower extremities long and slender, trunk relatively small, infantile sternum and pelvis, epiphyses normally united, no fat increase, abdomen normal, finely chiselled face, voice high, neck long, genital atrophy, absence of or slight secondary sexual characteristics."

IV. Herter's Intestinal Infantilism.—The child stops growing as it stops gaining in weight; the mental development is fair. Marked abdominal distention is common, often with dilatation of the abdominal veins. There is a moderate anemia, a very characteristic fatigue, looseness of the bowels with occasional attacks of diarrhea, often with fatty stools, an excessive appetite and thirst, an increased secretion of urine, and cold hands and feet. There is an absence of the ordinary intestinal flora of the intestines of young children. The prevailing organisms are the Bacillus bifidus, Bacillus acidophilus and Bacillus infantilis. There is a rise in the ethereal sulphates, indican, phenol and aromatic oxyacids.

V. Other types include malnutrition, myxedema, lues, congenital heart lesions, etc. Gilford describes micromegaly, ateliosis (continuous youth and premature old age), or progeria. A pancreatic form, due to deficient pancreatic secretion, is associated with recurring severe diarrhea in children.

SECTION VIII.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

Definition.—A disease of metabolism characterized (*a*) by a permanent *lessened capacity* of the organs fixing, storing and consuming *grape-sugar;* (*b*) by excess of sugar in the blood (*hyperglycemia*), resulting in (*c*) grape-sugar in the urine (*glycosuria*); (*d*) acid-intoxication; and (*e*) *causal changes* in *the pancreas.*

Willis (1674) differentiated between diabetes mellitus and diabetes insipidus and Dobson (1776), of Liverpool, fully described the symptoms; Aretæus (150 A.D.) used the term diabetes.

The liver, the reservoir for carbohydrates, contains up to 14 per cent. glycogen. It collects carbohydrates from the portal vein, stores them as glycogen and again imparts them to the blood as grape-sugar, for use in the tissues. The muscles also manufacture glycogen from the grape-sugar of the blood. Glycogen may also be produced from the albumins and possibly from fats. When there is a temporary carbohydrate shortage, the reserve store of glycogen is drawn upon. Fat is converted in the liver into sugar when neither the carbohydrates nor the albumins suffice to maintain the normal proportion of sugar in the blood. The fate of carbohydrates when ingested in excess is: (*a*) glycogen storage (up to 400 gm.); (*b*) conversion into fat; (*c*) glycosuria from hyperglycemia, when sugar in the blood exceeds 0.2 per cent.; this "alimentary glycosuria" is physiological and occurs when the blood-sugar exceeds the assimilation limit. Alimentary glycosuria is tested by giving 100 gm. of grape-sugar, after a light breakfast of bread and coffee. If more than 1 per cent. of sugar appears in the urine or if 2 or 3 per cent. of the entire amount administered is found, it indicates diabetes rather than glycosuria. Most absorbed sugar goes to the liver, but some absorbed by the lymphatics appears in the urine, whence its presence is not necessarily a sign of diabetes; in liver cirrhosis, sugar may reach the blood by the collateral circulation. Traces of sugar (0.01 to 0.02 per cent.) are normally present in the urine, but are not detected by the usual tests.

Etiology.—1. The *pancreas* (*q. v.*) has long been considered an etiological factor. Opie (1900) discovered *atrophic changes* in the cells of the *islands of Langerhans* which produce an *internal secretion,* regulating or inhibiting the conversion of glycogen into sugar. Weichelbaum found "Langerhans's insufficiency" in all of 183 cases, as hydropic changes in

younger subjects or as sclerotic, atrophic or hyaline changes, in arteriosclerotic or obese cases. This internal secretion from Langerhans's islands is wholly distinct from the external secretion of the pancreatic juice voided into the intestine. (*a*) Total pancreatic extirpation in animals and man produces glycosuria; (*b*) after partial extirpation and suture of the remaining portion to the abdominal wall, diabetes fails to appear, but develops if this portion is later removed; (*c*) when a tenth part is left, glycosuria occurs only after the ingestion of carbohydrates; (*d*) subsequent destruction of the remaining portion of the gland produces a severe form; (*e*) when more than one-tenth of the functionating gland is left behind no diabetes ordinarily results; (*f*) diabetes does not occur in simple ligature of the pancreatic duct, excluding its juice from the intestine, nor when the fluid escapes through a cutaneous fistula, whence the argument for an internal secretion (Minkowski and v. Mehring); (*g*) Cohnheim suggests that a pro-enzyme secreted by the pancreas, joins another ferment secreted by the muscles and that the united ferments break down the sugar molecules to produce heat and energy.

2. *Other Ductless Glands.*—The *adrenals* stimulate the conversion of glycogen into sugar, mobilizing the sugar, and antagonizing the pancreatic action; adrenalin injected intramuscularly causes transient glycosuria, acting on the peripheral sympathetic. Adrenalectomy is followed by hypoglycemia; in Addison's disease the tolerance for sugar is high and it is impossible to produce adrenalin glycosuria. The *hypophysis* is antagonistic to the pancreas and is associated with adrenal hyperfunction and thyroid hypofunction; in acromegaly glycosuria is common; in dystrophia adiposogenitalis high carbohydrate tolerance is noted. The *parathyroids* inhibit thyroid action; after parathyroidectomy sugar tolerance is reduced and adrenalin glycosuria readily effected. The *thyroid* antagonizes pancreatic activity, but stimulates adrenal activity. After thyroidectomy and in myxedema, there is hyperfunction of the pancreas; sugar tolerance increases and adrenalin causes no glycosuria. Thyroid feeding and Basedow's disease produce glycosuria.

3. *Neurogenous diabetes* occurs through the sympathetic and chromaffin systems, controlling the various ductless glands. Claude Bernard's well-known puncture of the fourth ventricle (piqure glycosuria, 1844), may possibly lessen *via* the sympathetic the power of the liver to hold glycogen. Apoplexy, tabes, brain tumors, multiple sclerosis, meningitis syphilis, softening of the brain, diseases of the cord, vagus and sympathetic system may induce glycosuria. Hoffmann distinguishes between (*a*) *neurogenous diabetes*, which occurs chiefly in men, with no tendency to furuncles, carbuncles or cataract, with no connection with gout, and is temporary; and (*b*) *diabetes of the obese*, which occurs in either sex, often produces carbuncles, is often connected with gout and is very chronic.

4. *Obesity* embraces cases with ordinary diabetes, obesity without glycosuria (masked diabetes) and obesity with consecutive glycosuria.

5. *Gout, arteriosclerosis, liver lesions* (cirrhosis) and possibly acute infections have apparent relations with diabetes. Renal diabetes (glycosuria without excessive sugar in the blood) may develop in nephritis, renal hemorrhage, chyluria, etc. *Lactosuria* may occur in late pregnancy,

the puerperium and nurslings; von Noorden's hypothesis is that in the "puerperal state the capacity of the tissues for breaking up milk-sugar is diminished, an instance of adaptation to an end in that the cells of the mother refuse a material which is preëminently suited to the nutritive wants of the nursling."

6. *Predisposing factors* are (*a*) *sex;* men (3:2) are especially subject to diabetes; (*b*) *age;* its frequency is as follows: In persons from thirty to forty years, 18 per cent.; forty to fifty years, 25 per cent.; fifty to sixty years, 30 per cent.; in Stern's collection of 117 infantile cases 1 was apparently congenital. (*c*) *Heredity:* A family history of obesity, consanguinity, neuropathic taint or gout is especially significant; cases are reported in which the grandfather was diabetic, the son gouty and the grandson diabetic (alternating diabetes). (*d*) *Race:* Diabetes is comparatively rare in the colored race and is especially common in Jews. (*e*) *Occupation:* Diabetes is more often seen in private than in hospital practice. Wealth and culture increase tenfold the predisposition to diabetes. It is frequently found in scientists, lawyers, musicians, poets, teachers, statesmen, merchants, speculators and in those leading a luxurious or self-indulgent existence with sedentary habits; it also results from overwork, excitement, overeating and overdrinking.

Symptoms.—I. HYPERGLYCEMIA.—The blood-sugar rises to 0.1 to 1.6 per cent., either because the diabetic cannot store or oxydize sugar or because sugar is formed in excess.

II. THE URINE.—*Glycosuria* is the leading symptom of diabetes. A daily excretion of 5 to 12 grams is not uncommon; the maximum amount recorded is 1500 gm.; the largest quantity of sugar is voided late in the morning or after 6 P.M. A percentage of from 1 to 4 is common, 8 or 9 per cent. is rare and the maximum is 20 per cent. (personal observation). The diabetic's capacity to consume sugar is considered under Therapy. Muscular exercise usually decreases it. It is often increased by psychic disturbances and may be suspended during an acute intercurrent infection, especially typhoid; chronic diseases with fever, especially pulmonary tuberculosis, have less influence. It may be reduced or absent in the final stages of diabetes, diabetic coma and when granular nephritis develops. Gouty diabetics as a rule pass no sugar during an attack of gout (diabetes alternans).

Tests for Sugar.—(*a*) For *Fehling's test,* two solutions are kept separate; one contains copper sulphate and the other sodium and potassium tartrate and sodium hydrate. Equal parts of these solutions are heated, but not boiled, and the urine is added drop by drop. The volume of urine should never exceed that of the solution. Reduction of the blue copper solution to the yellowish-red suboxide of copper indicates sugar. Chloral, phenacetin, chloroform, formalin, morphine, uroleukinic and homogentisinic acids (the probable causes of alkaptonuria), glycuronic acid, cascara, rhubarb and salicylates may also reduce Fehling's solution, particularly after boiling. In doubtful cases (*b*) the *fermentation test* may be used; yeast is shaken thoroughly with a quantity of urine, the air allowed to bubble up and the mixture placed in a saccharometer—a tube closed at the top and graduated for estimation of percentages.

The sugar, fermenting, is measured by the amount of carbon dioxide formed. A control test should be made. (*c*) The *polarization test* shows dextrorotary reaction. (*d*) The very delicate *phenylhydrazin test* may be used. (*e*) In Robert's test, the sugar percentage equals the difference between the specific gravity before and after fermentation, multiplied by 0.23.

Quantity and Specific Gravity.—The urine is acid even after fermentation (lactic acid) and has a sweet taste. The quantity of urine secreted during the night is less than that secreted in the daytime, whereas in other *polyurias* frequently $\frac{2}{3}$ to $\frac{3}{4}$ of the urine is secreted during the night. A secretion of ten quarts is not rare and 17 and 28 quarts are recorded. Higher figures suggest simulation, patients having been known to add sugar to the urine. In part, polyuria results from the inability of the kidneys to concentrate the urine. *Pollakiuria* (frequency of urination) is common. The urine is *pale;* if dark and if uratic deposits are present, the prognosis is good. Foam often remains long on its surface and the sediment is scant or absent. Prout dated the onset of diabetes when the urine ceased having a turbid, uratic deposit.

Amount in litres or quarts.	Specific gravity.	Sugar.
1.5 to 2.5	1.025 to 1.030	2 to 3 per cent.
2.5 to 4	1.030 to 1.036	3 to 5 per cent.
4 to 6	1.032 to 1.046	4 to 7 per cent.
6 to 10	1.036 to 1.046	6 to 9 per cent.

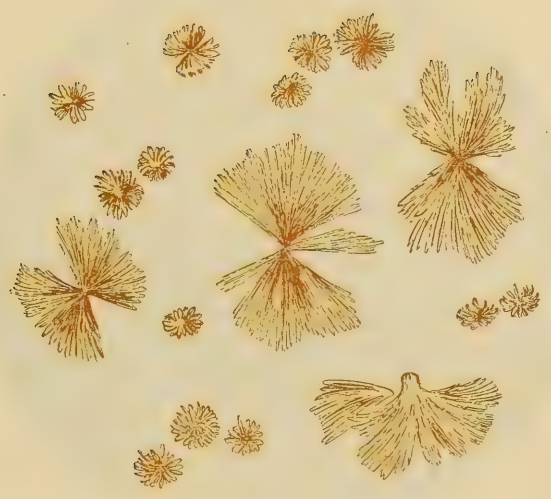
The average specific gravity is 1.030 to 1.040, it is rarely more than 1.050, and the highest record is 1.074; very high specific gravity suggests fraud; the lowest records are 1.003 and 1.002. If under a meat diet the urine becomes entirely or nearly free from sugar, the quantity approaches normal; the specific gravity remains high because the urine is rich in meat end-products. There is no absolute parallelism between the percentage of sugar and the specific gravity. In some cases, though there is a large percentage of sugar, only a pint or a pint and a half of urine is excreted, described by Peter Frank a century ago as "diabetes decipiens." Crystals of sugar may be seen on the shoes, clothing or in the vessel.

Nitrogenous Constituents.—Diabetics usually excrete 15 to 20 gm. or more of nitrogen daily (the normal being 10 to 15 gm.); the urea may reach even 60 to 150 gm., (metabolism being increased 15 per cent. in severe types); larger figures are reached in no other disease; the condition is pathological when the amount of nitrogen in the urine exceeds that in the food.

Ammonium, normally 0.5 or 1 gm. *per diem,* may increase to 3, 6 or even 12 gm., and may constitute 10 to 20 per cent. of the total nitrogen output (instead of the normal 3 to 5 per cent.); over 5 gm. indicate a severe type, probably with death within a year, especially in subjects over fifty years of age; the highest figures are seen exclusively in diabetic coma. The organism excretes increased ammonia to neutralize acidosis. Creatinin may reach 1 or 2 gm. *per diem.* The uric acid is nearly normal.

Acetonuria, or ketonuria, indicates the presence in the blood of β-oxybutyric and diacetic acids. This acidosis results from either (i) an

PLATE XIX



Crystals of Phenyl-glucosazone. (Musser.)

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

entirely abnormal type of metabolism or (ii) intermediate substances, which the diseased organism cannot split up into normal end-products (water and CO_2), thus robbing the body of native alkali and decreasing the power of the blood to take up CO_2. These acid sustances are derived from the fatty acids and possibly from protein. *β-oxybutyric acid* is absent in diabetics in good physical condition and not infrequently also in those who are suffering from marked loss of albumin. The urine and breath may be odorless or may smell like chloroform, fruit or wine. The daily excretion varies from a few grams to 50 to even 100 to 180 gm. Its abnormal excretion once begun, is uninterrupted and constantly tends to increase. It is of the gravest prognostic significance because in most cases, at the end of days, weeks or months, fatal diabetic coma develops. An excretion of more than 30 gm. indicates imminent coma. β-oxybutyric acid is thought by some to be the parent substance of acetone and diacetic acid. *Acetone and diacetone* are present in the urine in diabetes, fevers, cancer, inanition or auto-intoxications and indicate malnutrition. When the amount of acetone progressively rises from 1 gm. upward, the outlook is unfavorable, its normal daily output being 0.01 to 0.03 gm. Acidosis may be measured by the amount of ammonium excreted or, more roughly, by the amount of sodium bicarbonate necessary to render the urine alkaline.[1]

Albuminuria is found in 35 per cent. of cases; it is slight and is due (*a*) to cystitis, pyelitis, intercurrent diseases as gangrene or infection, pulmonary tuberculosis or without relation to diabetes, as in the arteriosclerotic and gouty types of diabetes, liver cirrhosis, etc.; (*b*) to circulatory disturbances, and (*c*) in uncomplicated diabetes it may be due to the action of the sugar, β-oxybutyric and other acids and toxins on the kidneys; albuminuria is a frequent forerunner of diabetic coma (decreased renal permeability with retention in the blood of toxins). Diabetes may develop into chronic interstitial nephritis and the sugar then disappears.

Sodium chloride, sulphuric and phosphoric acids and the ethereal phosphates are abundant, caused by the amount of food ingested. Much lime is excreted, which suggests osseous waste.

Pneumaturia.—The butyric acid bacillus, the Bacillus coli, yeast fungus and other bacteria may cause fermentation in the bladder, producing gas. Lipuria has been observed.

III. Direct Consequences of the Glycosuria and Polyuria.—(*a*) The exaggerated *thirst* (*polydipsia*) is secondary to the polyuria. (*b*) *Nutrition:* For each gram of sugar in the urine four calories are lost to the tissues, and for each gram of β-oxybutyric acid, five calories. Therefore the nutritive requirements are great, the appetite is excessive (*polyphagia*) and in severe cases emaciation and fatigue are pronounced. Increase in weight may follow reduction in the sugar. The temperature in uncomplicated cases is subnormal from inanition.

[1] L. Blum finds that from 5 to 10 grams of sod. bicarbonate are sufficient to render the normal urine alkaline, while with mild acidosis 20 grams are necessary. With an acidosis of moderate intensity from 20 to 30 grams are required, in severe cases of acidosis 50, and in diabetic coma 150 grams or more are necessary to make the urine alkaline.

IV. Complications.—Complications and other symptoms result from various causes: (*a*) excess sugar in the blood, (*b*) malnutrition and lowered physiological resistance; both conditions predispose to (*c*) infection, particularly by the Bacillus tuberculosis, (*d*) acidosis and other ill-understood degenerations and toxemias.

1. *Nervous.*—(*a*) *Intellectual depression,* headache and neurasthenic symptoms are common; psychoses are infrequent. In rare instances focal symptoms develop without anatomical findings. (*b*) *Diabetic coma* occurs in 20 per cent. and causes half the deaths in diabetes; it is most frequent in youth and in acute and severe types; Naunyn holds that it is favored by a strict antidiabetic diet. All coma in diabetes is not diabetic; patients may succumb (i) to cerebral *apoplexy,* embolism, thrombosis, sepsis or *uremia;* (ii) to *heart failure,* which may cause sudden death, or coma with death after a short time or (iii) *diabetic coma* proper, due to acidosis and closely resembling experimental acid poisoning. Most writers believe that β-oxybutyric acid is causal, though in three of the author's cases it was not present in the urine. Coma is imminent when the daily output of ammonium exceeds 3 gm. It may occur suddenly without obvious cause or may follow exertion, excesses, excitement or gastro-intestinal disorders. In some cases there are prodromal headache, stupor, restless anxiety or symptoms resembling acute alcoholism. The pupils are dilated, the reflexes abolished, the small pulse averages 110 and the temperature is usually subnormal. Convulsions are most uncommon. The breath has the wine-like acetone odor. The breathing is characteristic; inspiration is deep, long-drawn and energetic and expiration is short and slightly sighing; in this, *Kussmaul's dyspnea* (1876), the inspiratory energy contrasts strongly with the general bodily weakness; respiration is generally somewhat accelerated; there is no stridor nor, at the onset, cyanosis. The albuminous urine contains innumerable short, broad, granular casts (Külz); a few drops of ferric chloride give the Burgundy wine color; sugar is present, though it may disappear as the coma develops. Partial consciousness may return, but, with few exceptions, death occurs within a few hours to two days, with subnormal temperature, slowed respiration and cardiac weakness. The autopsy reveals no constant brain changes. (*c*) Toxemic degeneration in the posterior columns of the cord may develop. (*d*) *Neuralgia* is common and may indicate incipient neuritis or may appear and disappear varying with the amount of sugar in the blood and urine. It is often an early symptom; it is more often bilateral, most frequently affects the sciatic nerves and is a most obstinately recurrent symptom. *Neuritis* occurs in severe forms of diabetes and in its later stages. Neuritis may involve the cranial nerves, mostly the abducens, or with greater frequency the spinal nerves; the lower are affected more often than the upper extremities and, like diabetic neuralgia, bilateral involvement of the crural or sciatic nerves is the most common type. Its symptoms are those of neuritis or polyneuritis, as paralysis, obstinate pain, painful paresthesia in the legs, muscular cramps, abolished patellar reflexes, reaction of degeneration and trophic disturbances, as falling of the hair and nails, glossy skin, herpes or perforating ulcer of the foot (*mal perforant*). In severe cases tabes is simu-

lated, *pseudotabes diabetica,* described by Fischer (1886) (see MULTIPLE NEURITIS). (*e*) *Special senses.* Retinal disease (Jager, 1856) occurs under four forms: (i) albuminuric retinitis; (ii) *retinitis centralis punctata* with characteristic shining bilateral spots, with hemorrhagic points; (iii) the ordinary hemorrhagic type (see Plate IV, Fig. 1); (iv) lipemia retinalis, in which the vessels appear white and red. It occurs in 19 per cent. Neuroretinitis and retrobulbar neuritis with consecutive atrophy are progressive and occur more frequently in severe cases. Twenty per cent. of diabetics with retinitis die within one year and 60 per cent. live over two years (Nettleship).

Amblyopia and amaurosis often first induce the patient to seek medical advice. Paralysis of the ocular muscles occurs in 7 per cent. of cases; accommodation paralysis is the most frequent type, and then that of the external rectus; the most probable cause is neuritis. Cataract occurs in 4 per cent. Aural complications include external furuncles and otitis media, with a tendency to hemorrhage and mastoiditis.

2. *Circulatory.*—(*a*) The *blood* may become concentrated, for much water is required to eliminate the sugar; the blood-sugar is increased. Fat in the blood (lipemia) is a frequent finding; what appears to be fat is sometimes albumin or cholesterin in combination with fatty acids; Fischer in 1 case found 18 per cent. of fat. In lipemia, acidosis is always present, although acidosis may develop without lipemia. *Bremer's test* is based on changes in the hemoglobin; thick smears of blood are made on a slide, heated and treated with 1 per cent. Congo red solution for two minutes. Diabetic blood will not stain. Williamson's test is based on the fact that diabetic blood decolorizes a weak alkaline solution of methyl blue. Acidosis (*v. s.*). (*b*) The *arteries* are frequently sclerosed and cause cardiac hypertrophy or dilatation, angina pectoris, cardiac asthma, hemiplegia, albuminuria, senile gangrene and intermittent claudication. (*c*) The *heart* may be altered variously (*v. s.*). Hypertrophy occurs in about 10 per cent. and is less ominous than atrophy, which occurs in uncomplicated diabetes of the young. Heart failure may occur after moderate exercise or may develop precipitately, ending in coma or asphyxia and classified as an acidosis sign by Erdmann. Von Noorden observed 5 sudden deaths in 140 fatal cases. The pulse is accelerated and its tension reduced. Dropsy is uncommon.

3. *Respiratory.*—(*a*) *Tuberculosis* is most frequent in the poor classes and in the young, but much less so in older, obese and gouty subjects; 25 per cent. of diabetics contract tuberculosis. Hemoptysis is very rare; the sputum is abundant; in the secondary necrosis, to which all diabetics are prone, the marked clinical and pathological lung findings may sharply contrast with the few tubercle bacilli in the sputum. The course is rapidly fatal. (*b*) *Gangrene* is much less common than tuberculosis; it is characterized by fever, pulmonary hemorrhage, purulent sputum and fetor which is less conspicuous than in other forms of pulmonary gangrene. (*c*) Intercurrent pneumonia is usually fatal.

4. *Digestive.*—(*a*) The *mouth* is dry and acid from decomposition of sugar; the saliva is scanty; stomatitis and gingivitis are common and the growth of aphthæ is promoted by the acid reaction of the mouth,

though preventable by cleanliness. The teeth may decay from alveolar periostitis, or drop out from trophoneurotic changes. (*b*) The *stomach* is affected with remarkable infrequency, considering the oral findings and the polyphagia. Anorexia is uncommon, though the patient may refuse fatty and nitrogenous foods. The stomach is usually dilated. (*c*) The *bowels* are generally costive. Gastro-intestinal catarrh sometimes seems to precipitate coma, but it may be merely an associated symptom. Severe abdominal crises may simulate tabes or surgical conditions (cord degeneration). (*d*) The *liver* is usually enlarged and sometimes tender; it is rose-colored, transparent, homogeneous; fatty changes in Kupffer's stellate cells and peculiar refractile bands along the capillaries are constant (Rössle). Gall-stones are concomitants in 10 per cent. of cases, but they have no intrinsic relationship to diabetes. Hanot and Chauffard described a pigmentary cirrhosis in diabetes (*diabète bronzé*) which probably develops in the following sequence: (i) hemolysis, (ii) pigment deposit in the liver, pancreas and skin from the altered blood (hemochromatosis), (iii) chronic interstitial inflammation, chronic hepatic cirrhosis and (iv) chronic pancreatitis, causing diabetes (see pages 572 and 603).

5. *Genito-urinary.*—(*a*) Albuminuria may be present (see URINE). The kidneys are hypertrophied and hyperemic; various degenerations may be noted, including Ehrlich's glycogenic degeneration. Interstitial nephritis may follow and replace diabetes, chiefly in gouty and corpulent individuals. (*b*) Cystitis is common; the urine may ferment in the bladder, just as it decomposes in the urinal; this may lead to pyelonephritis or pneumaturia (*v. s.*). Cystitis may cause temporary disappearance of the glycosuria. (*c*) In the female, menstruation may be disturbed or suspended. The sexual inclination is usually lessened in severe cases, but may be increased from genital pruritus (*v. i.*). Conception may occur even in advanced cases, but in 33 per cent. there is spontaneous abortion or premature delivery. In severe diabetes interruption of pregnancy is indicated. In Offergeld's series, the maternal mortality was 30 per cent. from coma (collapse, infection) and, in all, 50 per cent. within two and one-half years, and the child's mortality was 66 per cent. either *in utero* or within ten days. Fungous development about the labia and vagina is promoted by lessened general resistance and local contact with the saccharine urine, particularly in careless subjects; *pruritus pudendorum,* vulvitis, vaginitis, urethritis, furunculosis and even gangrene may develop. (*d*) In the male, impotence results from spinal degeneration. Phimosis, balanitis, erosions near the urethra, urethritis, itching at the meatus causing tenesmus, curious nodes on the corpora cavernosa, furuncles and necrosis may first engage the physician's attention. Genital symptoms in men are less conspicuous than in women.

6. *Cutaneous.*—The skin is dry and harsh when there is much polyuria and emaciation. *Itching* is caused by toxemia irritating the cutaneous nerves, as in uremic and cholemic pruritus. Skin eruptions include pityriasis, erythema, eczema, bullæ, acne, purpura and herpes. Infection is favored by reduced physiological resistance and hyperglycemia; *furunculosis* (15 per cent.), due largely to the staphylococcus, may

initiate lymphangitis, phlegmon or gangrene; erysipelas is promoted by reduced resistance. *Gangrene* is most frequent in late or middle life, and in mild cases; it usually begins on the toes and extends upward, as dry or moist gangrene; it results from obliterating endarteritis or the ordinary plaque-like atheroma. Unfortunately gangrene is usually progressive. Wounds in diabetics heal better than in pre-antiseptic times, but delayed healing, infection and gangrene are still frequent. Enlargement of the lymphatic glands is seldom absent in severe forms with emaciation.

Course and Prognosis.—The onset may be acute and the course short, covering weeks, or protracted and remittent, covering twenty years or more; 80 per cent. lose their tolerance of carbohydrates within ten years. Complications modify the duration and outcome. In von Noorden's summary of the *prognostic signs, favorable* signs are: (*a*) onset late in life; (*b*) long duration, with no grave complications or emaciation; (*c*) traumatic or (*d*) syphilitic origin; (*e*) the occurrence of mild forms of diabetes in the family of the patient; (*f*) precedent and coexistent obesity; (*g*) coexistent uric acid diathesis; (*h*) marked oscillations and increasing tolerance of carbohydrates; (*i*) circumstances which permit of dietetic and hygienic prescriptions. *Unfavorable* prognostics are: (*a*) onset in early life; (*b*) great loss of strength, despite a brief duration of the diabetes; (*c*) a history of severe forms in the family; (*d*) early appearance of grave complications; (*e*) a high degree of glycosuria, with complete intolerance of carbohydrates; (*f*) excretion of *β-oxybutyric* acid; coma; (*g*) circumstances frustrating dietetic treatment or avoidance of bodily and mental overwork.

Diagnosis.—Only when the urine in every case is examined as a routine procedure, can the lighter cases, amenable to treatment, be discovered. In many cases the thirst, polyuria, emaciation, itching and the complications *per se* are suggestive. *Pentosuria;* 19 familial or hereditary cases are recorded. Pentose reduces Fehling's solution, even after fermentation of the urine.

Treatment.—1. DIET.—Regulation of diet, dating from Rollo (1797), is the most essential point. The carbohydrates normally supply nearly half the bodily requirements for food and energy. The diabetic loses a large part by their escape through the kidneys, which entails a waste of digestive energy, irritation of the tissues by the unused sugar and a growing decrease of the tolerance of the tissues for it. A man weighing 70 kilograms (150 lbs.) requires a diet representing 2500 calories, *i. e.*, roughly, 35 calories for each kilogram. The dietary must therefore regard not merely the symptom, glycosuria, but the maintenance of body weight and energy. The therapeutic aim is to limit the carbohydrates and replace them with albumin and fat, increase the tolerance for carbohydrates and supply sufficient calories.

General Outlines of Dietary.—Explicit written directions should be given each patient. *All diabetics may eat the following foods: meat:* beef, veal, mutton, game, tongue, lung, heart, brain, marrow, sweetbreads, fatty liver, peptones, gelatin and meat jellies; *fish:* caviar, cod-liver oil, clams, oysters, lobsters and crabs; *eggs* (10 to 20 per cent. of fat); animal

and vegetable *fats,* bacon, suet, olive oil, cocoa butter; butter contains 85 per cent. fat, and should be used on meats, eggs or spinach or melted in milk; rich cream; all *cheeses* (3 to 30 per cent. fat), especially Swiss varieties; *vegetables:* cress, tomatoes, cabbage, cauliflower, sprouts, artichokes, mushrooms, truffles, olives, onions, garlic, celery leaves, lettuce, cucumbers and radishes; of *fruits,* only whortle-berries, young raspberries and green gooseberries should be eaten, though fruit-sugar (levulose) is better tolerated than starch or cane-sugar. *Desserts* (sufflés) of eggs, gelatin and lemon; tea, coffee, Rademann's diabetic cocoa and von Hoevel's saccharine chocolate may be taken.

Of the calories necessary in health, 1800 are supplied by carbohydrates; these must be replaced by protein and fat; protein cannot be greatly increased, at the most 500 gm. of cooked meat (700 gm. raw meat) = one pound (lean meat = 500, and fat meat = 1000 calories). Fat must make up the balance.

To some cases we allow after determining the limit of tolerance, beans and peas; turnips, carrots, celery bulbs, wax beans; radishes; walnuts, hazel-nuts, almonds (no chestnuts); apples, pears, apricots, peaches, berries; cooked apples, plums, pears, peaches, cherries and milk; some diabetics tolerate levulose, cane-sugar or honey, in small amounts and intermittently. (See Food Values, pages 53 and 54).

Gradual withdrawal of carbohydrates is tolerated better than abrupt restriction. Glutin flours differ but little from ordinary flours in their percentage of starch. Aleuronat or soy bean flour contains 7 per cent. of starch. Fat is best absorbed when some carbohydrates are given with it. If some starch can be given Mossé prefers *potatoes,* which are more easily assimilated and contain but 20 per cent. of starch (bread contains 55 per cent.). Saccharin should be used in place of sugar, gr. iss daily; it is 280 times as sweet and is antifermentative, but an excess may occasion dyspepsia. Naunyn advised an occasional *fast-day,* and Austin Flint kept his cases in bed and without food from Saturday night until Monday morning. As the disease is eventually progressive, a schematic diet should not be insisted upon; reasonable restriction, with *occasional dietetic treats* is less often violated by the diabetic, who is always hungry for bread and potatoes. The patient's weight, color and strength are of more significance than the urine, remembering that there is little or no carbohydrate metabolism, increased protein and perverted fat metabolism. *Alcohol* aids in the absorption of fat, prevents tissue waste and contributes energy and heat; not over 30–70 gm. should be given; it is contra-indicated in obvious hepatic disease and sweet or white wines and beer are injurious.

Treatment of individual forms is conducted by determining the limit of tolerance; the percentage of sugar on a general diet is found, then the amount on a sugar-free diet and finally the amount of carbohydrate which can be given without glycosuria ensuing. (*a*) *Mild forms of glycosuria; in elderly persons* it is not necessary to determine with great accuracy the sugar tolerance (about 100 gm. bread). The patient may eat bread, potatoes and vegetables, for the quantity of sugar in the urine is usually insignificant (1 or 2 per cent.). The older the patient and the more

corpulent, the more caution necessary in reducing the body weight; fatty foods should be increased—butter used freely on bread, potatoes, vegetables and meat; and bacon, eggs, fatty cheese, light wine, coffee, tea and carbonated water are also given. In *young persons*, carbohydrates should be given with great caution, as carelessness in diet is more frequent and the glycosuria more often progressive. The patient may not tolerate, *e. g.*, 90 gm. bread yet pass no sugar on 80 gm. bread plus 20 gm., potato, 300 gm. milk and 20 gm. oatmeal. The limits of tolerance for carbohydrates should be closely watched. All carbohydrates must be forbidden excepting bread and potatoes, in addition to which the patient should have butter, 16 pats (each 9 gm. = 80 calories) with bread, cheese and potatoes (480 calories); 2 eggs (150 calories); olive oil 6 tablespoonfuls (750 calories) with salad, cucumbers, etc. (90 calories); 30 gm. of fatty cheese (115 calories); 1 quart of milk (590 calories); and one ounce of alcohol; and meats (*v. s.*). (*b*) *Moderately severe forms* embrace cases in which the excretion of sugar is reduced to 1 or 2 per cent. only by total abstinence from carbohydrates. The rise or fall of the tolerance limit should be watched every month, and every few months all carbohydrates must be withdrawn for three weeks. The diet must contain much fat; butter, 3 ounces (gm. 110 = 800 calories), must be weighed out every morning and eaten on bread, vegetables, meat, fish, etc., either in solid form or melted. Olive oil should be taken on salad (lettuce, chicory, cress, cucumbers, tomatoes, red cabbage), and as mayonnaise dressing for salad, cold meat, fish, lobster, etc. Bacon, 5 eggs and one ounce of brandy (280 calories) should be given. In addition the patient receives meats and some articles mentioned in the conditional list. (*c*) In *severe forms*, despite a continued rigid diet, sugar and acetone are constantly excreted. These cases usually occur with emaciation, complications and a fatal issue after a few months or a year. Careful dietetic treatment may retard the progress of the disease, prevent complications and delay the fatal issue (*v. i.* Acidosis).

2. General Treatment.—(*a*) *Exercise* lessens glycosuria and is permissible in mild cases, guarded in severe cases and interdicted in acidosis; fatal syncope and diabetic coma may follow exhausting exertion. (*b*) The *mouth* and *skin* require special attention. Cleanliness averts in large part gingivitis, furunculosis and genital complications. Frequent warm (not cold) baths and change of underwear, gentle brushing of the teeth and soothing salves for eczema, and similar eruptions are imperative. (*c*) Diabetics for the most part should be kept at home and at work, for they often become restless and desire to travel. A prolonged stay in a quiet place is better. Sea bathing and exposure to cold and dampness increase glycosuria. (*d*) *Suggestive* treatment and optimism are needed with depressed subjects. (*e*) *Mineral springs* are suitable only for mild cases; Carlsbad and other spas are contra-indicated in severe, youthful, very aged, reduced and nephritic cases. (*f*) The *mode of life* should be quiet and worry avoided.

3. Treatment of Acidosis.—If the patient has been on a mixed diet, the carbohydrates are gradually reduced; if he has been carefully dieted, the protein is reduced, and vegetables, carbohydrates, oatmeal and

alkalies are indicated. Often glycosuria and ketonuria are refractory, till protein is reduced to 200-150 gm.; vegetable is better than animal protein. The fatty acids of metabolism are burned in the fire of carbohydrates; *alcohol* is thus valuable and also *oatmeal,* which is poor in protein and stimulates sugar combustion, especially when preceded or followed for a couple of days by a green vegetable diet or actual starving. Oatmeal, 250 gm., boiled in Oiij of water and strained while hot is mixed with butter 250 gm. and 8 eggs; 200 gm. are given every three hours, as gruel or fried, with which coffee, tea or brandy is allowed. It may induce diarrhea, but increases sugar tolerance. Its favorable action is lacking when given steadily with meat, or in mild cases or after several trials. Levulose may operate equally well. Sod. bicarb. and citrate neutralize ketones and facilitate their renal elimination; a teaspoonful is given every three hours by mouth if swallowing is possible, 4 tablespoonfuls in water (Oij) by rectum (drop method) or in impending coma, a 5 per cent. solution intravenously, after phlebotomy if there is hypertension; subcutaneous injection may cause sloughing. Coma (acidosis, protein poisoning, dehydration) indicates large quantities of water. Like salt, the alkalies may be retained, causing increase in weight or even pulmonary edema. Levulose (10 per cent.) may be given by mouth, subcutaneously, intravenously or by rectum, lessening acidosis and not augmenting glycosuria.

4. Treatment of Types.—(*a*) Minimal or *transitory glycosuria* should be regarded as diabetes and dietetic carelessness avoided, not giving over 150 gm. of bread daily. (*b*) *Neurogenous* diabetes; nervous conditions are more potent than diet, excitement increasing and rest decreasing the sugar, which often amounts to 6 per cent. with 1500 c.c. urine; strict diet is poorly stood; bromides, opium or acetanilide may be indicated but opium alone exerts an appreciable influence upon the glycosuria (Dobson 1776); it affects the severe more than the light type, *i. e.,* it decreases the transformation of albumins into sugar, but does not control the transformation of starch into sugar; the average dose may be gradually increased without the patient exhibiting toxic symptoms or suffering from its reduction afterward. Codeine, gr. j–x, is less effective than opium; it may be combined with sodium bromide ʒss. *Neuralgia* (*q. v.*) and neuritis should be treated dietetically and by salicylates. (*c*) *Renal* diabetes is rare; hyperglycemia is absent and diet is without influence, the patient even standing 100 gm. glucose. (*d*) *Tuberculosis* is serious and allows of only moderate dietetic restriction; if incipient, the diet may be rigid. (*e*) *Gangrene* should be treated expectantly until there is a possible halt in the process and definite demarcation; positive rules of treatment are laid down with difficulty. If there is no acidosis, diet is usually efficacious; if acidosis, surgery is indicated (after oatmeal, alcohol, alkalies and much water); advanced diabetics stand operative shock and anesthesia poorly and most die from rapid syncope or dyspneic coma. Other operations may be performed, when indicated as absolutely necessary life-saving procedures and only, if possible, after strict dieting. Boils and furuncles are excised, not incised. (*f*) In arteriosclerotics and lithemics, protein should not

exceed 100 gm. Sudden carbohydrate reduction should be avoided in obesity. Uremia and decompensation contra-indicate strict diets and constipation, drastic purges.

DIABETES INSIPIDUS.

Definition.—A chronic malady characterized by large amounts of urine of low specific gravity. Willis (1674) differentiated between diabetes mellitus and insipidus (without taste, *i. e.*, non-saccharine); 55 cases occurred in 113,600 in the Berlin Charité.

Etiology.—Two groups of cases are observed: (*a*) the *idiopathic* (nervous or vasomotor) and (*b*) the *symptomatic*, which occur in (i) *brain disease*, especially basal syphilis and less often traumatism, basilar meningitis, apoplexy, lesions of the hypophysis, medulla, fourth ventricle, middle cerebellar lobe or corp. trapezoides, etc. (ii) It may result from *abdominal* diseases, aneurysms, tumors, kidneys (losing their power to concentrate the urine) or tuberculous peritonitis. (iii) Malnutrition with tuberculous, diabetic, syphilitic or gouty ancestry, acute infections and alcoholism are numbered among its possible causes. Weil collected 36 cases among individuals of four generations, all of whom enjoyed good health. Rare congenital cases are recorded. Most cases occur in *males;* 10 per cent. are under five years old, 14 per cent. under ten and 45 per cent. were between ten and twenty-five years of age.

Symptoms.—The onset is usually gradual, though abrupt symptoms have followed fright. (*a*) The *urine* is *greatly increased*, 20 to 40 pints daily, even 90 pints and more at night than by day; less urine is excreted than water ingested. It is passed rapidly (tachyuria). Its specific gravity ranges between 1000.5 and 1005. It is pale and sometimes the urea, chlorides and phosphates are increased, being washed out from the tissue. Albumin, sugar and inosite are very uncommon. (*b*) *Polydipsia* is secondary, though perhaps the earliest symptom noticed. In one case, a physician with brain syphilis, 2 gallons of water were drunk each night between 10 P.M. and 7 A.M. (*c*) The *skin* is dry; carbuncles are rare. The saliva is decreased. Phenomenal polyphagia marks some cases. Headache, irritability, increased knee-jerks, impotence and severe lumbar pain radiating into the legs, are quite frequent. Nutrition is often good in the idiopathic cases and no unusual metabolic findings are noted; in the symptomatic variety the nutrition depends on the causal disease; in children growth may be retarded. The temperature is low and chilling occurs readily. Aside from causal lesions, there is no constant autopsy finding except hypertrophy of the kidneys from increased activity, and sometimes dilatation of the bladder and renal pelvis.

Diagnosis.—Differentiation is required from (*a*) *diabetes mellitus*, with which it has no common symptoms except polyuria and polydipsia; there is no glycosuria, acidosis, gangrene, carbuncles or neuritis; (*b*) from *interstitial nephritis*, in which the specific gravity is higher, the urine is less abundant, is albuminous and contains casts, and which is associated with cardiovascular, retinal and uremic manifestations; (*c*) from *primary polydipsia*, which is exceedingly rare; and (*d*) from *transi-*

tory polyuria, distinguished by its course or etiology, as recent fever, hysteria, etc.

Prognosis.—The prognosis is more favorable in idiopathic forms. Less than one-half of the patients recover, though death results less frequently from the disease itself (save cerebral or abdominal tumors) than from intercurrent infections, notably pulmonary tuberculosis.

Treatment.—Aside from antisyphilitic therapy, drugs are of little use. Iodides improve some non-syphilitic cases. Salt or water restriction may work benefit. Opium may be useful and large doses are often well tolerated. Ergot (fluidextract ♏xx, t. i. d.), sodium bromide (ʒss, t. i. d.) and acetanilide (gr. v–x, t. i. d.) are recommended. Talquist advocates a vegetable diet.

GOUT.

Definition.—An obscure metabolic disease, characterized by acute periodic or chronic inflammation of the joints with uratic deposits.

Mentioned by Hippocrates, gout was clearly described by Sydenham (1683), who suffered from the affection for forty years. Forbes (1792) and Wallaston (1797) recognized its uratic nature.

Etiology.—1. The *actual cause* is unknown. Garrod (1848) held that uric acid accumulated periodically in the blood before the gouty seizure. His, Vogel and Magnus Levy maintain that in gouty subjects it is increased all the time, and that the urine contains less uric acid before and more during and after the seizure. Pfeiffer holds that it is really less a question of its amount than of its insolubility or form. The endogenous uric acid (which constitutes 90 per cent. of the purin bodies) is derived from the nucleins of the body tissues and the exogenous uric acid from the food nucleins. The subject is still much involved but it appears that the uric acid *elimination* (perhaps from reduced renal action) is at fault rather than any increased formation or suboxidation of the acid.

2. *Predisposing* factors. (*a*) *Heredity* is a factor in 60 per cent.; transmission is more common by the father than mother and to the younger rather than the older children. (*b*) *Mode of life;* lack of exercise, overeating, meat diet and alcoholism are potent factors. The old verse read that "Wine was the father, Eating the mother, and Venus the midwife" of gout. Though gout often affects the well-to-do (arthritis divitum) and arthritis deformans the poor (arthritis pauperum), gout is not uncommon among the lower classes. Heavy ales and beers are more injurious than whisky and light wine. Gout may occur in temperate individuals. Obesity, diabetes, arteriosclerosis and calculous tendencies are concomitant rather than causal conditions. (*c*) *Lead poisoning* is observed in 15 per cent. of cases, particularly in England; saturnine nephritis may explain it. (*d*) *Men* are particularly prone to gout (1 to 13 or 25). (*e*) *Age:* Gout begins between thirty and forty years in 38 per cent., thirty and fifty in 60 per cent., and before the seventeenth year, less than 1 per cent.; Gairdner observed 1 case in a nursling. (*f*) England leads in frequency of its occurrence. In the United States it is rare, though Futcher shows that the cases in Baltimore and London hospitals stood as 2 to 3. Few cases seek hospital treatment.

Symptoms.—These may be considered under two captions, (1) acute, and (2) irregular, chronic and visceral gout.

1. *Acute* (regular, sthenic or) *typical* gout usually appears without prodromes, though sometimes an attack is heralded by digestive disorder (pyrosis, gastralgia), cerebral congestion, vertigo, tinnitus, irritability, paresthesia, cramps or pain in the muscles or dark lateritious urine. The *paroxysm is remarkably characteristic.* (*a*) *Local findings:* The patient is awakened one to three hours after midnight by an agonizing pain in the metatarsophalangeal joint of the large toe (*podagra*), described as crushing by a vise or burning by a hot iron. Localization in the toe (95 per cent., Garrod) is practically diagnostic of gout; the skin becomes red, tense, slightly edematous and exquisitely sensitive; the shorter the attack the severer are the symptoms. Serum exudes into the joint. The pain abates toward morning, but the inflammation increases. Each early morning sees a repetition of the pain for three to five or less often six to seven days; the paroxysmal pain and the local findings then gradually subside; in two-thirds of the cases, the skin desquamates over the joint. Later attacks involve the thumb, knee and other joints, less commonly than in chronic gout (*v. i.*). The *pathological changes* probably develop in this sequence: (i) local stasis (inflammation), (ii) uratic deposit; (iii) necrosis; the ordinary urates (biurate of sodium in conjunction with sodium phosphate) do not cause such local reaction, but the biurate alone, even in most dilute solutions (0.004), produces inflammation; residual swellings (acute gouty tophi) consist of this biurate precipitated by the carbonates of the blood and tissues. Convalescence is complete in ten days, often with greatly improved spirits and health. During the seizure (*b*) *constitutional symptoms* are present, as the temperature, 100.5° or 101°; the pulse is slow and hard, sometimes arrhythmic and seldom exceeds 100. The heart may palpitate. Slight leukocytosis, nausea, vomiting, epigastric cramping and eructations are not uncommon. The urine is dark, decreased, acid and higher in specific gravity; Bain and Futcher find the uric and phosphoric acids, both derived from disintegration of nuclein, fall below normal in the quiescent period and increase during the attack.

Subsequent acute seizures are likely to occur, notably in the spring and fall, precipitated by excesses in alcohol or eating, trauma, exertion or excitement; in one case it would occur within fifteen minutes from a glass of champagne.

Retrocedent or *suppressed gout* was once thought to develop when an acute paroxysm abated "too rapidly"; many of its symptoms are doubtless coincident arteriosclerosis or uremia (*v. i.*).

2. *Chronic, irregular, atypical gout* generally follows acute attacks which increase in frequency, decrease in acuity and show slight tendency toward resolution. Sometimes *tophi* appear to begin as chronic gout, particularly in the aged, cachectic or women of a gouty family, all of whom lack reactive power (asthenic gout). (*a*) *Local findings:* There are less pain, fever and redness but greater and more permanent swelling than in the acute form, until the condition eventually becomes chronic; the "attacks" may be rudimentary and remissions alternate with exacer-

bations. The *lower extremities* and hands are most often affected; it may develop in the thumb (*chiagra*), knee (*gonagra*), elbow (*pechiagra*) and spine (*rhachisagra*), but the shoulder is very seldom and the hip almost never invaded. In some cases its moving from joint to joint (*urarthritis multiplex seu vaga*) may cause confusion with acute rheumatism. Uratic deposits (*tophi arthritici*) form in the cartilage and later in the capsule and ligaments, and the swelling never recedes essentially. These prominences and the attendant fibrosis lead to deformation, subluxation, fluctuation, crackling, anyklosis and contractures. The proximal joints of the fingers may point to the radial and the others to the ulnar side. Tophi may rupture, evacuating yellowish-white urates, though incompletely, as they are partly diffused through the tissues; these gouty ulcers heal tardily, if at all, for their basis is fibrous and indolent. Tophi occur also on the ear (25 per cent.), bursæ, tendons, aponeuroses (which may explain some cases of Dupuytren's palmar contracture), and even in the skin, eyelids, nose, larynx, penis or scrotum. (*b*) *Visceral gout* includes a curious mixture of real gouty complications and incongruous doubtful symptoms (*arthritisme*, *herpétisme* and the "uric acid diathesis").

Complications.—*Renal*, the most important visceral complications, sometimes dominate the clinical picture. In 1440 cases, Garrod observed albuminuria in 26.5 per cent., more often in advanced than in early cases. It may follow the metabolic changes, or may possibly constitute a primary gout. At first albuminuria may be considered toxic, congestive or arteriosclerotic, but later the renal, cardiovascular and retinal findings of interstitial nephritis develop with all its accidents; uratic deposits occur in the intertubular tissue and in 15 per cent. in the secreting tissue of the cortex and medulla, or as calculi in the renal pelvis; hematuria, oxaluria and pyelitis may occur.

Arteriosclerosis of the ordinary type is promoted by metabolic changes, alcoholism, plumbism and nephritis; with nephritis, it causes most of the so-called visceral symptoms, as the *cerebrospinal* (headache, vertigo, congestion, epileptiform and apoplectiform attacks, encephalomalacia), the *circulatory* (hypertrophy, myocarditis, angina, valvular lesions, phlebosclerosis), the *respiratory* (stasis, bronchitis, asthma) and retinitis.

Eczema (18 per cent.), scleritis and episcleritis (with circumscribed points of inflammation and sometimes semimicroscopic tophi), neuralgia or less often, lumbago, sciatica and brachial neuritis, burning, psoriasis-like patches on the tongue, digestive symptoms (*v.* PRODROMES) and burning in the eye-balls are probably often gouty. On the other hand, many so-called visceral gouty manifestations are neurotic, paroxysmal (calculous colic, migraine or asthma) and other conditions common in persons not suffering from gout.

Diagnosis.—(*a*) *Acute gout* is most typical in its etiology, history, acuity and localization. In a few cases (urarthritis multiplex), a diagnosis of acute rheumatism may be made; fibrous rheumatic nodes may be confused with tophi, but if the possibility of gout is recognized there is usually no error. (*b*) *Chronic gout* is suggested by a history of acute attacks, involvement of the distal joints of the hands and feet, unequal

distention and thickening of the soft parts and often by its association with early arteriosclerosis, contracted kidney, obesity, renal calculus and diabetes. The *x*-rays sometimes show the tophi as light spots and the joints smooth. Visceral gout is an entity only when it develops in a patient undoubtedly gouty.

Prognosis.—Gouty patients may live to an old age, but with a tendency to chronic gout, of which the issues are (*a*) uremia, (*b*) arteriosclerosis, as coronary disease or apoplexy or (*c*) terminal pericarditis, pneumonia or pleurisy, induced by the (*d*) gouty marasmus. In general the outlook is better in frank than in irregular gout and better in cases which develop after forty years of age.

Treatment.—1. Acute Paroxysm.—(*a*) Absolute *rest* is indicated. (*b*) For *pain,* the use of *colchicum* is empirical (Storck, 1763) but it relieves pain and inflammation and acts as a cholagogue (℥iss of the vinum colchici radicis [not seminis] daily). It requires care in nephritic and marantic subjects. It should be stopped when pain is alleviated and diarrhea intervenes. Full therapeutic doses produce a slow pulse, vomiting, pain and purging; inversely to its action on the bowels, there are diaphoresis and diuresis. It is eliminated by the kidneys, bowels and skin. After toxic doses, retching, serous and finally hemorrhagic purging, great pain, prostration, paralysis of the sensory nerves, motor cord and respiratory centre (the usual cause of death) develop.

If colchicum produces no effect, *sodium salicylate* (℥j–ij daily) usually relieves the pain and swelling and aids in solution of the uric acid; its early effect is greatest and it usually fails after the third day. *Acetphenetidinum* (gr. viij–x) and morphine are indicated in severe paroxysms. In prolonged attacks, potassium iodide, gr. v (given with care in nephritis) and wine of colchicum ♏x are useful. *Atophan* (gr. vij, t. i. d., for 4–5 days) increases the uric acid output three- or fourfold, more efficiently than salicylates and is less irritant to the stomach when combined with alkalies. (*c*) The *diet,* according to Pfeiffer, should consist of foods which "release uric acid in a free form," as meal soups, rice and other carbohydrates; meat, eggs, alcohol and lemonade are withheld. Plain or acid water, as Seltzer (not alkaline water as Vichy) and hydrochloric acid, are given freely. (*d*) *Local measures* induce necrosis and tophi, especially vesicants, the ice-bag and massage; warm applications of 50 per cent. alcohol or lead-water (tinctura opii, liquor plumbi subacetatis āā ℥ij and water ℥j) may be used.

2. Between Attacks.—(*a*) Regulation of diet is the cardinal indication. Moderation in the quantity is more important than the quality of food. *Quantity:* a full diet increases the uric acid, taxes resorption which is usually impaired and favors acid fermentations which engage the diphosphates and carbonates of the food necessary for the formation of soluble uric acid salts; three medium-sized meals are allowed. *Quality:* (i) *alcohol* is absolutely forbidden; (ii) *water* may be given freely on an empty stomach. (iii) Moderate amounts of *fat* are allowed. (iv) *Fruits and vegetables* increase diuresis and their malates and citrates help to dissolve the uric salts. (v) *Meats* are allowed; urea seems to dissolve uric acid which the meat does not increase if given in moderation (meat

1 part, vegetables 3 parts of the diet). Meat is essential in marantic cases. There is no difference between the effect of light and dark meats; those rich in purin (sweet-breads, liver and kidneys) are avoided; milk and eggs are purin-free. (vi) *Carbohydrates* should be largely *decreased*, though following their ingestion the uric acid (as compared with the urea) increases nearly twofold. In severe cases the purin contents of peas, beans, corn or oats, contra-indicates them. (*b*) *Exercise* is indispensable, avoiding fatiguing effort, hurry and worry. (*c*) *Medicinal treatment* is secondary. Alkalies are useless, though claimed to produce an increased output of uric acid; they operate only by preventing decomposition of the neutral (bi-) sodium phosphate (the chief uric solvent), *e. g.*, lithium carbonate, sodium bicarbonate or mineral waters. Luff advises guaiacum resin, gr. v–x, t. i. d. Piperazin, lysidin, urotropin, urosin and sidonal are inert.

3. Chronic Gout.—This is difficult to treat. For general dietetic, hygienic and medicinal measures, see above. Colchicum wine, ♏x, and potassium iodide, gr. v, may relieve arthritis, asthma, neuralgia and eczema. Tonics, principally arsenic, are indicated in marantic forms. Tophi should not be operated on; local measures are useless, save Bier's stasis, gentle massage, dry heat or salicylate salves (ac. salicylic. 4, lanolin 40, and oleum terebinthinæ 1 part).

RICKETS.

Definition.—A metabolic affection of early infancy, characterized chiefly by hyperemia and deficient calcification of the growing bones. The disease was first well described by Glisson (1650). The name is derived from an old English word meaning "twisted," and Glisson suggested the Greek "rhachitis" (vertebral inflammation) because the spine was often involved, and the term sounded like "rickets."

Etiology.—All theories advanced, such as abnormal calcium metabolism, infection or reduced activity of the thymus, are insufficient. (*a*) The *diet* is of prime importance; proprietary foods, condensed milk, cow's milk, excess of carbohydrates, prolonged lactation or nursing during pregnancy are probable factors. A deficiency in animal fats and proteids is causative; after 20 litters of lion cubs had been lost from rickets in the London Zoölogical Gardens, Bland Sutton found that a diet of milk, powdered bone, meat and cod-liver oil prevented it. (*b*) *Unsanitary surroundings* may be causal, as lack of fresh air and sunlight, crowded quarters and kindred factors observed largely among the poor classes in large cities; among nurslings Kassowitz (Vienna) observed it in 89 per cent. and Joucousky (St. Petersburg) in 90 per cent.; in the United States it is most frequent among negroes and Italians. Hereditary syphilis is a predisposing factor. (*c*) *Age.* In over 600 cases Baginsky did not observe it in children under three months of age; his figures show 7 per cent. in children between three and six months and 88 per cent. between three months and two years. The occurrence of congenital rickets is usually denied. Rickets which develops later, up to puberty, is called *rhachitis tarda.*

Symptoms.—An experienced observer may diagnosticate the gradual onset from the *prodromes,* as the irregular or tardy dentition; irritability, insomnia and throwing off of the bedclothes; sweating about the head and neck; bronchitis; digestive disturbances, anorexia, malnutrition, meteorism or diarrhea; immobility, the child crying when moved or touched and general hyperesthesia. However, a positive diagnosis is not justifiable until the bone symptoms develop.

1. Bone symptoms are most marked in the skull and thorax. (*a*) The *skull* shows the earliest changes, generally in the first year of life. The *face appears small* and the *head large.* Nodes of bone appear on the frontal and parietal regions, which with the occipital flattening produce the caput quadratum (*tête carée*). The anterior *fontanelle* and sutures (which normally close at the fifteenth or eighteenth month) *remain open,* even until the fourth year. The occiput softens (*craniotabes*) in 40 per cent., near the lambdoid suture; the occipital protuberance remains hard. Craniotabes may less frequently occur in the frontal bones; besides the actual bone changes (*v. i.*), the pressure of the pillow and brain further its development. In extreme cases the peri- and endocranium actually touch, and in moderate degrees the skull feels "parchment-like." Though it occurs also in syphilis, craniotabes is rather characteristic, with the tender quadrate head, head sweating, thin occipital hair and blue scalp veins. The frequent *systolic murmur* heard over the greater fontanelle is also heard in other affections or in health and is said to occur only in conjunction with a similar bruit in the carotids. The *upper jaw* becomes long and narrow and the lower jaw hexagonal, from muscular traction on the soft bones. The eruption of the teeth is delayed until the second or even the third year; the second teeth are crowded, carious, curved and eroded. (*b*) In the *thorax,* the rhachitic "*rosary*" appears toward the end of the first or early in the second year; it is so-called from the bead-like succession of enlarged chondrocostal joints—enlarged epiphyses. The *chest* is altered toward the end of the second year, due to the softness of its bones, traction exerted by the diaphragm, atmospheric pressure and sometimes by external influences, as holding of the child with a hand on the chest. Louis's angle is often prominent, and the sternum juts forward, causing the "*chicken breast*" (pectus carinatum). There is also a depression outside of the costochondral junctions which extends outward and downward, and is accentuated by some eversion of the costal arch. The thoracic deformity promotes respiratory complications and dyspnea, which in turn aggravate it. With this deformity we may group lumbar *kyphosis* which is arcuate and not angular, the thickened or bent clavicles and the thickening of the spine or edges of the scapulæ. (*c*) In the *extremities,* the *epiphyses,* particularly of the ulna, radius, tibia and fibula, *are enlarged,* at the same time as the rosary, craniotabes and wide fontanelle. The epiphyses also are tender and painful and may look like "*double joints.*" Deformities may develop from muscular traction or the weight of the body; the arms may incline outward and forward or the forearms become convex toward their extensor surfaces. The thighs sometimes become convex anteriorly and outwardly; the legs bow outward or less often

forward, especially when early walking is encouraged. Spiral deformities are less frequent. The gait is waddling. Green-stick or actual fractures sometimes occur. (*d*) The *flat rhachitic pelvis* develops by the weight of the body pushing the sacrum into the pelvis and the counter-pressure of the thighs narrowing the anterior pelvis, so that the pelvic aperture may become heart-shaped, the acetabula lie more anteriorly and the iliac crests flare outward. (*e*) *Pathology.* Normal bone grows in length as follows: there are two zones in the epiphyseal surface facing the shaft; (i) the *first zone* is nearest the shaft, is faintly yellow and measures 2 mm.; it is the zone of preliminary calcification, into which normally the bloodvessels grow and form medullary canals by absorbing small areas of bone, and in which osteoblasts appear and calcification develops. In rickets the calcification is imperfect and irregular in this zone and the bloodvessels grow not merely into these calcified areas but beyond them, even into the cartilage area, and absorb what little new bone is formed. (ii) The *second zone* lies outside the one described, is sharply demarked from it, is normally bluish, measures 1 or 2 mm. in thickness and is known as the hyperplastic zone, in which the cartilage cells divide and proliferate. In rickets this zone becomes larger, proliferates irregularly and is invaded by bloodvessels and irregular foci of abortive calcification; the demarkation between the first and second zones is ill-defined. Normal bones grow in thickness by bony increase beneath the periosteum. In rickets this layer becomes several millimeters thicker, irregular and nodose. Normal bone develops in the skull and face in the connective tissue. In rickets the lime salts are either not deposited, are insufficiently deposited (craniotabes) or overdeposited as bosses (osteosclerosis). The essence of this inflammation or dystropy is *undue hyperemia* and *irregular, deficient calcification;* which one is primary remains a disputed point; the bones may contain but 20 to 30 per cent. of the normal amount of lime salts.

2. General Symptoms.—(*a*) *Muscular weakness* is pronounced, as evidenced by impaired breathing, the tardy efforts to hold up the head and disinclination to walk; in this myopathia rhachitica there is a great reduction of the caliber of the muscle fibers, much nuclear multiplication and increase of their longitudinal and decrease of their transverse striation. (*b*) *Nutrition;* there is anemia, sometimes with leukocytosis and often with emaciation. (*c*) *Nervous system:* Mentality is unimpaired; rhachitic patients frequently grind their teeth; spasmodic conditions are not infrequent, as generalized *convulsions, tetany* and *spasm* of the *glottis.* (*d*) Temperature always denotes complications. (*e*) *Digestive disturbances* are common; there may be anorexia, diarrhea, more commonly constipation and *distention* of the *abdomen,* from enlargement of the liver and spleen, and possibly from thoracic changes. The amount of chalk in the feces may be increased. The *spleen* is *enlarged* in 70 per cent., and, less often and less markedly, the liver. (*f*) Rickety children *catch cold easily* and bronchitis frequently leads to atelectasis and bronchopneumonia. (*g*) The heart may be luxated from the altered thoracic conformation, which frequently causes hypertrophy of the right ventricle. (*h*) Eczema is common.

Course, Complications and Prognosis.—(*a*) *The onset* is insidious and the clinical *course covers a year* or more. (*b*) *Complications* include but few of immediate danger, as bronchitis, pneumonia and spasm of the glottis. Later complications include the contracted rhachitic pelvis of obstetrical importance, kyphosis and pulmonary tuberculosis, which is found in 36 per cent. of the fatal cases; its early diagnosis depends largely upon auscultation, for percussion is peculiarly deceptive when the chest is deformed. (*c*) The prognosis is, with these exceptions, excellent as to life, and with care, as to deformity, for deformities may regress.

Diagnosis.—The diagnosis is based chiefly upon the bone changes. Errors are possible from precipitate diagnosis, based on the prodromal phenomena. (*a*) *Barlow's disease* (*q. v.*), misnamed acute rickets, has no connection with it. (*b*) *Syphilis* predisposes to rickets but is otherwise distinct and separate; it usually develops at a time (first to sixth month) when rickets is most uncommon and is usually stamped by other syphilitic stigmata, as cutaneous and mucous syphilides; when it invades the epiphyses, the femur is usually its seat, and between the cartilage and bone, the wall-like, very tender, painful and doughy swelling is rather fusiform, compared with the rounder rhachitic nodes; epiphyseolysis may occur in syphilis, but never in rickets; in doubtful cases an *ex juvantibus* diagnosis and the sero-test may be made. (*c*) *Vertebral tuberculosis* is easily distinguished; the rhachitic kyphosis is arcuate, not angular.

Treatment.—1. Hygiene.—Fresh air and sunlight produce wonderful results, particularly in the country, mountains or at the sea-side.

2. Dietetic.—In nurslings (who seldom develop the disease), a wet-nurse may entirely cure the disease. In older children it may be less a question of food than of digestion.

3. Medicinal.—No specific is known, but *phosphorus* is more important than any other drug. Under its use the child becomes easier and brighter; the nervous symptoms abate, as insomnia, convulsions, tetany or laryngospasm; the osseous symptoms are improved, bone is deposited and dentition occurs, both clinically and in experiments. Phosphorus itself is given in doses of gr. $\frac{1}{200}$—$\frac{1}{100}$ in pill form or as the ol. phosphoratum (1 per cent. solution) ♏j, t. i. d., or better, *in 1 per cent. cod-liver oil;* the oil or emulsion should be freshly prepared, for both deteriorate readily on exposure to light. (Its toxic symptoms are fever, thirst, anorexia and weakness; vomiting of food, mucus, bile, later blood and sometimes phosphorescent material; abdominal pain, diarrhea or constipation, phosphorescent, clay-colored or bloody feces; jaundice; the liver at first may be large, but becomes small; there may be cholemia; albumin, bile, casts, leucin and tyrosin in the urine and hemorrhages from the uterus, abortion or miscarriage and bleeding from other surfaces.) *Cod-liver oil* was recommended by Bretonneau in 1827; if it is well tolerated by the stomach, the general level of health may be elevated by its use; if not, cream and butter should be given. *Iron* is a good tonic. It is proved that a sufficient quantity of calcium salts is present in the food.

4. Orthopedic.—During the florid stage the child should be kept quiet upon a firm mattress and off his feet. Later, corrective measures are indicated for the kyphosis and deformity of the limbs.

OBESITY.

Etiology.—Obesity (adiposity, corpulence or polysarcia adiposa) is favored (*a*) by *heredity;* it frequently occurs in entire families; congenital cases are known; (*b*) by *mode of life, e. g.,* lack of exercise, overeating and alcoholic indulgence. (*c*) The *menopause* is frequently attended by increase in weight. (*d*) *Diseases* of the thyroid or hypophysis or nervous or osseous system; and gout, diabetes, anemia, valvular disease and emphysema, which act rather by preventing adequate exercise than by deficient oxygenation.

Symptoms.—The general *habitus* requires no description. Fat develops in the subcutaneous tissues, in parts where it is normally found, as in the omentum and around the kidneys and it may cause fatty infiltration and degeneration of all internal organs excepting the brain and spleen; even the blood may contain fat (lipemia).

1. Cardiac.—The heart is embarrassed by increased resistance to the capillary flow, fatty infiltration of the myocardium, and fat accumulation in the chest wall, omentum and mediastinum. The pulse is more often frequent than slow. Hypertrophy may occur. Edema is not uncommon.

2. Respiratory.—The breath sounds and heart tones are faint and thoracic percussion is difficult because of the subcutaneous fat. *Bronchitis* is common and probably results from the ready sweats and consequent chilling. Dyspnea may occur on exertion.

3. Digestive.—Digestive disturbances may develop. The large liver is less frequently fatty than congested and hypertrophic. Constipation and hemorrhoids are frequent.

4. Urinary.—The urine may be scanty in oxaluric, lipuric, lithuric, albuminuric or glycosuric subjects. Actual impotence is not uncommon, as well as mechanical obstacles to coitus.

5. *Lessened physiological resistance* to infections, surgical measures or phlebotomy, and (6) such *associated affections* as diabetes, gout, calculous disease and less often contracted kidney, arteriosclerosis and apoplexy may be noted.

Treatment.—Treatment is (*a*) that of the fundamental condition and (*b*) *dietetic.* The various "cures" enumerated possess more than historical interest—(i) *Banting's cure.* Banting, a merchant, was treated so successfully by Wm. Harvery for obesity that he addressed a "Letter to the Public on Obesity" (1863). This diet, largely *albuminous,* consisted of fish, meat, eggs, tea without sugar or milk, vegetables, fruit, some red wine and toast without butter. It may cause indigestion, nervousness, palpitation or gout. (ii) *Ebstein,* himself a sufferer, proposed (1872) a diet with larger proportions of *fat;* his diet consisted of meat, fish, eggs, tea, leguminous vegetables, fruit, fats and butter, but restricted the carbohydrates; digestive disorders may also occur under this system.

(iii) *Oertel* allowed *more carbohydrates,* limited the fats and fluids and insisted upon sweats and graduated exercises. From the following it will be seen that all these methods are "starvation cures" The average man requires 118 gm. of nitrogen, 56 gm. of fat and 500 gm. of carbohydrates, equaling 3054 calories.

The diet of Banting contains 172 gm. of nitrogen, 8 gm. of fat, and 81 gm. of carbohydrates, equaling 1112 calories.

The diet of Ebstein contains 102 gm. of nitrogen, 85 gm. of fat, and 47 gm. of carbohydrates, equaling 1401 calories.

The diet of Oertel contains 183 gm. of nitrogen, 38 gm. of fat, and 143 gm. of carbohydrates, equaling 1690 calories. In any reduction-cure the patient may suffer from breaking-down of the body proteids, which causes the weakness. In all cases the total amount of nitrogen in the urine, which constitutes 90 per cent. of the nitrogen excreted, should be watched; if more is excreted than ingested the nitrogenous food should be increased to obviate muscular and cardiac weakness. The diet then should consist of *moderation in all foods,* with *restriction of the carbohydrates,* whose easy reduction into water and carbonic acid spares the tissue waste. *Alcohol* should be *forbidden.* Beer contains not only 3 to 5 per cent. of alcohol, but double this percentage of soluble starch and dextrin. The *fluid* ingested should be *reduced* to 2 or 3 pints daily. A milk or vegetable diet is sometimes successful.

(*c*) *Active and graduated exercise* is indispensable, except in marked cardiac disease (*v.* Fatty Heart, Myocarditis and Dilatation).

(*d*) The patient should *sleep not more than seven hours* and should bathe daily to avoid skin complications and to promote perspiration. *Thyroid* extract is not to be taken, unless thyroid insufficiency is suspected, for it breaks down the albumins rather than the fats.

ADIPOSIS DOLOROSA OR DERCUM'S DISEASE.

This dystrophy was described by Dercum (1888), as a "disorder characterized by unequal symmetrical deposits of fatty masses in various parts of the body, preceded or attended by pain." There were only 50 reported cases up to 1909, most of which were females. It develops in middle life, chiefly after the fortieth year.

Pathology.—In the 8 autopsies the thyroid gland was affected in 7 and the hypophysis in 5. In the cases of Dercum and Burr an interstitial neuritis was found, which is likewise noted in the fatty tumors removed during life; symmetrical lipomatosis has been observed in multiple neuritis.

Symptoms.—*Two cardinal clinical features* are observed, as the term adiposis dolorosa indicates: (1) The *obesity* is either diffuse or is localized fatty tumors or both in combination; lipomata, from the size of a pea to large dimensions, occur on the trunk and proximal parts of the extremities, but never on the face, forearms, hands, legs or feet. Indeed the thinness of the wrists and legs contrasts sharply with the massive body, thighs and upper arms. The pendulous abdominal fat may hang down like an apron. In one of the author's cases the localized abdominal

fat resembled a circular life-buoy, sagging down over the thighs. In another it almost resembled myxedema, save for its circumscribed distribution and facial exemption. (2) The *pains* may be neuralgic or paresthetic.

Subcutaneous hemorrhages, hematemesis, epistaxis and metrorrhagia are not uncommon. The patients are often nervous, perhaps hysterical and lack initiative. In the later stages epilepsy has developed and death from asthenia, psychoses, nephritis or intercurrent affections is recorded. A number of cases improved under thyroid extract.

SECTION IX.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

CEREBRAL LOCALIZATION.

Autopsy findings in carefully studied cases, animal experimentation and constantly increasing surgical intervention have developed the *nature of lesions* and important results regarding their *localization*. The doctrine of Flourens that all parts of the brain were equally important met its first contradiction in Broca's discovery (1861) that motor aphasia was due to disease of the third left frontal convolution. In 1870 Fritsch and Hitzig discovered that stimulation of certain areas, now known as the motor cortex, resulted in contraction of definite muscles of the opposite side of the body. Since then Bevor, Horsley, Meynert, Jackson, Flechsig, Ferrier, Munk, Glotz, Nothnagel, Charcot, Wernicke and others contributed to the development of brain localization.

I. The Motor Cortex.—This embraces chiefly the central convolutions on either side of the fissure of Rolando (the ascending frontal and ascending parietal lobes and anterior two-thirds of the superior parietal lobe), the foot of the three frontal convolutions and on the inner surface of the hemisphere, the paracentral lobule. In this area lie the large pyramidal cells which Betz discovered were the origin of the motor or pyramidal tracts. The centres run somewhat into adjacent areas, especially those with physiological associations. The *leg centre* occupies the upper quarter of the ascending frontal and ascending parietal convolutions and part of the superior parietal lobe (all adjacent to the longitudinal fissure) and passes over to the inner aspect of the hemisphere to the paracentral lobule, which is the centre for the thigh, pelvis and gluteal muscles (Fig. 50). A lesion in the falx cerebri or dura near the longitudinal fissure may, in rare cases, involve both leg centres and produce cerebral paraplegia, although most paraplegias are spinal in origin. The *arm centre* is the largest, covers the middle two quarters of the central convolutions, lies below and somewhat overlaps the leg centre. In Fig. 51 it is seen that there are certain general as well as separate centres for the thumb and fingers. The *head centre* is below the arm, covering the lowest quarter of the central convolutions (the entire operculum), the upper part of the Sylvian fissure, the posterior part of the lowest frontal convolution and possibly the first and second frontal gyri. The

face centre lies directly beneath the arm centre, and is mostly located on the anterior ascending convolution, the upper facial component lying higher than the lower facial centres. The eye centre of the facial is bilateral, probably consisting of several foci. The mouth has several centres. The *motor speech area* is principally in the lower left frontal gyrus and is probably the chief centre for chewing. The *trunk centre* lies in the posterior part of the first frontal convolution and also on its median aspect, just anterior to the paracentral lobule; there is a trunk representation between the leg and arm centres and a neck centre between the arm and face. The more complicated the function, the larger is the centre (as for the tongue, fingers, mouth) and the more unilateral the localization in the brain. Muscles used rhythmically or in pairs are

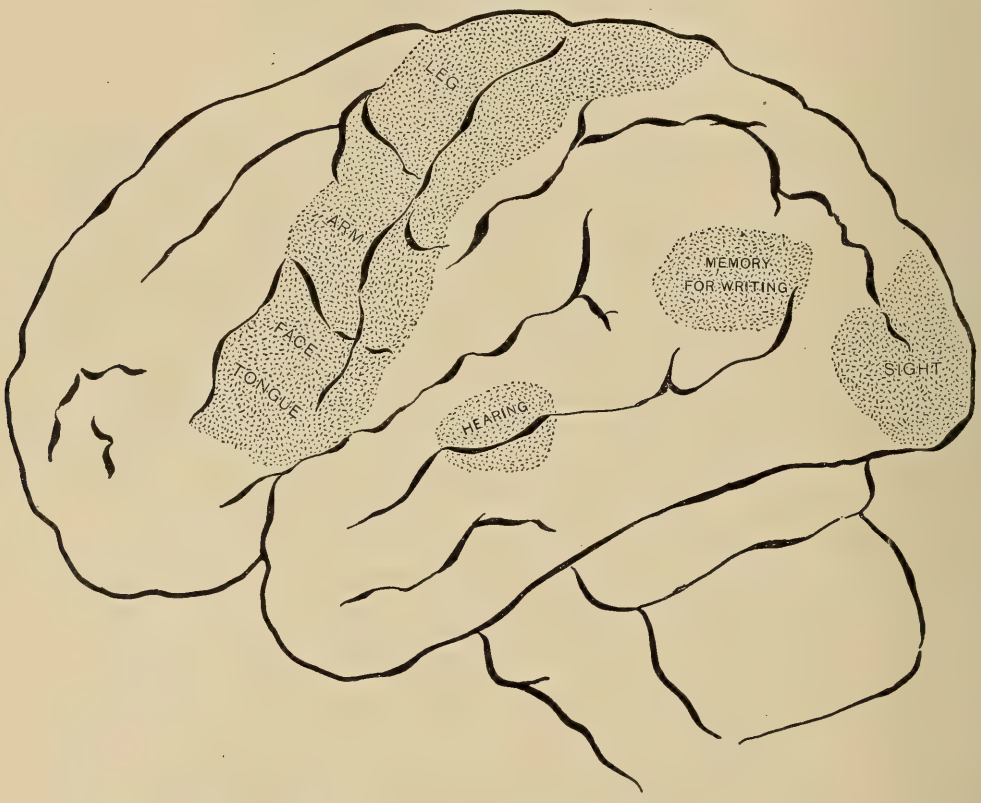


FIG. 49.—General distribution of centres in cerebral localization; outer surface of brain.

supplied from both hemispheres, as the muscles of the forehead and respiration or swallowing and to some degree the legs and trunk. Protrusion of the tongue to one side has a monolateral centre, though its protrusion in the median line and its withdrawal depend on both hemispheres.

Total lasting hemiplegia of the opposite side of the body, with sensory disturbance, aphasia and intellectual disturbance, results from complete destruction of the entire motor cortex of one side. Cortical hemiplegia is less common than *monoplegia* (paralysis of one member) which always suggests a cortical lesion. Crural monoplegia is rare and is due largely to vascular lesions in the paracentral lobule (anterior cerebral artery). Brachial monoplegia may result from vascular lesions, tumors or trauma;

the finest selection, as of the thumb alone, fingers or wrist, is noted. Sensory disturbance is common (*v. i.*). Pure facial monoplegia is unknown, although a faciolingual form with motor aphasia is observed. *Associated monoplegia* is more usual, most commonly paralysis or paresis (partial paralysis) of the arm and face (brachiofacial monoplegia, although strictly not a monoplegia). Next in frequency is the arm-and-leg type (brachiocrural) in which two contiguous centres are involved. A leg-and-face type never occurs, because their centres lie too far apart. A lesion involving one part after another speaks for cortical localization. Cortical paralyses are not attended by the reaction of degeneration. In cortical lesions with paralysis, twitchings or convulsive movements are common, "cortical, partial or *Jacksonian epilepsy;*" the lesion partly destroys the motor cortex (paralysis) and at the same time causes irritation (convulsions) precisely as contractions are produced in animals

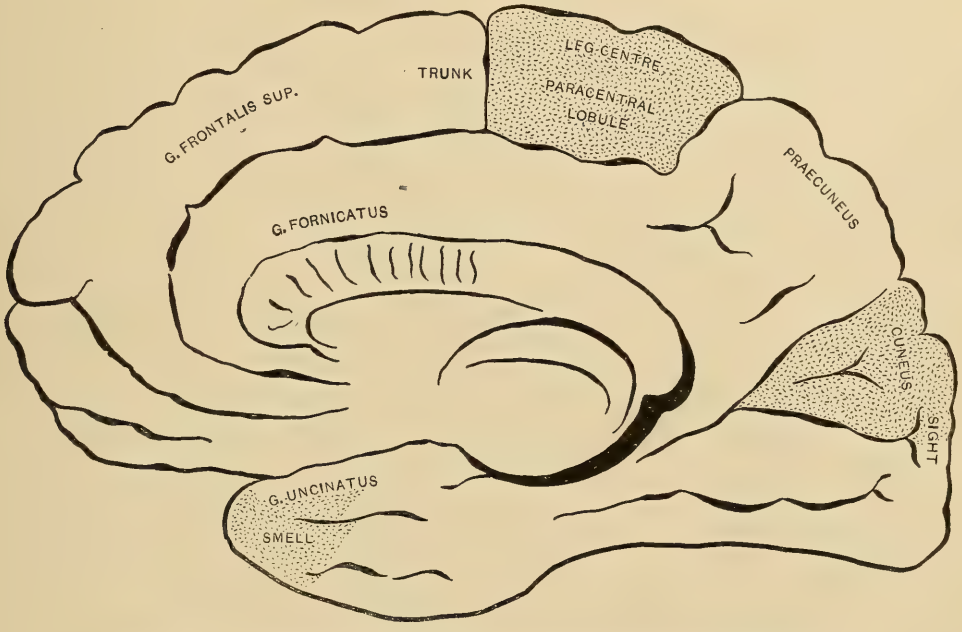


FIG. 50.—Cerebral localization; inner surface of hemisphere.

by electrical stimulation of the cortex; they are mainly (*a*) *mechanical*, as irritation or increased intracranial tension (50 per cent. of cases are rapidly growing tumors), or (*b*) *vascular* (hemorrhage, embolism or inflammation); stationary lesions rarely produce Jacksonian epilepsy unless they are located in the cortex. The spasms may involve but one part (*monospasm*), as the arm, exclusively a sign of cortical irritation, or they may pass from one centre to others. They always *commence* in one centre and *proceed in a fixed order;* if the arm region is the seat of a tumor the Jacksonian epilepsy flexes the fingers, moves the thumb, then the forearm, arm and shoulder and then passes down the cortex region, so that it involves the face, mouth and finally the leg (see Fig. 49). If the lesion is in the face area, the various facial muscles contract and the eye deviates, then the next area above participates, and finally the higher leg centre is implicated. That is, the spasm never skips over a centre.

The spasms are often preceded by a tingling in the part and are mostly clonic, or clonic broken by tonic spasms. *Consciousness is maintained*, unless the fits reach the other side, are very frequent or the lesion is deep, below the cortex. The opposite side is involved through the commisural fibers of the corpus callosum. Jacksonian differs from regular epilepsy in that no cry is observed, the commencement is local and more gradual and consciousness, if lost, disappears gradually, not precipitately. The seizure lasts several seconds to a minute or two and is followed by some vertigo, nausea, stupor or disturbance of speech; if spasms occur without paralysis, paresis in the involved parts follows,

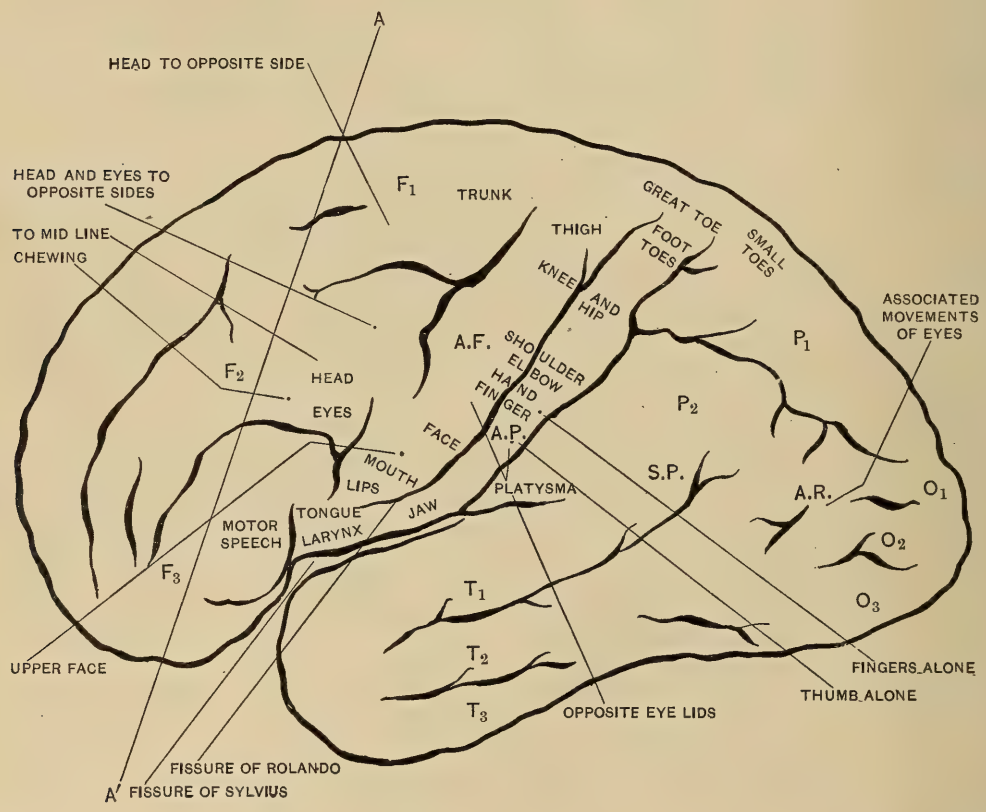


FIG. 51.—Cerebral localization in detail: F_1, F_2, F_3, first, second and third frontal convolutions; *A.F.*, ascending frontal convolution and *A.P.*, ascending parietal convolution; *S.P.*, supramarginal and *A.R.*, angular convolution; O_1, O_2, O_3, first, second and third occipital convolutions; T_1, T_2, T_3, first, second and third temporal convolutions. The explanation of line *A′*, *A* is given under INTERNAL CAPSULE.

due to transient exhaustion of the cortex. Jacksonian epilepsy in its lightest forms may be sensory.

Regarding *sensory localization* in the cortex, Munk held that the sensory and motor areas are the same (*v. i.*). *Vasomotor disturbance* is common in cortical lesions.

After total lesions of the central convolutions, the hands improve very little in adults; other members improve, to some extent, through assumption of function by the sound side of the brain; in young individuals the sound pyramidal tracts in the cord may hypertrophy to double their size. The motor fibers running from the pyramidal cells of the cortex to the anterior horns of the spinal cord, constituting the *upper motor*

neurone, undergo descending degeneration, that is, degeneration in the direction of the motor impulse.

II. The Cortex of the Parietal Lobes.—Our knowledge of function is less definite. (*a*) Lesions of the *first lobe* (P_1, Fig. 51, the precuneus in Fig. 50), supplied by the anterior cerebral artery, cause disturbance in motility of the leg, with altered muscle sense, "mind-blindness," inability to read (alexia) and perhaps hemianopsia. (*b*) Disease of the *supramarginal convolution* (*S.P.*, Fig. 51), also involving the motor cortex, produces greater disturbance of sensation than when the lesion is confined to the motor centres; muscular sense is disturbed, usually with some motor paralysis or paresis. Muscle sense is disturbed in 23 per cent. of all cases of cerebral hemiplegia; in more than 50 per cent., no special sensory disturbance is noted. There may be loss of the stereognostic sense, whereby the patient fails, when his eyes are closed, to recognize the geometric form of solid objects, as a key or ball. Cortical ataxia sometimes results, on account of disturbance in the association fibers. It differs from the tabetic and cerebellar ataxias in that the muscular strength is usually diminished and the finer movements impossible. Cortical hemianesthesia is not frequent. (*c*) Ptosis and paralysis of the orbicularis palpebrarum are said to result from lesions of the *gyrus angularis* (*A.R.*, Fig. 51) and also inability to read (alexia), optic aphasia and "mind-blindness." Wernicke diagnosticated softening in this lobe by conjugate deviation of the eyes and head. Disease of the posterior inferior part probably causes crossed amblyopia.

III. The Cortex of the Occipital Lobes.—The *visual centre* lies in the cuneus (Fig. 51) and the first occipital lobe. A lesion produces (*a*) bilateral *homonymous hemianopsia* (see AFFECTIONS OF THE OPTIC NERVE). (*b*) *Hemichromatopsia,* an homonymous color-blindness, in which the lesion is superficial and anterior; (*c*) *total cortical blindness,* if the lesion is bilateral or both optic radiations are destroyed. (*d*) "*Mind-blindness*" results from a lesion in the anterior outer surface of the *left* occipital region, especially the second occipital lobe or of the gyrus angularis. The visual memory is lost; things actually seen are misinterpreted and the patient confuses persons and things. (*e*) *Alexia,* inability to read, also called "word-blindness," wherein the patient can read but fails to understand the letters or words he reads. His memory for words is lost. It is due to destruction of the subcortical association fibers in the second occipital lobe and the gyrus angularis; it most commonly follows vascular disease. (*f*) *Optic aphasia* results from foci in the left occipital lobes (and also in the angular and supramarginal convolutions) and consists of misinterpretation of visual impressions, so that the patient miswrites and misnames objects seen, though able to name them when he feels them.

IV. The Cortex of the Temporal Lobes (T_1, T_2, T_3, Fig. 51).—The outer surface of the posterior half of the first (and second) temporal convolution is the *auditory centre;* disease in this cortex produces (*a*) *deafness* in the opposite ear; atrophy of this lobe is encountered in congenital deafness. Deafness is usually transitory, fibers passing to both ears from each centre. If this lobe is destroyed on both sides, total

deafness results. (*b*) *Conjugate deviation of the eyes* is said to be a reflex from auditory impressions. (*c*) Disease of the first temporal lobe produces "*word-deafness*" or "mind-deafness," a loss of the comprehension of speech. In the *uncinate gyrus* (Fig. 50) the special sense of smell probably has its centre. The gyrus fornicatus (Fig. 50) is said to be the centre for *taste* and *pain*.

V. The Frontal Cortex and Aphasia.—Regarding the frontal lobes anterior to their participation in the motor cortex, profound disease may exist without clinical symptoms. They have long been thought to be the residence of (*a*) the *psychical functions*, especially for ethical and abstract thought (Meynert and Flechsig). "Defect in character" followed the passage of a large crowbar, $1\frac{1}{2}$ inches thick and $3\frac{1}{2}$ feet long, through both frontal hemispheres, in Harlow's famous case. (*b*) Flechsig considers the frontal lobe the anterior *association centre*, the middle one being the cortex of the island of Reil and the posterior one the large area posterior to the motor cortex. (*c*) *The motor speech area lies in the lower frontal convolution.* The history of aphasia marks the beginning of cerebral localization; Bouillaud, in 1825, referred aphasia to the frontal lobes; Dax, in 1836, narrowed the localization to the left frontal region and the *motor centre* was finally confined to the posterior part of the *lower left frontal convolution* in 1861 by Broca, whose name is given to this convolution. The sensory element, necessary to all voluntary movements, was unknown until later, when Meynert and Wernicke (1874) discovered the auditory centre in the first temporal convolution; Wernicke localized "*word-deafness*" in the same convolution of the left side and Naunyn referred disturbance of writing, visual aphasia, etc., to the *gyrus angularis* and adjacent parts of the *occipital* lobes.

Motor Aphasia.—The *motor speech* centre in Broca's lobe controls the motor centres next described and retains the impressions or memories necessary to their exercise. The corresponding lobe on the right side is the actual centre in left-handed persons and often assumes the functions of the left speech centres after disease of the latter, especially in children. Articulate speech depends upon the integrity of the lower part of the ascending frontal convolution, in which lie the centres for the mouth, lips, tongue, jaw and larynx. (*a*) These constitute the *cerebral mechanism* of speech, whose disturbance is designated motor aphasia. Motor fibers run from the cortex through the centrum ovale, internal capsule and crus to their nuclei in the pons and medulla, whence the peripheral bulbar nerves to the tongue and larynx take their origin. (*b*) These lower tracts constitute the *bulbar mechanism* of speech, disease in which causes abolition of articulation (anarthria) or more often, imperfect articulation (dysarthria). To speak, the individual must have ideas, conceptions. To illustrate, a cortical lesion in Broca's convolution, as small as a hazelnut, may produce motor aphasia, either absolute or partial; the patient may pronounce correctly some few words, possibly only under emotion or in certain relations, or he may pronounce words wrongly, although he recognizes his mistake. He comprehends what is said to him, for the lesion involves no special sense element. He can read writing and print, especially if educated, because "sight may stimulate images with-

out intervention of the motor centre," but he cannot speak voluntarily nor repeat what is said to him; he cannot read aloud; as a rule he cannot write (agraphia), because impulses cannot pass to the hand centre. He may be able to copy. The cortical is the usual form of motor aphasia.

A *subcortical lesion* in the centrum ovale may sever the fibers between Broca's convolution and the motor cortex for the tongue, lips, etc. The patient cannot speak, but the speech centre is normal, and he can write. This pure motor aphasia is a rare lesion. It involves the commissural fibers of the corpus callosum to the right side and produces permanent aphasia, because compensation by the right hemisphere is precluded; if the lesion is in the internal capsule, aphasia is transient (compensation through the corpus callosum).

Sensory Aphasia.—In speech sensory relations and memories are necessary—*hearing, sight, taste* and, in the blind, *touch.*

Auditory Aphasia.—Hearing is most important and develops in the child long before speech. When a child begins to talk, or when one who has lost speech relearns it, the brain cortex, through an association of centres, learns to connect certain sounds or words with definite objects; through the auditory nerves which run from each side to both *auditory centres* in the *first temporal convolution* of either side, he hears mechanically, as we may hear without understanding a foreign language, and learns to associate the sounds with objects, conditions or ideas, for which an *auditory speech centre* in the *left first temporal convolution* (in right-handed persons) is developed. Remembering a sound he has heard but perhaps has never spoken, the child calls into play Broca's convolution, the cortical centres for the lips, tongue, etc., and speaks. Hearing is necessary to articulate speech, unless, as in deaf-mutes, education replaces, by sight and touch, the associations usually acquired by hearing. Destruction of the auditory speech centre profoundly disturbs speech. The patient then hears by the auditory nerves and centres or reads by the visual apparatus, but does not understand what he hears or reads, *sensory (auditory) aphasia* or *word-deafness;* recovery is more common than in motor aphasia. The subject in speaking transposes words (paraphasia) but does not realize his mistakes, an important distinction from motor aphasia. Often he later becomes speechless. He cannot repeat words, write from dictation, or revive words. Writing is impossible, but copying of words is sometimes maintained.

Subcortical auditory aphasia or pure word-deafness (the rarer subcortical form), results from disease *between* the auditory speech centres and the two centres for hearing. Auditory impressions cannot reach the auditory speech centres of the patient, who cannot understand what is said, cannot repeat or write from dictation, but can read, speak and write, as the auditory speech centre is normal.

"*Amnestic*" *aphasia* may be a simple difficulty in recalling certain words in speaking or writing, or it may be very pronounced (word-deafness). It is also poorly called conduction aphasia (Wernicke) and is due to interruption, probably in the island of Reil, of the fibers between the motor and sensory speech centres.

Visual Aphasia.—*Sight* is very important in sensory speech. The child who has learned and is able to speak words, reads by associating the sounds heard and spoken, with words, letters or symbols seen by him. While in most individuals mental processes are carried on by the memory of words, in rare instances visual memory may be more important. Destruction of the *angular* and *supramarginal* convolutions by causing a loss of the *visual memories* produces *alexia*, word-blindness, an inability to read. In some cases the patient can read, but without understanding. He usually cannot write (agraphia) and cannot copy, but can speak fairly well and understand what is spoken. If the optic centre in the occipital lobes is intact he cannot name objects after seeing them, since the visual memory centre is destroyed, but may recognize a watch by hearing it tick or by feeling it. Lesions just below the visual memory centre, involving the afferent fibers, produce *pure subcortical word-blindness, or alexia*, with hemianopsia and preservation of voluntary speech and writing. Word-deafness and word-blindness often coexist; 41 instances of congenital word-blindness were collected by McCready.

Pure are less common than complicated forms. The anatomical lesions are largely the result of softening, the entire sphere of speech being supplied by the artery of the fossa of Sylvius. Motor aphasia is likely to occur with disease of the motor cortex or cerebral pyramidal tracts, and the sensory form with disease of the optic fibers. Apoplexy, trauma and in children tuberculosis of the brain or meninges are also causative. Lasting aphasia in children is usually due to mental causes, since the right frontal area usually takes up the function of Broca's convolution. Congenital aphasia may follow bilateral meningeal hemorrhage. Aphasia also occurs in idiocy, melancholia, migraine, convulsions and from functional causes in children, as from fever, fright, worms, etc. In adults the outlook is less favorable; reëducation may yield fair results.

VI. Centrum Ovale.—Focal symptoms may be entirely absent, if a lesion occur between the associative, commissural, motor or sensory fibers (corona radiata). A lesion must be (*a*) very near the cortex to produce Jacksonian or general convulsions. A monoplegia, hemianopsia and subcortical aphasia may occur in this location. A lesion in the island of Reil may produce conduction aphasia. Psychic symptoms may result from interruption of the association fibers which run from one part to another of the same hemisphere or commissural fibers running from one hemisphere to the other, as through the corpus callosum. (*b*) Internal capsule symptoms result from lesions low in the centrum ovale.

VII. The Internal Capsule.—It is compacted and consists of nearly all the motor, sensory and special sense fibers between the brain and the lower parts. The capsule is divided into an anterior limb, the knee and a posterior limb.

Anterior Limb.—It contains fibers from the frontal cortex to the optic thalamus and fibers which, in lower horizontal sections, run into the knee and are found in the basal median bundle of the crus, whence they run to the pons nuclei for the eye, head, neck and probably larynx, tongue and mouth. Lesions here are very rare.

Knee.—The knee (see Fig. 52) contains from before backward fibers for opening of the eyes, associated movements of the eyes and head, the tongue and mouth and phonation fibers for speech, probably including those of the larynx.

Posterior Limb.—The posterior limb contains motor fibers in its anterior and middle thirds (*v.* Fig. 52); in its posterior third (Charcot's "sensory crossway"), those for sensation and the special senses. The number of fibers to a muscle depends less on its size than on its fineness of movement; *e. g.*, the fingers receive a large number. If a ruler be placed anterior to the line (*A'*, *A*) in Fig. 51 and slowly moved to the right, preserving its parallelism, the cortical motor areas touched will approximately correspond with their order in the internal capsule. The most common sequence of lesions of the internal capsule is *cerebral hemiplegia* of the opposite side of the body. Though hemiplegia may result from lesions elsewhere, in the vast majority of cases, hemiplegia is the result of disease of the internal capsule. If the latter is destroyed, *total hemi-*

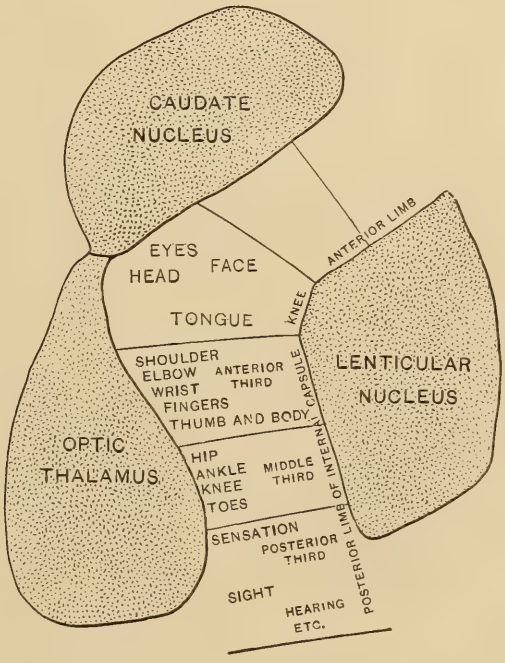


Fig. 52.—Localization in internal capsule.

plegia results, paralysis of the arm, leg and lower part of the face (the upper part of which and the head escape because they have a bilateral supply from both hemispheres). The hypoglossus is but moderately involved and aphasia is usually temporary. There is also associated hemianesthesia, hemianopsia and unilateral disturbance of smell, taste and hearing, by lesion of the fibers ascending from the tegmentum; with hemiplegia are observed contractures, descending degeneration, decrease or absence of the skin reflexes, especially the cremasteric, and increase of the tendon reflexes. Monoplegia is rare, because the tracts are so compact; a *linear* lesion may involve the outer part of the capsule's knee, affecting the face and leaving the arm free. Hemianesthesia (skin

sensation and muscle sense) rarely exists without some paresis of the leg. Some tingling or, in incomplete lesions, some pain may result, for some sensory fibers course with the motor fibers. Hemitremor, -athetosis and -chorea result from lesions of the sensory area, usually associated with disease in or just below the optic thalamus. Disease back of the general sensory tracts may injure the optic radiation (running between the occipital sight centre and the lateral geniculate body) or interrupt the auditory and other special sense fibers. Psychical disturbance, aphasia, alexia, agraphia and involvement of any cranial nerve are not symptomatic of disease of the internal capsule.

VIII. The Corpus Striatum.—This is composed of the caudate and lenticular nuclei. The lenticular nucleus is extensively connected with the superior peduncle of the cerebellum and the tegmentum of the crus. The caudate nucleus is connected with the internal capsule and the crus; these fibers seem to end in the pons and cerebellum. Nothnagel held that hemiplegia is permanent only when the internal capsule is injured, but Landouzy reports a case of contralateral hemiathetosis from isolated disease of the lenticular nucleus.

IX. The Optic Thalamus.—This is surrounded by gray matter, continuous with that surrounding the third ventricle and central canal of the cord and is connected by fibers with all parts of the cerebral cortex, the tegmentum and superior cerebellar peduncle. Nothnagel (1879) first described its topical symptomatology. The following findings are observed, alone or in combination: (*a*) *Contralateral hemianopsia* may develop from a lesion in the posterior part of the thalamus, the pulvinar or a lesion which also invades the lateral geniculate bodies. These areas are connected by the optic radiation with the optic centre. This hemianopsia is distinguished from that caused by a cortical lesion by the hemianopsic pupillary reaction and the absence of the marginal field of vision. (*b*) *Disturbance of the mimetic movements of expression* may develop (Nothnagel, Bechterew). The optic thalamus is the reflex centre of the involuntary movements of expression, as weeping or laughing. A patient with hemiplegia from destruction of the left *internal capsule* cannot voluntarily move the lower right part of the face, but the paralyzed portion of the face moves when the patient laughs, for the centre is in the posterior part of the thalamus. On the other hand, in isolated thalamic disease, the opposite side—indeed both sides of the face—can be voluntarily moved, since the facial nerve runs in the internal capsule, but the patient cannot laugh or weep. (*c*) *Sensory disturbances* may occur, such as hemianesthesia, hemichorea, hemitremor, hemihyperalgesia or hemiataxia (involvement of the internal capsule?). There may be vasomotor changes, atrophy and disturbance in the evacuation of the bladder and rectum (*v.* Thalamic Syndrome, page 794).

X. The Corpora Quadrigemina.—There is variance of opinion regarding the function of these masses of gray matter whose fibers lie over the aqueduct of Sylvius and the tegmentum.

Disease of the *anterior corpora quadrigemina* produces (*a*) *moderate involvement of sight*, of greater localizing significance if there is no optic neuritis. These bodies are connected with the external geniculate bodies

and, by way of the internal capsule, with the optic radiation. (*b*) In acute lesions, narrowing of the *pupil* is suggestive. In old lesions, one or both pupils are wide and without reaction to light or accommodation. (*c*) *The eye muscles* may be partially paralyzed (ptosis and upward movements) but are not pathognomonic, because the lesion is so near the nucleus of the third nerve. Disease of the *posterior corpora quadrigemina* may result in (*a*) *ataxia;* some refer it to cerebellar disease, to connections with the cerebellum through the nucleus ruber or to disease in the fillet of the tegmentum; (*b*) *trochlearis paralysis,* in which the eyes cannot be turned inward or outward; and sometimes abducens paralysis; (*c*) *difficult mastication* from involvement of the descending branch of the quintus; (*d*) contralateral *disturbance of hearing,* since

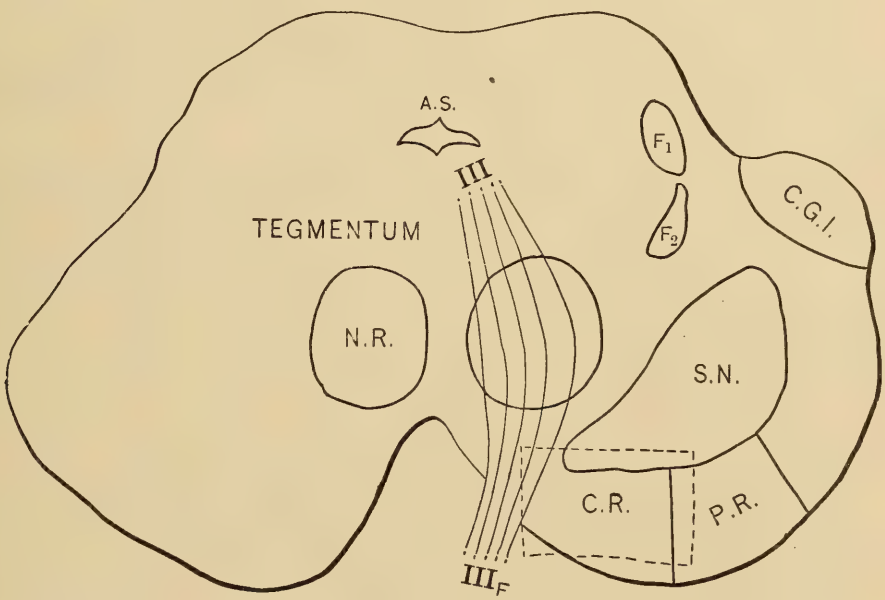


FIG. 53.—Lesion in crus (shown by dotted lines), producing a superior alternating or crossed hemiplegia; the lesion involves the pyramidal tract (*P.R.*), the cranial nerves (tongue and face, *C.R.*) and the oculomotor nerve (*IIIF*) which is paralyzed on the same side as the lesion, while *P.R.* and *C.R.* are paralyzed on the opposite side. *A.S.*, aqueduct of Sylvius; *III*, nucleus of third nerve; *C.G.I.*, corp. geniculatum int.; F_1, upper part and F_2, lower part of fillet; *N.R.*, nucleus ruber; *S.N.*, substantia nigra.

the post. corp. quadrigemina are connected with the fillet of the tegmentum and, by means of the "sensory crossway" of the internal capsule, with the hearing centre.

XI. The Crus (Cerebral Peduncle).—The *motor fibers* to the cranial nerves decussate higher than the pyramidal tracts which decussate in the medulla; the fibers to the third nerve largely cross in the upper crus. *Crossed paralysis* (hemiplegia alternans oculomotoria superior) is common in crus lesions; it consists of a *hemiplegia of the side of the body opposite to the lesion* and a *paralysis of the third nerve on the same* side as the lesion, known as Weber's paralysis, though first described by Gendrin (Fig. 53); the oculomotor paralysis is peripheral, with strabismus, ptosis, inability to look up or down and a wide, reactionless pupil. This crossed or alternating hemiplegia is called superior because inferior

alternating hemiplegia may occur in lesions of the pons. We distinguish whether the hemiplegia and paralysis of the third nerve occurred together (indicating a crus lesion) or whether the third nerve paralysis followed the hemiplegia (indicating a lesion near the internal capsule pressing secondarily on the oculomotorius).

A lesion in the tegmentum may cause (*a*) *sensory disturbance,* as hemianesthesia (skin sensation and muscle sense); such a lesion is in the fillet; hypesthesia (reduced sensation) is more common. (*b*) The *special senses* are sometimes involved, hemianopsia, or poor hearing in the opposite ear. (*c*) *Ataxia,* alone or with other symptoms, differs from the cerebellar type in that it is manifest only on motion. Involvement of the red nucleus produces cerebellar ataxia. (*d*) *Oculomotor paresis* on the side of lesion is characteristic when combined with crossed hemiataxia, hemiplegia or hemianesthesia. Fig. 53 shows how a tegmental focus can rarely miss the diffuse nuclei of the third nerve. If the lesion is in the lower part of the crus, the oculomotor paralysis is usually complete, but if in the tegmentum it is always partial, electing the pupil, the lid or the superior or internal rectus.

XII. The Pons.—A small lesion, as a thin tumor, may give no localizing symptoms. Lesions (commonly tumors, hemorrhage, softening or abscess) usually produce a typical picture: From a *lesion of the upper pons, i. e.,* above the facial nucleus, a hemiplegia results like that produced by lesions of the internal capsule. (See Fig. 54, focus *A.*) The facial paralysis is supranuclear, partial and with no reaction of degeneration. *In a focus in the lower pons* (focus *B,* Fig. 54), where the facial fibers have already crossed, the tongue, arm and leg are hemiplegic on the side opposite to the focus and the facial paralysis is on the side of the lesion, hemiplegia alternans facialis, first described by Millard and Gubler. The facial paralysis is peripheral and nuclear, involves all branches and gives the reaction of degeneration. Other alternating paralyses are also observed, as *hemiplegia with crossed paralysis of the third, fourth, fifth and sixth nerves,* both nuclear and peripheral. Hemiplegia may occur with conjugate paralysis of the lateral eye muscles, as the external rectus (abducens nerve) of one side, and the internal rectus (third nerve) of the other, due to a lesion just in front of the abducens centre, involving its fibers, or sometimes also including its nucleus. In the conjugate paralysis, the antagonist muscles pull the eyes to the other side and the *eyes look away from the side of the lesion* toward the paralyzed side. In this form even the paralyzed internal rectus will functionate and converge when one eye is tested at a time. Involvement of the *motor branch* of the fifth nerve produces difficult mastication. While alternating paralysis is the usual pons lesion, sometimes double hemiplegia occurs, or again, involvement of the third to seventh cranial nerves without hemiplegia. The *auditory nerve* is rarely involved, save in acute or very extensive lesions affecting the lateral lemniscus. Dysarthria and dysphagia, due to disease of the cortical motor paths of the hypoglossus and vagus, are less common in lesions of the pons than are affections of their nuclei in diseases of the medulla oblongata. *Ataxia* may result from involvement of the fillet. *An alternating sensory paralysis* may

occur, as (*a*) hemianesthesia involving the muscle sense and cutaneous sensation of the extremities opposite the lesion, caused by disease of the tegmentum, and (*b*) an anesthesia of the fifth nerve on the side of the lesion. *Irritative* symptoms sometimes develop, as pain, paresthesia, painful convulsions of both arms or both legs, tonic and clonic spasms in the opposite arm and leg, contractures, paroxysms of coughing and trismus. The patient may "look toward the lesion" when the eye muscles are irritated (conjugate deviation). A tendency to fall toward the side on which the lesion is located is indicative of disease of the middle peduncle of the cerebellum. *Aphonia, dysarthria and dysphagia* are symptomatic of *localization in the medulla* (*q. v.*), especially when the

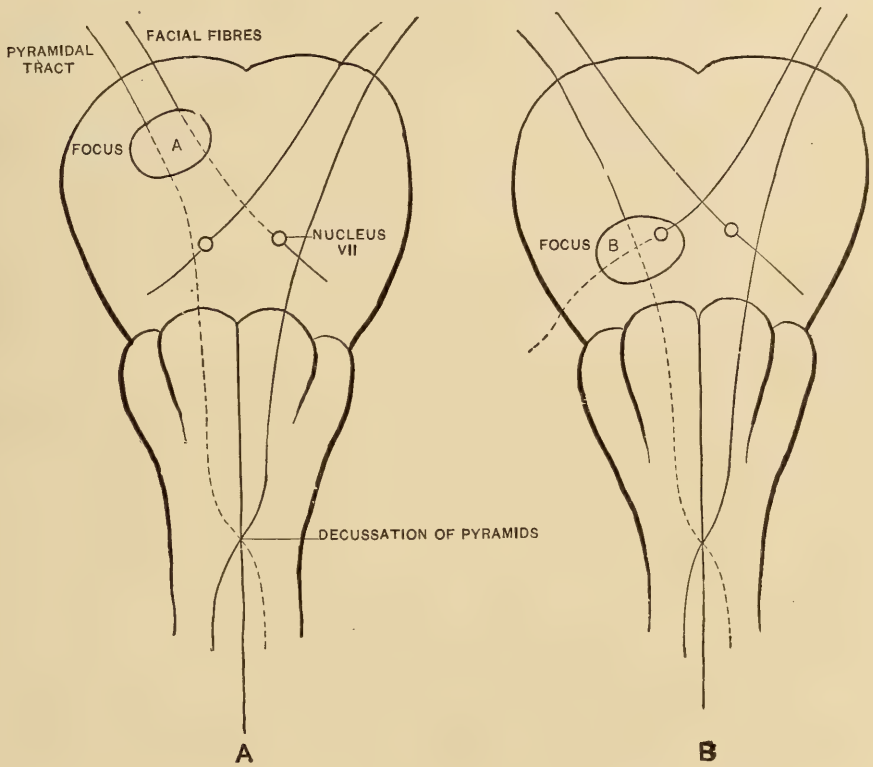


FIG. 54.—*A* illustrates a lesion in the upper pons, producing the usual hemiplegia (face, arm and leg paralyzed on the opposite side). *B* illustrates a lesion in the lower pons, producing alternating paralysis; all branches of the face (peripheral paralysis) and tongue on the side of lesion are paralyzed with paralysis of the contralateral arm and leg.

hypoglossus is paralyzed, shows the reaction of degeneration and produces atrophy of the tongue. Glycosuria, albuminuria, disturbed respiration or heart action (vagus) and vomiting are other bulbar symptoms. Disease may cause paralysis of both arms and legs or rarely of one arm and the opposite leg (hemiplegia cruciata).

XIII. The Cerebellum.—Physiology.—Physiologists admit the possibility of some psychical function. Its *sensory relations* are important, because of its reception of numerous fibers from the spinal cord, including the direct cerebellar tract and the posterior median column of Goll, of which it is an "end organ" (Luciani). It *harmonizes the movements of the body, maintains equilibrium, conserves muscular strength and inhibits*

the contralateral cerebral motor cortex, i. e., it coördinates (*a*) the sensory impressions ascending from the spinal cord with (*b*) those gained by hearing (the auditory nerve having connections with the middle cerebellar lobe) and (*c*) probably with those impressions resulting from the eye movements.

General Pathology.—Disease of a lateral lobe affects the same side of the body, but lesions of the middle (vermiform) lobe influence both sides. Krauss found, in 100 cases of cerebellar disease, tumors in 80 per cent., abscess in 10, cysts in 7 per cent. and softening, hemorrhage and agenesis, sclerosis and gliomatosis in the balance. Acute disease of the cerebellum produces symptoms more frequently than does chronic disease, in which latter the cerebellum or the cerebrum has opportunity for accommodative compensation; rapidly growing tumors, abscess and acute vascular disease often produce the following symptoms:

Topical Symptoms.—1. *Cerebellar ataxia* is almost always present and occurs when the patient stands; the body sways (static ataxia), as though the subject were trying to balance himself. Ataxia also occurs when the patient moves (dynamic ataxia) and concerns especially the trunk and legs. It is due to disease of the middle lobe or vermis and the consequent withdrawal from the cerebrum of the cerebellar coördination. The gait is that of an inebriate (*démarche d'ivresse*); the individual stumbles a zig-zag course sometimes toward the side of the lesion. The arm and hand movements are usually normal. In ataxia resulting from other cerebral localization (cortex, internal capsule and tegmentum) the incoördination affects the finer movements, is associated with disturbed muscle and stereognostic sense and very often with paresis. Bouillaud first described cerebellar ataxia and differentiated it from the tabetic type; cerebellar ataxia disappears when the patient is lying down and closing the eyes does not increase it (indeed it may often disappear). The affected limbs can be held steadily.

2. *Vertigo* is very frequent and usually occurs rather when the patient sits than when he lies down; objects turn around him or he feels himself being turned. The vertigo is constant or paroxysmal and is due to lesion of the vestibular nerve which connects the semilunar canals with the cerebellum by the corpus restiforme.

Secondary Symptoms.—The following are less significant than ataxia and vertigo: (*a*) *Choked disk* is rarely absent in cerebellar tumors and *amaurosis* results from hydrocephalus internus with pressure on the chiasm. (*b*) *"Forced" postures* and movements are due to disease of the middle peduncle (*crus cerebelli ad pontem*). Constrained postures of the head and trunk are observed; one eye looks upward and inward, while the other is directed downward and outward. Forced movements consist of turning to one side, movement in a circle and a tendency to fall toward the side of lesion and sometimes backward. (*c*) *Muscular weakness* is frequent in animals and occasional in man, as hemiparesis on the side of lesion. The patient may not be able to rotate the trunk, straighten the body after bending or get up after falling. (*d*) The *patellar reflexes* are usually normal or increased. (*e*) *Vomiting* and *pain* in the head or neck are of some localizing value. (*f*) Occasionally the following develop:

nystagmus, rhythmical contractions of the head, convulsions, paralysis of the cerebral nerves from pressure on the midbrain, disturbed hearing, intention tremor, muscular rigidity, irritability and weak memory.

CIRCULATORY DISEASES OF THE BRAIN.

I. Anemia of the Brain.—This term has been much abused.

Etiology.—(*a*) General systemic anemia may result from cachexia and hemorrhages of medical, surgical or obstetrical nature. (*b*) *Decreased blood supply*, due to cardiac insufficiency, valvular stenosis, vessel stenosis, evacuation of an enormously distended bladder and tapping of pleurisy or ascites leading to paresis of the thoracic or abdominal vessels may be causal. (*c*) *Capillary compression* by hydrocephalus or tumors may produce anemia. (*d*) *Local causes* in the vessels will be considered under softening. Convulsions by pressure on one carotid are indicative of cerebral atheroma.

Pathology.—The brain is pale, firm and dry and *puncta vasculosa* fewer. The fluid in the ventricles is often increased.

Symptoms.—Local anemia of the brain leads to imperfect function, then loss of function and finally to necrosis, as in embolism or thrombosis (*q. v.*). In *sudden general anemia* after hemorrhage or fainting, the subject becomes drowsy, syncope occurs and subsequent amaurosis, or ringing in the ears is frequent. The pupils at first contract from irritation of the third nerve and later dilate from its paralysis, a sign of danger. The skin is pale and clammy, the muscles lax and nausea frequent. Epileptiform convulsions often occur when much blood is lost. Anemia of the medulla causes the slow, sighing respiration and yawning. Death after syncope was called nervous apoplexy by the old writers. *In gradually developing anemia* symptoms develop, as described under Pernicious Anemia. Palpitation and thoracic oppression are common symptoms. The pulse varies in rate and rhythm.

Marshall Hall described *hydrencephaloid*, a variety of cerebral anemia, seen in children suffering with acute diarrhea, and characterized by contracted and later dilated pupils, depressed fontanelles, sunken face, pallor, early irritation followed by stupor or even coma. It may resemble meningitis from the strabismus.

Treatment.—Treatment, diagnosis and prognosis are chiefly those of the cause. The *anemia* is treated by putting the patient in the recumbent posture and employing opiates, stimulation, coffee by rectum and enemata or subcutaneous infusions of salt solution. Firm bandaging of the extremities, sometimes called autotransfusion, is valuable. Nitroglycerin is valuable in cases due to vasomotor spasm.

II. Hyperemia of the Brain.—Andral (1836) described eight varieties, and when Trousseau, in 1861, referred some varieties to epilepsy and Ménière's disease, he drew down upon him the wrath of the entire Academy. The hyperemia in nephritis is usually uremic and the arterial fluxion once attributed to erysipelas is toxemic. When the vessels dilate from paralysis of the sympathetic system the rapidity of the capillary flow decreases, whence there is no hyperemia, but a lessened capillary

flow, *adiämorrhysis*. When the vessels contract from sympathetic irritation, the brain is not anemic, but the capillary flow is accelerated, *hyperdiämorrhysis*.

Etiology.—*Active congestion* results from cardiac overaction; overfilling of the brain vessels, as in stenosis of the isthmus of the aorta; contraction of the arterioles in other parts; dilatation of the brain vessels by alcohol, nitroglycerin, coffee, thyroidism, excessive brain work, neurotic states and insolation. In plethoric men with florid faces, thick necks and short thoraces, active congestion may cause fugitive brain symptoms.

Passive congestion results from general venous stasis, as in valvular heart lesions, or local venous stasis, as sinus thrombosis.

Pathology.—The brain may appear anemic after death by strangulation and may become congested after death because of its posture alone. The sinuses and veins are turgid and the color of the brain is darker. Microscopic examination shows distention of the capillaries or rupture into the perivascular lymphatic sheaths or brain.

Symptoms.—In *active congestion*, chronic cause fewer symptoms than acute factors. Whether toxemic symptoms of the acute infections are due to hyperemia is an open question. Paroxysmal throbbing of the vessels, flushing, headache, delirium, sometimes nausea and fever are relieved by nosebleed and venesection. Irritability, spots before the eyes and ringing in the ears are said to occur. In progressive paralysis the transient pyrexia, heat in the head or coma is referred to congestion of the brain.

Passive congestion, if gradual in onset, may be well tolerated or may produce stupor, dulness or delirium. If intermittent, as from coughing, there are a fulness in the head, headache and sometimes convulsions.

Diagnosis.—Focal symptoms, as hemiplegia, indicate organic disease. Flushing, heat and pressure in the head, are most often neurasthenic or hypochondriacal. Marie and Leube never made the diagnosis of brain hyperemia.

Treatment.—Active congestion is treated by elevation of the head, *venesection* in plethoric adults or in children by *leeches* over the mastoid bone, the veins of which connect with those of the brain; by *free purgation*, by 2 drops of croton oil, followed by concentrated salines, to draw the blood to the large abdominal vessels; by *hot baths*, to draw the blood to the periphery; bromides and *vasoconstrictors*, especially digitalis. Alcohol and opiates should be avoided; ice may be applied to the head and carotids; a *fluid diet* should be given.

III. Edema of the Brain.—Etiology.—Edema of the brain results from the same causes as general edema: *circulatory disturbances*, as venous congestion in cardiac disease and the *marantic group*, including cachexia and nephritis. Collateral and inflammatory edema may develop around brain tumor, abscess or hemorrhage. Brain edema may develop in the death agony or when the brain shrinks, *hydrops ex vacuo*.

Pathology.—The brain is pale, moist and glistening on section, the so-called "wet-brain." The affection may be general or local; the ventricular and subarachnoid fluid is increased. The volume of the brain may be increased in the generalized form.

Symptoms.—A diagnosis cannot be made between edema and decreased capillary velocity. When the edema is localized, transient focal symptoms may develop, as unilateral convulsions or hemiplegia, or even crossed paralysis, observed personally in nephritis. Traube held that brain edema causes uremia, a view opposed by Cohnheim. Variability in the symptomatology of organic brain disease may be due to associated edema.

IV. Cerebral Hemorrhage.—This most common and important of all brain lesions involves an enormous part of brain pathology. Apoplexy strictly refers to a "stroke," which is symptomatic of various other lesions, yet it is used to designate spontaneous hemorrhage into the brain (Rochoux).

Etiology.—(*a*) *Miliary aneurysms* are the direct cause of cerebral hemorrhage. Their pathogenesis relates to (*b*) *arteriosclerosis* (alcoholism, plumbism, hard work, contracted kidney, overeating), but some maintain that atheroma does not *per se* predispose to hemorrhage. Eighty per cent. of hemorrhages occur *after the fortieth year* of life, and *in men.* It may occur in early life from glioma, whooping-cough or trauma. *Heredity* relates rather to renal and arterial lesions than to an actual heredity. Hemorrhage is most common among civilized races and in temperate zones, because of the strenuous habits of life. The importance of the *apoplectic habitus* (plethora, ruddy face, short, thick neck, wide shoulders and chest, round, small body and large muscles) has been much emphasized; Gowers finds most cases in thin individuals. *Contracted kidney,* found in 33 per cent. of cerebral hemorrhage, is a prominent cause of atheroma and miliary aneurysm. (*c*) *Increased arterial tension* is less important than its relation to arteriosclerosis and miliary aneurysms, which occur where the blood-pressure is greatest, as where the vessels branch. Rupture is seen in cardiac hypertrophy from stenosis at the isthmus of the aorta. Syphilis more frequently produces softening than hemorrhage; it is a causal factor of aneurysms of the large cerebral vessels. Violent exertion, alcoholic excesses, excitement, coitus, overeating, vomiting, coughing or convulsions may rupture an already-diseased vessel or a miliary aneurysm; a healthy vessel almost never bursts. Embolism from heart disease may produce the "embolic aneurysm." Sometimes trauma is a factor, usually, however, with the causes above mentioned. (*d*) *Permeability of the bloodvessels* causes a few cases, by hemorrhage by diapedesis, as in nephritis, purpura, anemias and infections.

Pathology.—Hemorrhage most often occurs in the branches of the arteria fossæ Sylvii; the *lenticulostriate artery* of Duret, Charcot's *artery of hemorrhage,* is the seat of hemorrhage in 75 per cent. of cases. This small vessel is likely to rupture because it is so near to the large internal carotid and its course is very direct; it has no special external support; and there is no collateral circulation, hence no relief to pressure, rendering the vessel tortuous. Rupture occurs very often in the long direction of the fibers of the internal capsule, where also the veins stagnate very easily. *Miliary aneurysms* were described by Brunner (1700) and Morgagni (1761), but Charcot and Bouchard (1866) emphasized their relation to brain hemorrhage. They are fusiform, more rarely sacciform,

enlargements of the arterioles, usually multiple, like a bunch of grapes, and measuring $\frac{1}{100}$ to $\frac{1}{25}$ of an inch; they are false aneurysms; they may be seen with the naked eye, by careful washing or macerating in water, or on a slide under a low power. They are found where hemorrhages are most common, *i. e.*, the central ganglia and where the blood-pressure is highest. Charcot and Bouchard described periarteritis but Eichler and Zeigler held that intimal changes were primary; Roth finds atrophy of the muscular coat the accepted theory. Large aneurysms will be considered separately.

Localization.—Hemorrhage is most common in the caudate and lenticular nuclei, internal capsule and adjacent centrum ovale, extending to, but rarely beginning in, the optic thalamus. Hemorrhage in the white matter has usually an elongated form. It is much less common in the cortex, pons, crus, cerebellum or medulla. It occurs mostly in the substance of the brain, but may rupture on the surface or into the ventricles, causing sudden death, the usual autopsy finding. Hemorrhage is usually single. The multiple form is observed in anemia. When the skull is removed, the dura is tense, the convolutions flattened and pale, the falx pushed toward the other side and the fissures indistinct. Fluctuation may be felt in rare cases; on section the focus is seen, usually the size of a hazel-nut or walnut; it sometimes involves the entire hemisphere or may be no larger than a pea. The brain around the focus is softened from pressure, edematous and in the focus, torn and pulpy. The dark clot becomes chocolate-colored, then yellowish-red and finally yellowish-white. The wall of the focus is irregular in the gray nuclei and regular in the white matter and threads of bloodvessels run through it. Leukocytes laden with fat drops, hematoidin and disorganized tissue are found. As the blood absorbs, the cavity becomes encapsulated by inflammation and contains a serous, milky fluid (the *apoplectic cyst* of Virchow). An *apoplectic scar* is much less common. *Traumatic hemorrhage* may occur infrequently near the point of injury or *contre coup* on the opposite side; the late traumatic hemorrhage of Bollinger takes place after a week or more, near the fourth ventricle and aqueduct of Sylvius, and probably in areas of traumatic softening. Brain hemorrhage may sever the internal capsule.

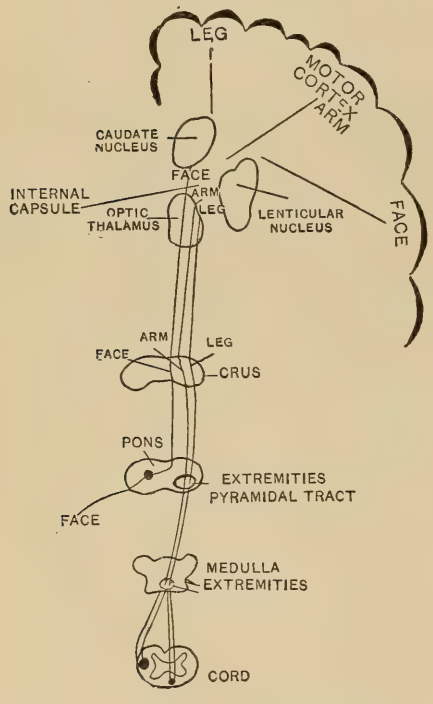


Fig. 55.—Showing course of individual fibers of motor tracts.

Secondary degeneration occurs in the *pyramidal tracts*, chiefly from a lesion of the internal capsule; sound and diseased fibers intermingle; it descends through the crus, pons, medulla and cord (the direct anterior pyramidal tract and part of the crossed lateral tract). These areas

become progressively smaller and cease just above the conus medullaris. In children a cerebral lesion may cause degeneration in the anterior horns of the cord and their fibers. An ascending degeneration occurs only when the large pyramidal tracts of the cortex are diseased, except in the young, in whom degeneration of all neurones is prone to follow the destruction of one neurone. (See figures under LOCALIZATION IN THE

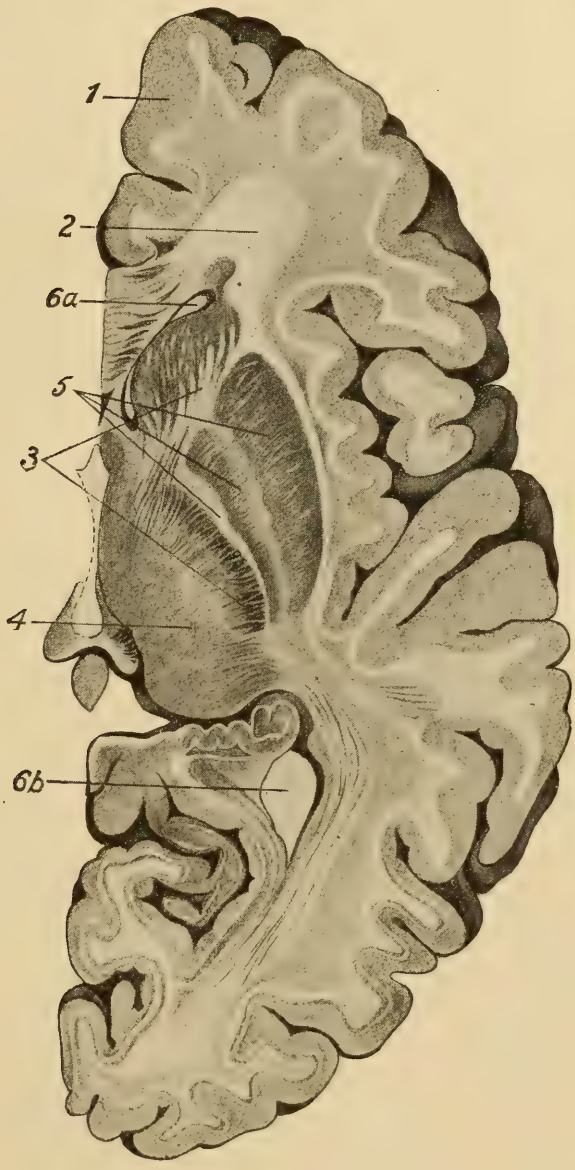


FIG. 56.—Horizontal section through the right hemisphere: 1, cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6*a*, anterior horn; and 6*b*, posterior horn of the lateral ventricle. (After Déjérine.)

SPINAL CORD.) Other degenerations: (*a*) The *optic radiation* degenerates when a lesion occurs in the cortex of the cuneus and also degenerative changes in the external geniculate body and pulvinar. After years it may reach the optic nerve. (*b*) Hemorrhage in the second and third frontal lobes leads to degeneration in the *anterior limb of the capsule,* median basal fibers of the crus and the fibers connecting the optic thala-

mus with the cortex. (*c*) Lesions in the temporal lobes cause degeneration in the *lateral part of the crus.* (*d*) Degeneration in the median lemniscus follows diseases of the cortex, the subthalamic region and the upper part of the pons; it crosses to the opposite nuclei of the medulla and after years destroys the ganglion cells in the posterior columns of the cord.

Symptoms.—Miliary aneurysms produce no symptoms until they rupture and premonitory symptoms are due to atheroma and are not pathognomonic. They may be general (*v. i.*, softening); cardiorenal, as hypertrophied heart or hypertension; or rarely focal, as alexia, facial paresis, preparalytic chorea, etc. Prodromal symptoms are seldom due to an initial small hemorrhage or to hemorrhage developing very slowly.

1. The Insult and its Primary Symptoms.—The "stroke" is usually *sudden;* its most common general symptom is *coma,* which may be absent in small lesions and its most common focal symptom is *hemiplegia.* It may develop during effort or excitement, or more often without cause, as during sleep. In ingravescent apoplexy the onset is gradual, with delirium, convulsions and finally coma; it is due to hemorrhage external to the lenticular nucleus, which later involves the internal capsule. Death may, in very rare instances, occur in five minutes from rupture into the ventricles or direct pressure on the medulla (fulminating apoplexy).

The insult is due chiefly to *anemia of the cortex* and to some extent to direct brain trauma, pressure on other vessels, serous infiltration and alterations in the cerebrospinal fluid in which lumbar puncture sometimes shows blood. Its *cardinal typical symptoms* are *coma,* which is greater the nearer the lesion is to the optic thalamus and third ventricle; *flushed face,* sometimes pallor or cyanosis; a tense, regular and *slow pulse;* long, deep, *stertorous breathing* (due in part to palate paralysis); the *cheeks puff out,* but are drawn in on inspiration, as a result of paralysis of the buccinator muscle; inspiration is sometimes irregular; yawning is frequent; the *temperature* usually suffers an *initial fall,* but rises when reaction sets in, though lesions in the pons, medulla or central ganglia may cause an initial rise. The *pupils* are usually wide, from paralysis of the third nuclei or sympathetic stimulation; they are sometimes unequal and in deep coma *reactionless.* They may be narrow from irritation of the oculomotor nucleus in lesions of the pons, or when the ventricles are involved. The head and eyes look toward the side of the brain lesion ("conjugate deviation"), *a flaccid hemiplegia* occurs, in which the limbs fall limp when they are lifted; the *reflexes* are *gone* and there are *involuntary evacuations* of urine and feces. An increase of the urine with a low specific gravity (1.004) and transitory *albuminuria* and *glycosuria* are due to a large lesion exerting indirect pressure on the medulla or to a lesion in the medulla itself.

2. The permanent damage can be estimated only when the reaction is passed. The *direct focal symptoms* are those of the hemorrhage itself and the subsequent cyst or scar; they are permanent if essential structures are involved, as the internal capsule. *Indirect focal symptoms* are those of other structures disturbed by pressure, edema or inflammatory

reaction (the pulse and respiratory symptoms, conjugate deviation, albuminuria and temperature). Time is required to separate these two classes of symptoms.

3. Details of Important Symptoms.—(i) *Convulsions* are not common (pons, ventricles, cortex or corpus striatum lesions).

(ii) *Conjugate deviation* is a phenomenon in which the *lateral muscles of the head and eyes are paralyzed;* their fellows on the sound side contract, so that "the patient *looks at his lesion*" (Prevost); in left hemiplegia the eyes and head look toward the right side of the brain. The centres paralyzed are the gyrus angularis and gyrus supramarginalis. It disappears in a few days because the sound side of the brain compensates or because it is a distant pressure symptom. Conjugate deviation may also be due to *irritation* or Jacksonian epilepsy when the patient "looks away from his lesion" (Landouzy), *i. e.*, toward the side affected with convulsions. These differ from the deviation in localization in the pons (*q. v.*).

(iii) The *hemiplegia* is due (75 per cent.) to a lesion of the internal capsule (*v.* p. 757). During deep coma it is often difficult to determine which side is paralyzed. The affected side is usually flaccid and the mouth shows greater puffing on the hemiplegic half. If the coma is not profound the sound arm and leg may move. Sometimes, especially with hemorrhage into the lateral ventricle, the paralyzed side exhibits early rigidity. The temperature is at first lower on the hemiplegic side from paralysis of a cortical centre. Later, during reaction, it is higher on the side of paralysis. After consciousness returns the contralateral hemiplegia is more clearly seen. The lower two-thirds of the face (nose, mouth, cheek) are paralyzed; the nasolabial fold is obliterated, the lips cannot be puckered as though to whistle, the mouth is oblique or a little triangular when the teeth are shown. The upper third (eye and forehead) is unaffected because it is innervated from both hemispheres or has two distinct centres and paths; in peripheral facial paralysis all branches are involved, with atrophy and the reaction of degeneration. If the optic thalamus (*q. v.*) is intact, the paralysis is not apparent on laughing. The *tongue,* when protruded, deviates slightly toward the diseased side because the sound genioglossus muscle pulls the tongue forward and over toward the hemiplegic side; the base of the tongue is higher on the paralyzed side which may lead to some difficulty in the external speech mechanism (dysarthria), while internal speech is not often disturbed unless there is psychical alteration (aphasia). The *palate* may hang lower on the paralyzed side, but this is of little importance because it varies physiologically. Deglutition, mastication and phonation are but little or only transitorily involved; marked difficulty in mastication is usually associated with total hemianesthesia. In the *trunk* paralysis of the trapezius and levator anguli scapulæ causes sagging of the shoulder; certain cerebral fibers of the spinal accessory may be involved. Hughlings-Jackson finds respiratory movement greater on the paralyzed side in automatic breathing and greater on the sound side in forced breathing. Early, the paralysis of the *arm and leg* is often absolute, sometimes partial (hemiparesis). In the *typical*

case the arm is most involved, though all parts on one side usually are somewhat paralyzed. The arm abductors, hand flexors and finger extensors, the extensors of the leg, dorsal foot flexors and knee flexors are most implicated. The *paralysis is flaccid.* In rare linear lesions the arm, leg or face remains free (see page 757); monoplegia is much less common than in cortical and subcortical disease. The hemiplegia is almost invariably contralateral, *i. e.*, opposite to the side of the lesion; Lederhose collected 45 cases of collateral hemiplegia due to more than one lesion, to a focus pressing on the other side of the brain, or to mistaking the early rigidity of the paralyzed side for the normal side.

(iv) *Sensory disturbance* (*v.* page 757) is less important, less frequent, less complete and less stable than hemiplegia. Some hemianesthesia is frequently found in early cases after careful examination, as disturbed tactile sensation, stereognostic sense and sometimes muscle sense, pains and hyperesthesia or paresthesia. A large lesion only can cause a complete hemianesthesia. It affects the skin and mucosa of the nose, mouth, palate and eyes, but not the cornea. Cortical hemianesthesia is usually partial and most often associated with monoplegia; lesions just above the capsule, may produce hemianesthesia, involving the fibers from the thalamus to the cortex. It also occurs in lesions of the crus, pons, medulla and cord (*q. v.*).

(v) *Special senses.* Hemianopsia, due to pressure on the optic thalamus, usually occurs with conjugate deviation, is temporary and is often overlooked.

(vi) The *reflexes* of the skin, cornea, pupils, mucosæ and tendons are suspended during complete coma, in which the central nervous system is almost wholly paretic. After the stroke they return slowly, first on the sound side. On the *hemiplegic side* they return more slowly; the periosteal, tendon and patellar reflexes often become exaggerated from interrupted inhibition; the skin reflexes return slowly, especially the epigastric, cremasteric, scrotal and mammillary, a point of early diagnostic value.

Babinski's toe sign usually indicates organic pyramidal disease and may be found very early; when a sharp object is slowly drawn over the outer part of the sound sole there is plantar flexion of the large and sometimes of the other toes; when drawn over the diseased sole there is *great dorsal flexion* (extension) of the great toe.

4. Chronic or Secondary Symptoms.—The indirect symptoms, inflammatory reaction and collateral edema disappear.

(i) If the *hemiplegia* is indirect, from a lesion without the capsule, marked improvement or rarely, recovery ensues. Complete lasting hemiplegia follows total division of the pyramidal tracts, due to hemorrhage from the anterior choroid artery (Kolisko). The face often recovers rapidly; *Broadbent* held that the *intercostals*, *masseters* and trunk muscles used in pairs, innervated from both hemispheres and having little differentiation in function, escape permanent paralysis. The functions of the *arm* and *hand* are especially complicated, are more often used alone, are innervated mostly from one hemisphere and therefore suffer the greatest permanent damage. The *legs* are much used together and often show

improvement. The bilateral, spinal and bulbar nuclei enable "paired muscles" to be innervated from the sound hemisphere, or possibly uncrossed fibers in the anterior (and lateral) columns explain thephenomenon. Compensation is offset by the fact that the sound side is reduced in strength in 50 per cent. of cases. In the *arm* the residual paralysis affects functionally associated muscle groups rather than individual muscles, as the muscles supinating the forearm, those abducting and lifting the arm and shoulder, those moving the elbow, opening the hand and apposing the thumb. The shoulder improves a little, the elbows considerably, the hands very little. In the leg the flexors, dorsal flexors of the foot which shorten the leg in walking, abductors and internal rotators of the hip are most involved. The hemiplegic gait is awkward, the foot is swung out in a half-circle and scrapes the ground. The power of the paralyzed leg is greater when it is used with the sound leg, as in walking, than when it is used by itself.

(ii) The *contracture,* the important posthemiplegic manifestation, fixes a joint in an abnormal posture. Early contractures are often regarded as myotatic irritability. The typical late contracture develops in two to four weeks; the shoulder is lifted, the arm adducted to the chest, the elbow rigid, the forearm pronated, the wrist flexed and the fingers flexed even violently into the palm. When the wrist is passively flexed the fingers can sometimes be extended. Contractures involve both extensors and flexors, but more often the latter, and occur in the least paralyzed muscles. In the lower extremity, the contracture is not marked, in the thigh there is often only a trace, the knee is extended, plantar flexion of the foot occurs (*pes equinovarus*), and the great toe is dorsally flexed. Locomotion is difficult, perhaps painful, for there is bending of the trunk to the sound side, lifting of the pelvis because of the abduction paralysis and swinging of the paralyzed foot over the sound foot. The sound leg also suffers slight contracture. Contracture may occur in the face, platysma and sternomastoid, but rarely in the trunk. In rare cases, largely in children, the paralyzed side remains lax (*hémiplégie flasque* of Bouchard).

(iii) *Associated movements* are explained as movements reflected to the associates of the paralyzed muscles or as due to bilateral representation in the hemispheres; an effort to grasp with the sound hand produces a similar movement in the one paralyzed; movement of the diseased side occurs after passive movement of the sound one, etc.

(iv) *Hemitremor* is uncommon. The muscles are stimulated, but not enough vibrations occur for actual involuntary movement.

(v) *Atrophy* may occur in spastic, though more in the flaccid, cerebral forms. Charcot referred it to degeneration of the anterior horns, while Quincke and Senator assume an injury to some trophic paths. It has no relation to the degree of paralysis, is often associated with disturbed sensation and muscle sense, affects principally the arm and occurs in large lesions which are cortical in the region of the third branch of the artery of Sylvius or central in the lenticulothalamic distribution. The skin may atrophy.

(vi) *Posthemiplegic chorea* results in 80 per cent. of cases from lesions in the internal capsule or near it, in the optic thalamus, lenticular or

caudate nucleus, from which irritation is reflected to the brain cortex. It develops months or years after *small lesions*, when the hemiplegia and rigidity are largely gone. It occurs especially in the arm, causes movements similar to but coarser than those of chorea and is often associated with pain and sensory disturbance (see page 757). It sometimes is preparalytic. In *posthemiplegic athetosis* the fingers and toes are incessantly moved; athetosis means "without fixed position," a "mobile spasm" consisting of spasmodic, slow, coördinated movements in paralyzed members; it is frequent in partial paralysis acquired early in life, appears long after the paralysis has regressed and has the same localization as hemichorea.

(vii) *In trophic and vasomotor disturbance* (*v. s.*) the paralyzed members are usually cool, moist and sometimes edematous. The skin is glossy, even livid and fissured. In some cases paralysis of the cervical sympathetic causes redness, narrowing of the eye fissure, drooping of the upper lid, retraction of the eye-ball, lachrymation and narrow pupils. Sometimes there is overgrowth of the hair. Bed-sores were considered trophic by Charcot, but can usually be avoided by care regarding pressure or heat and by cleanliness. Swelling of the joints and hypertrophic neuritis on the hemiplegic side are rare.

(viii) *Psychic disturbance*, referable not to brain hemorrhage but to arteriosclerosis, is evidenced by self-concentration, weak memory and irritability and more when the left half of the brain is diseased. Laughing and weeping are referable to disease of the thalamus (*q. v.*) or median fibers of the pons.

Diagnosis of Brain Hemorrhage.—Two cardinal features, *coma* and *hemiplegia*, together with the *typical insult*, facilitate diagnosis.

1. Coma.—In *syncope* the pulse is fast, weak and often irregular; in "cardiac apoplexy" (an insult from heart rupture, myocarditis, etc.) differentiation from rapidly fatal hemorrhage may be impossible. It is dangerous to make a diagnosis of *alcoholic coma;* an intoxicated individual may suffer basal fracture or actual apoplexy; an opinion should be withheld until such complications can be excluded, though the alcoholic coma is usually incomplete, the vomitus and breath smell of alcohol and violent struggling is frequent. *Opium coma* is characterized by slow onset, slow respiration, slow, hard pulse, flushed skin, contracted pupils, opium in the stomach washings and later Cheyne-Stokes's respiration, rapid pulse, clammy, cyanotic skin, and dilated pupils; in all intoxications the focal symptoms and reflexes of apoplexy are lacking. Hemorrhage in the pons produces narrow and often unequal pupils. While diagnosis of apoplexy during the coma is precarious, *uremic coma* rarely occurs without premonitory symptoms, as *headache*, drowsiness, delirium, *restlessness, vomiting*, myosis, *retinitis* or convulsions; the coma is often less profound. The cardiorenal findings of nephritis may also occur in apoplexy, of which nephritis is a frequent cause (33 per cent.) and the author repeatedly observed hemiplegia and crossed paralyses in uremia, sometimes with Babinski's sign. In deep coma rigidity may be observed in both uremia and apoplexy, but in uremia it is often variable. In *diabetic coma* the fruity, acetone breath, acetonuria, unrest, Kussmaul's

breathing, granular casts, history of glycosuria, and absence of focal symptoms are characteristic. *Symptomatic apoplexy*, transient coma and hemiplegia sometimes occur in brain edema, congestion, progressive paralysis, multiple sclerosis, epilepsy and tabes.

2. Hemiplegia.—This is a prominent finding in embolism and thrombosis (*q. v.*); Kolisko states that most cases diagnosticated embolism prove to be hemorrhage and *vice versa*. When hemiplegia occurs in meningitis with coma, there are headache, general convulsions, rigidity, hyperesthesia, optic neuritis, fever, etc. In extradural hematoma and pachymeningitis the Jacksonian epilepsy followed later by cortical paralysis is distinctive. Hemiplegia may also occur in hysteria (*q. v.*), syphilis, brain tumor or abscess, trauma, chorea, paralysis agitans, alcoholism, plumbism, uremia, etc.

3. Other Locations of Hemorrhage.—(*a*) Hemorrhage may occur in diseases of the meninges (*q. v.*). (*b*) It occurs rarely in the *cortex;* Jacksonian epilepsy and rigidity may be severe in subcortical lesions. (*c*) *Ventricular hemorrhage* is secondary to hemorrhage in the corpus striatum or thalamus and usually reaches all the ventricles; in rare cases it is primary from aneurysms or ruptured veins in the choroid plexus. One-third of all cases are in persons under forty years of age. It occurs usually with violent symptoms, convulsions (33 per cent.), early and sometimes bilateral rigidity. The conjugate deviation changes to the other side, the pulse is slowed, the temperature falls, the reflexes are suspended. Cheyne-Stokes's breathing, glycosuria and polyuria develop and death usually results within a day. (*d*) Hemorrhage in the *crus* (*v.* page 759) is usually descending, from bleeding near the internal capsule. Crossed paralysis may be observed. (*e*) Apoplexy in the *pons* (*q. v.*) occurs in 10 per cent. of cases. Paralysis and convulsions may occur in the arms or legs only; the trunk musculature is involved more frequently than in higher hemorrhages. The eyes look away from the side of the lesion. The pupils may be large from paralysis or small from stimulation of the third nerve, as in opium poisoning. Respiration is affected early, vomiting is frequent, there is often early high fever and death is usual within twenty-four hours (it has occurred in seven minutes). (*f*) Hemorrhage in the *medulla* will be considered later. It is rarely diagnosticated. Early or instantaneous death is frequent. (*g*) *Cerebellar apoplexy* occurs in 7 per cent. of cases. Vomiting is very frequent. The insult is sometimes less marked and the occasional hemiplegia is due to indirect pressure or secondary ventricular hemorrhage. Cerebellar symptoms may result.

Prognosis.—The prognosis concerns the immediate danger and ultimate outcome. (*a*) The *immediate prognosis* is always serious; 66 per cent. survive the first stroke, 33 per cent. the second and very few the third. When death occurs it is usual in one-half to two days, but the outcome is uncertain for a week, because light cases, even rare forms without coma, may rupture into the ventricle with rapidly fatal result. Coma lasting over thirty hours, early low fall of temperature or its early rapid rise is unfavorable, indicating a large focus. Conjugate deviation, relaxed sphincters, impaired breathing, bilateral or ventricular symptoms,

glycosuria, albuminuria, decubitus and convulsions are unfavorable. The mode of death is as follows: the breathing becomes more rapid, irregular and rattling from accumulated secretion and of the ominous Cheyne-Stokes's type; the slow, regular pulse becomes rapid, small, irregular, often with a rise of temperature and finally ceases; death occurs in coma.

(*b*) *Ultimate Outcome.*—The symptoms of shock disappear and the patient becomes partly conscious or delirious. *Reaction* begins usually within forty-eight hours after the onset; the pulse becomes faster, the temperature rises, pain develops in the head and limbs. Life hangs in the balance and the patient may die from hypostatic pneumonia, pulmonary edema, decubitus or a second hemorrhage. Reaction is over in one to six weeks. If the patient survives there is *improvement,* depending on the extent and localization of the injury; if movement appears in three weeks the outlook is good; if it does not return in three or four months it is not likely to develop later. Disappearance of indirect pressure leads to bettering (i) of damage to the sensory nerves and those of special sense, (ii) of motility, first of the trunk and face, then of the leg, and less of the arm. Absolute recovery is improbable. Hemichorea and hemiathetosis are incurable. Broken neurones never unite and secondary degeneration results; exaggerated reflexes, developing a few weeks after the insult are forerunners of contractures and preclude full regression. The outlook is fairly good in children when the hemorrhage is not due to blood affections, because some compensation is likely to occur. *Recurrence* is possible in decrepid and aged subjects, although less common than usually believed; the average expectancy is five years.

Treatment.—1. Prophylaxis concerns the causative factors.

2. The Insult.—(*a*) Absolute *quiet* is indicated; movement, transportation or extended examination is avoided. (*b*) The head is slightly elevated, but flexion of the neck, tight clothes or any hindrance to the return venous flow avoided. Bowles thinks that stertorous breathing is due to the position of the paralyzed palate; turning the patient on his side relieves it more effectively than does venesection. (*c*) *Venesection* is indicated only in robust individuals with strong hearts and tense vessels; about 12 ounces of blood are withdrawn; the chief objection to venesection is the difficulty of differentiation; in thrombosis and embolism, venesection increases the anemia of the brain, and Cushing has shown that the high tension is a vicarious effort to keep the medulla supplied with blood. This objection holds to compression of the common carotid. (*d*) *Leeches, sinapisms* and the *ice-cap* cannot influence the circulation in the brain. (*e*) Evacuation of the *bladder* and *bowels* is indicated. Croton oil was Hughlings Jackson's only remedy; two drops act in an hour or two and may be repeated. (*f*) The *diet* should be fluid and administered by the nasal catheter or by rectum. (*g*) Decubitus is prevented by change of posture and cleanliness; heat and sinapisms must be avoided because the hemiplegic side is prone to necrosis. (*h*) *Symptomatic* treatment of cardiac failure is met by ammonia, etc; restlessness or convulsions are managed by chloral per rectum or morphine.

3. The Reaction.—During the *inflammatory reaction* the treatment is expectant and if possible should be conducted without drugs.

4. The Chronic Stage.—(*a*) Gentle *massage* of the muscles, movement of the joints and alcohol rubs are instituted as soon as the coma subsides. (*b*) *Potassium iodide* is indicated when the reaction subsides. (*c*) In four weeks, the patient should be encouraged to sit up and walk. (*d*) The *faradic current* is valuable after four weeks and, supplemented with massage of the paralyzed groups and their antagonists, minimizes the contractures. (*e*) *Hydrotherapy:* cool baths or rubs are efficacious except in poorly nourished individuals. Strychnine is contra-indicated in all cases.

V. Cerebral Embolism.—Embolism and thrombosis of a cerebral artery result in *brain softening* (encephalomalacia), which, next after hemorrhage in importance and frequency, is one of the best-developed subjects in brain pathology. Brain softening from arterial occlusion is not the "softening of the brain" known to the laity who give that name to progressive paralysis of the insane. *Encephalomalacia* must not be confused with *encephalitis*. Softening was first recognized by Abercrombie (1818) and Rostan (1820).

Etiology.—Embolisms involve the kidneys in 77 per cent., spleen in 54 and brain in 22 per cent. (*a*) About 90 per cent. are due to *heart disease,* most commonly *acute endocarditis.* Calcareous fragments or parts of torn valves may be thrown into the brain. Of valvular diseases, mitral stenosis produces embolism most frequently because of stasis in the left auricle. Less frequent are cardiac clots formed in the weak hearts of decrepid, cachectic subjects, in myocarditis or acute diseases, as typhoid. (*b*) *Atheroma of the arch and aneurysm* are far less frequent causes, as are (*c*) *lung lesions,* as tuberculous cavities, abscess, gangrene or empyema (after irrigation), tumors, echinococcus, gumma or abscess of the heart. (*d*) In the rarest instances lung tissue, fat embolism from fractures, pigment embolism in malaria or liver cells in acute yellow atrophy may produce cerebral embolism. (*e*) With a patent foramen ovale, thrombi may reach the brain from the venous system, as in manual expression of the placenta. (*f*) Most cases occur *in youth* when acute infections are most frequent, as chorea, scarlatina or endocarditis.

Pathology.—(*a*) The *embolus* first is grayish-red but later paler and friable; "riding emboli" straddle the point of arterial branching; they may by slipping produce another embolism farther on. Secondary thrombosis may occur at the infarction. (*b*) The *artery* beyond, save where the embolus retracts or softens, becomes a thin, hard, impervious cord. Behind the plug an aneurysm may form. If the embolus is septic, inflammation intervenes in and around the vessel. (*c*) As to the fate of the *brain tissue:* (i) If the stoppage is partial, only stasis, edema and swelling occur; if total, necrosis results. (ii) The brain arteries are "end arteries," as Cohnheim pointed out, whence complete collateral circulation is unlikely, although the *necrosis* is *rarely as extensive* as the *distribution of the artery occluded.* There is more collateral compensation at the cortex than at the base and the least in the deep parts of the centrum ovale or basal ganglia. The brain tissue is *ischemic,* but a degree of

collateral fluxion saves some tissue, especially if the heart is strong. With a weak heart or diseased collateral arteries, *ischemic necrosis* must follow. A marked hemorrhagic infarct is not common, being prevented by swelling of the nervous tissue. Microscopic changes are seen after one or two days; the brain becomes edematous, gelatinous, marbled, fusing with the slightly edematous brain tissue surrounding the focus; the brain rapidly becomes fluid and pulpy, although its remarkable cohesion is a matter of frequent comment; certain convolutions may waste or wholly disappear, as the hippocampus, lingual and fusiform lobules, especially after plugging of the posterior cerebral artery. The white matter often disappears and is replaced by lax cellular tissue, in whose meshes are cystic formations with turbid fluid and flocculi of brain tissue. Durand-Fardel described three forms of softening: (*a*) The *red softening* found most often in embolism is usually fresh and there is capillary extravasation; it is irregularly oval or wedge-shaped in basilar lesions; it usually measures but a centimeter or two; it is prominent on section from blood and serum; it is soft, but not fluid in the centre; it is mostly located in the cortex or central ganglia and lasts one or two weeks. (*b*) *Yellow softening* follows the red; the yellow color is due to old blood pigment or fatty change; it is sunken on section, from atrophy of the brain substance; is fairly demarked and may contain fluid, *hydrops ex vacuo;* it may last years and is found especially in the cortex. (*c*) *White softening* may represent (i) a *fresh lesion* about tumors or abscesses or in various cachexias, is sunken below the cut surface and occurs where few vessels are involved and little regurgitation from adjacent vessels occurs; (ii) old snow-white *lesions,* occurring when the blood and fat are absorbed; if diffuse it gives rise to the spongy *état criblé.*

Demarcation may occur after weeks, producing a "cyst." More rarely neurogliar increase may lead to shrinking and *scarring.* Myelin and fat drops, leukocytes laden with granules and fat, collapsed vessels and necrotic nervous relics are observed. *Secondary degeneration* occurs when the pyramidal tracts are involved (see page 776).

Symptoms.—As in apoplexy, we consider the *insult,* the *permanent symptoms,* and those of the causal disease (mitral stenosis, etc.).

1. The *insult* is (*a*) *sudden* and *without prodromes* (save when the embolism does not wholly occlude the vessel and symptoms appear only when secondary thrombosis develops). Embolism in a small vessel may occur without symptoms. The patient may be stricken *without coma* and sit "astonished" at the stroke. The insult is caused by *brain anemia,* negative pressure and shock. In some cases (*b*) *coma* occurs, generally *less profound* and *less protracted* than in hemorrhage; irritative symptoms, as (*c*) *convulsions, tonic spasms* and conjugate deviation are *more common;* convulsions are often unilateral, corresponding with the *hemiplegia.* (*d*) *Intermediate types* are observed; cases with vertigo; tendency to fall to one side; hemianopsia, alexia, hemianesthesia, aphasia or only confusion and delirium. (*e*) The *pulse is full but not slow.* (*f*) The *temperature* rarely suffers initial depression and later may rise.

2. The *permanent symptoms* and *secondary degeneration* are the same as in hemorrhage.

Localization of Embolism.—1. The Artery of the Fossa of Sylvius.—This most frequently involved (80 per cent.), largest and most direct branch of the middle cerebral is practically a continuation of the internal carotid. Convulsions occur in 33 per cent., with or without loss of consciousness, and are most frequent when the smaller branches are embolized. High temperature is common. The clinical picture varies with the site of infarction; embolism at *A* (Fig. 57) where the perforating branches (*P.P.*) are given off to the internal capsule, corpus striatum and anterior part of the thalamus, must involve (*a*) these structures,

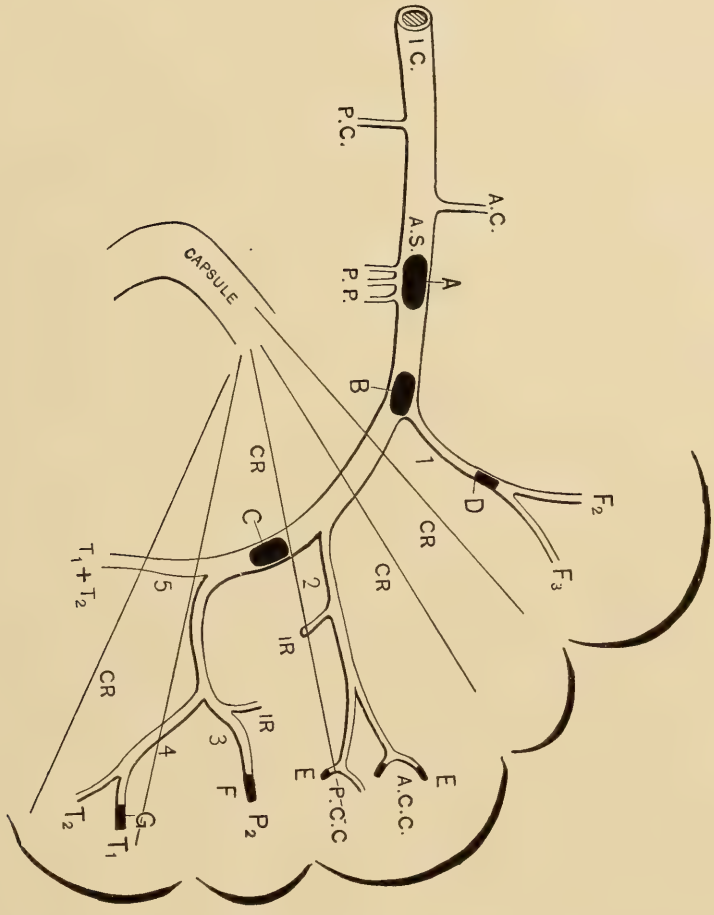


Fig. 57.—Embolism of artery of the fossa of Sylvius: *I.C.*, internal carotid; *P.C.*, post. communicating; *A.C.*, anterior cerebral artery; *A.S.*, art. fossæ Sylvii and 1, 2, 3, 4 and 5, its main branches. *CR*, corona radiata from cortex to internal capsule. (See text for explanation of lesions at *A*, *B*, *C*, *D*, *E*, *F* or *G*.)

and (*b*) the motor and sensory cortex, with *hemiplegia, hemianesthesia, motor aphasia* (alexia), *deafness* and *word-deafness* (if it occurs on the left side). Convulsions are absent, because the injured internal capsule cannot convey the cortical irritation to the extremities; the large areas rendered anemic may cause *marked reaction,* sometimes death. In rare cases, only the central perforating branches are involved and the cortex obtains the blood by anastomoses with the posterior and anterior cerebrals. The left Sylvian artery is more frequently involved (63 per cent.) because the left carotid comes directly from the arch. If the lesion is at

(*B*) (Fig. 57), the motor speech area, F_3, the anterior and posterior motor convolutions *A.C.C.*, *P.C.C.* (hemiplegia), the gyrus angularis and gyrus supramarginalis P_2 (alexia) and the temporal convolutions T_1 and T_2 (deafness or word-deafness) are involved. Somnolence and delirium are common; convulsions occur, because the internal capsule escapes. Convulsions and pain are most frequent in embolism of the smaller vessels. Little hemianesthesia is observed. A lesion at *C* is the most frequent site. It affects the island of Reil (*IR*) in part; cortical convulsions, hemiplegia, monoplegia, sensory aphasia (T_1, T_2), alexia (P_2) and sometimes sensory disturbance occur. It is seen how an isolated lesion at *D* could produce motor aphasia; at *E*, monoplegia; at *F*, alexia; or at *G*, word-deafness.

2. Posterior Cerebral Artery.—Embolism (in 2 per cent.) comes from the vertebral artery and often occurs with embolism of the Sylvian artery; it supplies (*a*) all the ventricles, the thalamus, ant. corp. quadrigemina, the geniculate body and the peduncle by its central branches, and (*b*) the mesial aspect of the occipital, part of the parietal and most of the temporosphenoidal lobes. The most common general symptoms are convulsions, coma, vertigo, conjugate deviation and nystagmus; while the most important focal symptoms are *sensory hemianesthesia, sensory aphasia* and *hemianopsia.* The lesions are sometimes symmetrical.

3. Anterior Cerebral Artery.—Few clear cases are recorded, since the artery originates at a right angle to the carotid.

4. Internal Carotid Artery.—In this embolism (4 per cent.) the plug must be large to produce damage, for if the vessels of the circle of Willis are normal, only transient hemiplegia results. Disease of these vessels, abnormalities in size or secondary thrombosis may produce permanent or fatal results. Sepsis is a frequent factor.

5. Basilar Artery.—Embolism occurs in 6 per cent.; the clot never fills the vessel, because it first passes the smaller vertebral artery; symptoms occur more frequently when secondary thrombosis occurs—convulsions, trismus, paralysis of the third, fifth and seventh nerves, crossed hemiplegia, diplegia, narrow pupils and conjugate deviation. The temperature often falls low and then rapidly rises. Death may follow in two to five days from respiratory failure.

6. Vertebral Artery.—Involvement of the tongue, larynx, lips, pharynx and spinal branch of the fifth nerve results in anesthesia, dysphagia or other signs of acute bulbar palsy. Hemianesthesia, hemiplegia and hemiataxia may develop. It is often fatal.

7. Cerebellar Arteries.—This is the rarest of all embolisms.

Diagnosis.—The main points are (*a*) recognition of the *cardiac cause;* (*b*) absence of *prodromes;* (*c*) *sudden* insult, frequent *convulsions* and infrequent *coma* (see page 781).

Prognosis.—The outlook depends (*a*) on the *primary* disease, being most favorable after acute infections and in youth; (*b*) on the *intensity of the onset* itself, during which death may occur; (*c*) on the *localization.* The initial symptoms may improve; the outlook is more favorable than in hemorrhage. Psychical symptoms are cortical in origin, while the vegetative or trophic are due to a lesion in the basal ganglia; basilar

and vertebral embolisms are most unfavorable. (*d*) Recurrence is possible, although it is less likely than usually stated. (*e*) The condition of *other organs*, as the vessels of the circle of Willis, the strength of the heart muscle, etc., determine the prognosis.

Treatment.—(*a*) The *insult* is treated as in hemorrhage in regard to diet and quiet. (*b*) All derivative measures, as *drastics* and venesection, are *absolutely to be avoided.* (*c*) The heart must be *stimulated*, if hemorrhage is excluded, to relieve the brain ischemia by a strong collateral circulation and to strengthen the heart to avoid recurrence. Digitalis acts too slowly, and camphor, strychnine and ammonia are preferable. (*d*) *Convulsions* should be treated by chloral given by rectum, avoiding alcohol; more than a dram of chloral in three hours is very dangerous. (*e*) The *after-treatment* is as in the corresponding stage of apoplexy.

VI. Cerebral Thrombosis.—It is more common than embolism.

Etiology and Pathology.—1. Changes in the Vessel Wall.—(*a*) *Arteriosclerosis* (*q. v.*) *is the most important cause.* Blood plaques are deposited on the rough intima of the tortuous, inelastic, calcareous vessels, with their unequal caliber; layer after layer of fibrin is deposited, aided by local slowing of the blood stream and weakening of the heart. Tumors, abscesses and traumatic meningitis may initiate thrombosis. Brain atheroma usually occurs with general atheroma. The vessels most involved are, in order of frequency, the carotid, middle cerebral and its Sylvian branch, basilar, vertebral and posterior cerebral. (*b*) In *syphilitic endarteritis* the initial proliferation may of itself, without thrombosis, obliterate the vessel lumen (see Brain Syphilis). The pathological sequence is the same as in embolism; ischemia, necrosis (softening) and loss of function occur, save that in gradual thrombosis the focus is less homogeneous than in the sudden lesion of embolism. The foci are often multiple.

2. Changes in the Heart.—Sudden cardiac insufficiency in arteriosclerosis may precipitate thrombosis, as brought out especially by Kolisko. An acute infection in the aged, low arterial tension from exhaustion, grief and similar causes may induce encephalomalacia.

3. Changes of the Blood.—This constitutes the smallest class of cases. Burns, poisoning by carbon monoxide and illuminating gas, gout, chlorosis, leukemia, metallic poisons, jaundice and marasmus in very young children or in extreme old age may cause thrombosis. Acute infections and heart weakness are frequent factors in this class. Thrombosis affects the smaller vessels and is usually multiple.

Symptoms.—In some cases thrombosis is found at necropsy without previous symptoms ("latency").

1. Prodromes.—These are frequent and may appear hours, days, even months before focal symptoms develop. The most common are headache, which is severe in the syphilitic variety, vertigo, numbness and weakness in a limb or in one-half of the body, disturbance in sight, mind, articulation or character.

2. Onset.—The onset varies; (*a*) an *acute insult* is not frequent. If consciousness is lost, the coma is less deep and protracted than in

hemorrhage; a "stroke" indicates thrombosis in one large or in several vessels. Conjugate deviation, flaccidity and paralysis are frequent during the coma. Consciousness is usually preserved in the syphilitic type. (*b*) The *onset is usually gradual.* Layer after layer is added to the clot but strengthened heart action may for a time maintain collateral circulation. When the heart weakens, the clot increases and the collateral blood supply decreases, so that, after alternating advance and regression, the final thrombosis results, with irreparable softening. The cardiac strength and the number and size of the vessels involved determine the acuity or chronicity of the course. *Reaction* may be marked, especially in the aged. (*c*) In the *chronic* or long *latent* variety, prodromes are absent, psychic alteration is apparent and focal symptoms develop.

3. Chronic Stage.—Focal symptoms are common. Apoplectiform seizures mark the advance of the process in new vascular areas. The motor or sensory paralysis presents much the same picture as hemorrhage and embolism in their chronic stage.

Localization.—Localization is impossible when multiple foci exist, as in the Sylvian artery of one side and the posterior cerebral artery of the other; to the few reported cases Senator adds one in which left-sided hemiplegia was associated with aphasia.

1. Sylvian Artery.—The most common cause is syphilis or atheroma. When the *main trunk* is involved, the same symptoms appear as in embolism, viz., hemiplegia, etc., with this modification—the *focal symptoms are more severe* in thrombosis, because other vessels are somewhat diseased; the *general symptoms are less severe.* Prodromes appear first, as tingling, then hemiplegia, possibly with delirium or loss of consciousness; the symptoms may improve, then there may be recurrence with aphasia and coma. The course is a "step-like" progression and regression. *Monoplegia* is more common than in embolism, because thrombosis seeks the cortical vessels, whence the face and arm may be affected; aphasia may exist alone, or alexia, mind-blindness or deafness.

2. Anterior Cerebral Artery.—This artery is rarely totally occluded. A clot beyond the anterior communicating vessel produces crural monoplegia with arm paresis. Very *gradual ischemia* produces *wasting* without softening; sudden ischemia produces softening. The frontal lobes present the "worm-eaten" atrophy, with abundant *hydrops ex vacuo;* its close arterial connection is obvious.

3. Posterior Cerebral Artery.—Thrombosis may result in hemiplegia alternans oculomotoria, hemihypesthesia and optic changes varying as the optic radiation, cuneus or other parts are affected.

4. Basilar Artery.—If the thrombosis is acute the symptoms are like those of embolism. If slow, ophthalmoplegia which is rarely lasting, or other motor paralyses, hemiplegia with alternating palsy of the fifth, sixth and seventh nerves, may develop.

5. Vertebral Artery.—*Acute bulbar palsy,* with stormy symptoms, cannot be distinguished from embolism. *Subacute bulbar palsy,* with *dysphagia,* dysarthria, hemianesthesia, *homolateral ataxia, contralateral hemianalgesia* or hemiplegia with crossed paralysis of the tongue may result.

Hemorrhage.	Thrombosis.	Embolism.
(1) *Predisposing etiology.* Before third year (glioma); 80 per cent. after fortieth year.	After eightieth more year frequent than hemorrhage. Twentieth to fortieth year syphilis (84 per cent. occur between these years).	Youth; hemorrhage practically never in youth.
Miliary aneurysms; atheroma and cardiac hypertrophy.	Weak, irregular heart; fevers, cachexia. Atheroma, especially syphilis, alcoholism, plumbism.	Endocarditis (rheumatism, sepsis), especially mitral. Weak, fatty, dilated heart. In heart disease Eichhorst found in 60 per cent. hemorrhage and in 40 per cent. embolism.
Heredity. Nephritis; cause in 33 per cent., "granular kidney."	Rare.	Rare.
(2) *Attack precipitated* by heavy meal, acute alcoholism, high art. tension (excitement, effort, shock).	Rare.	Rare.
(3) *Prodromes* unusual, unless from causal arteriosclerosis.	Low arterial tension; by sleep, grief, exhaustion, fatigue.	By exertion, dislodging clot.
(4) *Insult:* Usually sudden; without prodromata; long duration (fatal if more than two days). Cause; positive pressure producing brain anemia.	Very common, mental irritability, weak memory, headache, vertigo, numbness, tingling, weakness in extremities.	Absolutely none.
(5) *Intra-cranial pressure:* "Pressure symptoms" present, polyuria, albuminuria, glocosuria, red face, beating arteries, Cheyne-Stokes's breathing; slow, hard pulse is the fundamental difference (Brissaud); bruit de gallop speaks strongly for hemorrhage. Irregularity, if combined with strength not against hemorrhage. Pulse may be fast (Brissaud).	Rarely acute onset (unless in a large vessel); usually gradual, especially in senile softening. If an insult, focal and general symptoms usually appear during the coma.	Sudden, shorter and less severe than in hemorrhage. Cause; negative pressure. Slow insult only with secondary thrombosis.
(6) *Temperature:* Rectal temperature usually depressed at first, may fall till death. May rise later (inflammatory reaction). Early rise (pons, medulla, ganglia). Sudden rise of bad omen; rises before death.	None. Pulse soft, weak. (Wassermann test). Initial depression, usually lasting. Seldom temperature unless lesion in pons. More probably early reaction. Especially in the aged.	None (pallor rather than congestion). Temperature may rise high at once without bad omen. Temperature no absolute guide; occurs especially when large trunk embolized.
(7) *Coma:* More frequent, greater in degree, longer in duration, usually complete. If lesion seems small, coma speaks for hemorrhage. Deep and prolonged speaks for hemorrhage and usually fatal if over twenty-four hours.	Less frequent and shorter. Recovery possible after several days of coma. None in syphilitic thrombosis. If lesion seems large, consciousness speaks for softening. If coma, usually focal symptoms are then seen. Coma in large bilateral lesions.	No coma usually.
(8) *Paralysis,* sudden, complete, hemiplegic, non-progressive after first seizure. Foot gains more rapidly than hand. Persistent aphasia exceptional. Spasmodic laughing and weeping common.	Gradual, incomplete, monoplegia, hemianopsia; repeated, often symmetrical paralysis. Converse. Aphasia common. Absence of focal symptoms rather more common (central ganglia). Laughing and weeping exceptional.	More like hemorrhage. Monoplegia rare. In 63 per cent. it is in the left side of the brain. Fairly common.
(9) *Convulsions:* Usually generalized; very rare from cortical irritation or in corpus striatum; post-hemiplegic are rare.	Less than in embolism. More localized, Jacksonian epilepsy speaks for softening. May recur. Post-plegic frequent.	More common (33 per cent.) than in hemorrhage or thrombosis (lesion cortical.)
(10) *Sensation:* Anesthesia usually transient.	Paresthesia persists and more marked. Cerebral pains (hyperesthesia dolorosa).	Paresthesia persists and more marked. Cerebral pains (hyperesthesia dolorosa).
Postplegic chorea, trembling, athetosis, common.	Less common.	Less common.
(11) Atheroma retinal arteries, hence retinal hemorrhage (not significant unless large). Aneurysms very rare but speak strongly for hemorrhage. Retinitis albuminurica.	Arteriosclerosis.	Optic retinitis sometimes from endocarditis (Broadbent and Mackenzie). Occasionally embolism in retinæ.
(12) *Secondary reaction;* not much. *Psychical symptoms;* far less.	Greater secondary reaction. Often greater; intelligence, memory aphasia, word-deafness, etc.	Very seldom; after attack may be some delirium.

Diagnosis and Differentiation.—The *prodromes, gradual onset* and *cortical location* are most distinctive, although the differentiation may be wholly theoretical or impossible (page 781). In *brain tumor* the

course is slower, and stabile focal symptoms less frequent—that is, headache, vomiting, choked disk and Jacksonian epilepsy are far more frequent than permanent paralysis. We may consider *hemiplegia in the aged*, Marie's *les lacunes de désintegration cérébrale;* multiple *degeneration clefts* occur, chiefly in the large basal ganglia, sometimes in the internal capsule, pons, or centrum ovale. Marie holds that the lesion is a rarefaction (cavity formation) of brain tissue due to chronic sclerosing *encephalitis, without vessel occlusion.* It is said to cause 90 per cent. of "insults" in the aged, usually with *partial paralysis;* sometimes with aphasia, dysarthria or dysphagia; rarely with loss of consciousness; and never with sensory disturbance or contractures. The gait is the *démarche à petits pas.* The patient may live ten years.

Prognosis.—The prognosis depends on (*a*) the *cause;* it is unfavorable in *arteriosclerosis*, because collateral vessels are probably diseased. If the other vessels are sound, enormous disease may be compensated, as in Kussmaul's case of occlusion of both the subclavian and carotid arteries. In *recent syphilis* the prognosis is more favorable, though old specific thrombosis is incurable. The younger the patient the better is the outlook. (*b*) *The onset;* most cases with a brusque beginning die. Coma indicates thrombosis in large vessels or in both hemispheres. If there is no insult the focal symptoms are irreparable, *i. e.*, there are no indirect symptoms. The first seizure does not usually threaten life. (*c*) The *location and extent of the lesion.* The number of vessels involved is obviously important. In Sylvian thrombosis involving the branches to the central ganglia, the patient dies in four to six weeks with involuntary evacuations, cachexia and bed-sores. Cortical lesions are prone to produce psychical alteration. Basilar or vertebral thrombosis is directly dangerous. (*d*) The *focal symptoms* are likely to *persist* and *recur.* Ophthalmoplegia may regress. (*e*) The condition of other organs, above all the *heart*, is of prognostic import.

Treatment.—(*a*) The *general treatment* is the same as in hemorrhage. (*b*) The *causal treatment* relates to the iodides, valuable in atheroma and syphilis. (*c*) The *heart* must be stimulated with digitalis; absolute quiet in the prone position and free administration of food and fluid by rectum or by nasal catheter are indicated. Drastics and depleting measures must be avoided. On the heart devolves the entire possibility of collateral compensation. (*d*) In the *period of reaction* alcohol only accentuates the inflammation. For delirium and headache, bromides are most useful; no cardiac depressants, *e. g.*, chloral or coal-tar products may be used. (*e*) The *chronic stage* is treated as in hemorrhage. At all times catheterization must be conducted antiseptically and great care exercised against bed-sores.

VII. Intracranial Aneurysms.—Etiology.—Aside from miliary aneurysms, aneurysms may occur in the large brain vessels, more frequently in males (60 per cent.), and more (53 per cent.) before than after the fortieth year. The causes are (*a*) *atheroma* in whose causation alcohol is particularly important; (*b*) *trauma*, initiating arteritis, especially in the internal carotid, as after basal fracture; (*c*) *syphilis*, especially productive of basilar aneurysm, often within a year after infection; (*d*)

embolism, the most frequent cause in early life; *endocarditis* is the usual cause when trauma and syphilis are excluded. The embolus produces arteritis from which the vessel dilates. The clot may later disappear.

Pathology.—LOCATION.—Statistics show the following frequency; middle cerebral, 29 per cent.; basilar, 26; internal carotid, 14; anterior cerebral, 8; anterior communicating, 6 per cent. They are rather more frequent on the left side, in 20 per cent. are multiple, are usually sacculated, of the size of a pea or walnut (rarely as large as an egg) and are genuine aneurysms involving all coats. Beadles collected 555 cerebral aneurysms with autopsy (1907).

Symptoms.—In only 37 per cent. are symptoms present. They are (*a*) *general*, as *headache*, which is the most common sign, or *convulsions*, which result from aneurysm of the Sylvian branch; a systolic murmur was found in but 21 per cent.; in a personal observation, cured by operation, a continuous murmur over half the head was associated with exophthalmos. (*b*) *Local* symptoms; according to their location, hemianopsia, ocular paralysis, hemiplegia, optic neuritis (extension of inflammation from the sac), aphasia and symptoms referable to the pons or medulla may be noted. *Rupture* occurs in 67 per cent., into the meninges, brain substance or ventricles, from which death rapidly results; healing has been observed.

Diagnosis.—*Rupture* is generally the first symptom and differentiation from apoplexy is therefore necessary; cerebral hemorrhage in a young individual with a history of trauma, heart disease or syphilis suggests aneurysm, especially when the location is basilar; iodides and mercury usually are not beneficial in aneurysm. The suggestive murmur is rare and may be found in tumors which compress vessels or in very vascular brain tumors. Aneurysm is most often embolic in the Sylvian and specific in the basilar artery. Loss of sight in an eye, sometimes with impairment of smell, rather indicates aneurysm of the anterior cerebral or, if the eye muscles are paralyzed, of the internal carotid artery. Oculomotor paralysis without loss of vision suggests aneurysm of the posterior communicating artery. Some cases closely resemble arterial thrombosis.

Treatment.—The indications are rest and iodides. Ligation of the carotid is occasionally opportune.

VIII. Sinus Thrombosis.—Lebert (1854) diagnosticated the first case and with Tonnelé built up its clinical recognition.

Etiology and Pathology.—There are two varieties, the marantic and the inflammatory types.

1. *Marantic thrombosis of the sinuses and veins* is the *primary non-inflammatory* type, due to altered blood states and weakened circulation. It occurs (*a*) in *children* mostly, especially in the first six months of life and after acute infections, particularly infective *diarrheas;* (*b*) less frequently in the *aged* from weak heart and venous stasis; (*c*) in *cachexia;* (*d*) in *chlorosis;* (*e*) *compression*, as by tumors, is rare. Its most frequent site is the superior longitudinal sinus, especially in children, because the sinus is fixed, its lumen is triangular and irregular, crossed by trabeculæ and compressed by the Pacchyonian granulations; the tributary veins ascend against the current of the sinus and their stream is very weak.

Simple slowing of the current is not sufficient for coagulation and the vessel wall must be altered. In the aged, marantic thrombosis elects the cavernous or transverse sinus. The marantic clot may enter the tributary veins which are seen as firm cords over the hemispheres.

2. The *inflammatory thrombosis, thrombophlebitis,* is secondary to disease near the sinus; as (*a*) *ear disease,* chiefly chronic, suppurative otitis or *caries* of the bone; infection reaches the sinus by (i) infecting the mastoid cells, which infect the transverse sinus; (ii) by infecting

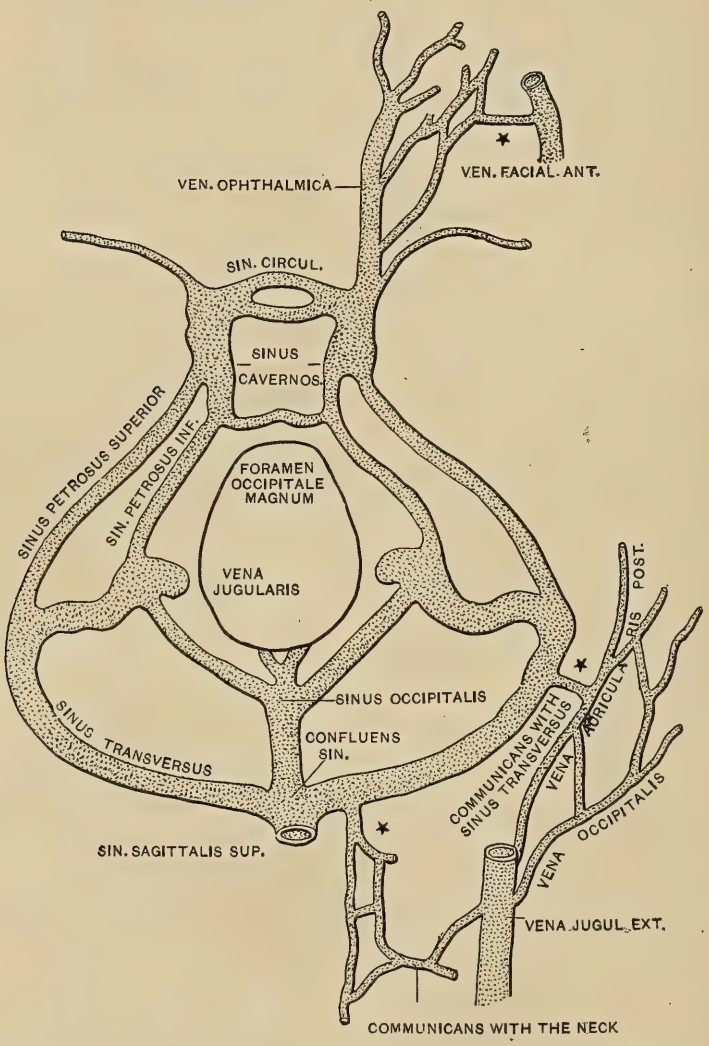


Fig. 58.—Diagram showing the connections (*) of the transverse and cavernous sinuses (Leube).

small communicating veins or (iii) its passage along the petromastoid canal. (*b*) *Acute infections,* with otitis media; *sepsis; trauma, tuberculous meningitis,* disease of the orbit, nose and face. The most common *sites* are the transverse, petrosal and cavernous sinuses, whence it may extend to their branches. The clot is puriform, fetid, soft, discolored and contains pyogenic organisms; the process is frequently associated with meningitis and brain abscess.

Most frequently the thrombosis is bilateral; suggillations in the pia, bloody imbibition of the brain or reddening of the cerebrospinal or ven-

tricular fluid may occur. Simple or septic pulmonary infarcts occur in 50 per cent.

Symptoms and Diagnosis.—Clinical signs may be absent.

1. Extracranial Stasis.—(*a*) In thrombosis of the *superior longitudinal sinus*, stasis in the nasal veins is often shown by *epistaxis*, an effort of nature to deplete the cerebral congestion. Gerhardt observed *cyanosis* in the anterior frontal veins and *edema* in the temples between the great fontanelle and ear (by way of the emissaria Santorini through the foramen parietale). Edema is often absent in chlorotic thrombosis. (*b*) In thrombosis of the *cavernous sinus* stasis develops in its tributaries, producing *exophthalmos, edema of the lids* and conjunctiva (vena ophthalmica); *retinal stasis*, more rarely choked disk or retinal thrombosis,

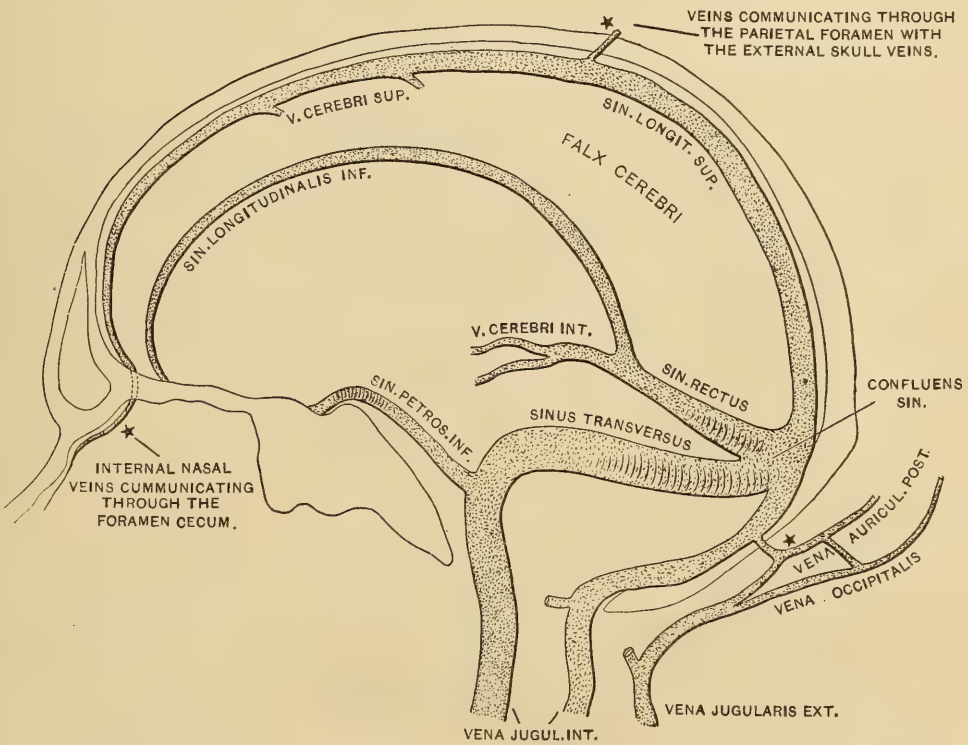


Fig. 59.—Leube's diagram showing the connections (*) of the superior longitudinal sinus with the external veins.

results from congestion of the vena centralis retinæ. Sometimes edema of the forehead results. (*c*) In thrombosis of the *lateral sinus, edema behind the ear* and *in the neck* (communicating with the posterior auricular vein) occurs in over 50 per cent. Occasionally the *swollen thrombotic internal jugular vein* may be felt. The *external jugular collapses*, or if the internal jugular is thrombosed it becomes overfilled (Gerhardt). In 33 per cent. the disk is hyperemic and in another 33 per cent. there is choked disk from complicating abscess or meningitis.

2. Intracranial Stasis.—Progressive clouding of the mind, delirium in adults, convulsions in children, conjugate deviation of the eyes and head, stiff neck, vomiting, headache, irregular pupils, nystagmus or strabismus are noted. Bilateral symptoms, as paraplegia, are due to the involvement of the veins in both hemispheres or more often to

meningitis. Bouchut thought that antemortem convulsions in children were often thrombotic in origin. The fontanelles, sunken at first in infantile diarrhea, may later become prominent (increased cerebrospinal fluid). Sometimes chlorotic thrombosis may extend to the vena Galeni magna, producing hydrocephalus internus, stupor, coma and death. Irritation or paralysis of the third, fourth and sixth nerves may occur because of their intimate relation to the cavernous sinus; involvement of the first branch of the fifth nerve may result in ophthalmia neuroparalytica.

The temperature is often normal in the simple, or in the thrombophlebitic variety attended by fever, chills and sweats.

DIFFERENTIATION.—Differentiation from meningitis (*q. v.*) and brain abscess (*q. v.*) is often impossible; the cause, the extracranial stasis and evidences of sepsis are of diagnostic importance. Körner held that metastases occur in the lungs from sinus thrombosis and in the bones and joints from mastoid caries.

Prognosis.—Death occurs in the second week, possibly later. Few recoveries from either type are on record.

Treatment.—*Prophylaxis* relates chiefly to the otitic variety; discharges from the ear are too lightly regarded by the laity. In *marantic thrombosis* stimulation and avoidance of constriction of the neck are the chief indications. In *septic types* operation is indicated; recovery is most likely when there is early diagnosis with early operation. Opening and packing the lateral sinus was first performed by Zaufal in 1884. Ligature of the internal jugular veins, as first performed by Thersley and Lane in 1888, gives recovery in 50 per cent. and in 72 per cent. of Macewen's series.

IX. Infantile Cerebral Paralysis.—Cerebral infantile palsy is classed under circulatory diseases because of the frequent etiological hemorrhage, embolism and thrombosis. Pathologically, it is sometimes encephalitis, sclerosis or porencephalia. Clinically, it is variously classed according to the dominant syndrome, hemiplegia, diplegia, Little's disease, paraplegia, hemiathetosis and athetosis.

1. **The Hemiplegic Form.**—ETIOLOGY.—It is equally frequent in either sex. Gowers finds 88 per cent. in the first five years of life. *Infections* as scarlatina, measles, pertussis and diphtheria are apparent, and heredity, maternal syphilis and trauma are doubtful causes. The causes may operate before, during or after birth.

SYMPTOMS.—(*a*) Initial symptoms, present in 66 per cent., are severe in character, consist of *fever*, vomiting and delirium, and last from a few hours to several days; convulsions, present in 50 per cent., are often unilateral and are usually followed by coma. In 33 per cent. of cases the onset is insidious. (*b*) The *paralysis* (hemiplegia) usually develops when consciousness returns. It is more frequently right- than left-sided. The lower face is involved, but usually soon improves; the leg improves more than the arm and hand; and the paralysis, at first flaccid, soon becomes *spastic* and permanent. Tactile and stereognostic sense may be reduced, but sensation is slightly implicated. (*c*) The *residual symptoms* are the same as in adults, with some exceptions; the paralysis is *spastic*

(spastic infantile hemiplegia, Heine) but also *atactic* and *athetotic; retarded development* appears, *intellection is retarded or actual idiocy results* and *epileptiform seizures* are common. *Contractures* are frequent, the *reflexes* are increased and the gait is somewhat impaired—the foot-dragging. In rudimentary or benign cases the paralysis may almost disappear, and, after months or years, may be replaced by epileptic seizures, hemiathetosis, hemichorea, hemitremor (described under Hemorrhage) or hemihypertonia (rigidity with but little paralysis). *Aphasia* is rarely lasting; (*a*) *reflex aphasia* is very common in childhood from fever or digestive difficulties; (*b*) the brain readily compensates in childhood; (*c*) permanent aphasia indicates great or total loss of intelligence. *Trophic changes* are in part a moderate atrophy and in part a lack of later development; they are most common in porencephalia (*v. i.*). *Epilepsy* increases in frequency after the paralysis and is rated at from 13 per cent. to even 66 per cent.; it occurs at the onset with the paralysis or a year or two later; it is more marked in rudimentary palsy; it is most often like the Jacksonian type, but may end in total loss of consciousness; there is less violence, initial cry, foaming and involuntary evacuations than in ordinary epilepsy. After a decade or more it often becomes very marked.

Diagnosis.—Its separation from poliomyelitis is thus made:

	Cerebral type.	Spinal type.
Tendon reflexes.	Increased.	Decreased or abolished.
Muscular atrophy.	Only after a long time.	Rapid.
Electrical reaction.	Normal, or little quantitative decrease.	Partial or total reaction of degeneration.
Distribution of paralysis.	Hemiplegic—diplegic.	Chiefly monoplegic.
Intelligence.	Often involved.	Usually not.
Epilepsy and hemichorea.	Common.	Rare.

2. **The Double Hemiplegic or Diplegic Form.**—Etiology.—(*a*) *Prenatal causes,* apparent in 20 per cent., include fright, trauma, malnutrition or syphilis, especially in cases which develop athetosis. (*b*) *Anomalies of labor* include difficult or long labor, malpresentation, resistant maternal tissues, twin pregnancy, version and cord prolapse; delivery by forceps is less important than the causes calling for their use. *Asphyxia* is an important etiological factor. In premature delivery the soft skull may be injured or venous stasis occurs. (*c*) *Extra-uterine causes* include trauma and acute infections.

Pathology.—The *initial lesion* cannot always be determined. (*a*) In the *traumatic class, meningeal hemorrhage* causes 66 per cent. *of the diplegic cases;* it is promoted by *asphyxia* and the sliding of the skull bones of one side under or over those of the other side. The blood is absorbed slowly, if the brain is lacerated, resulting in atrophy of both motor areas. (*b*) *Vessel disease* (18 per cent.) may be arterial (thrombosis, embolism or hemorrhage) or venous (in the cortical veins). (*c*) Strümpell advanced the views that *acute poliencephalitis* in the motor cortex causes the acute onset, an argument largely based on analogy (poliomyelitis in children).

Chronic Lesions.—The most important are diffuse lobar sclerosis and porencephalia, which are peculiar to infantile brain palsies: (*a*) *Diffuse lobar sclerosis* and atrophy of the brain occur in 55 per cent. About the atrophic areas and in the ventricles the cerebrospinal fluid is increased. The lobe affected is shrunken, hardened. The convolutions are wasted and smooth or "worm-eaten." The nervous tissue is wasted, the neuroglia increased and the bloodvessels tortuous and thickened. The nature of the initial lesion is disputed; it may be encephalitis, poliencephalitis or *embolism.* (*b*) *Porencephalia* (Heschl, 1859) consists of loss of brain substance and cavity formation. It occurs in 26 per cent. of cases. The initial lesion is ascribed to agenesis, encephalitis or meningo-encephalitis, trauma or *anemic necrosis* (Kundrat), the most widely accepted view. It is most frequent in the distribution of the Sylvian or occipital vessels. Most often it is congenital and then the convolutions radiate from the defect, while in *postnatum* cases, the convolutions stop short of the defect "as though cut off." Sixty per cent. are bilateral and somewhat symmetrical. (*c*) The *hypertrophic, nodular sclerosis,* described by Bourneville, consists of multiple discrete, prominent, often umbilicated nodes the size of a quarter- or half-dollar. They are overgrowths of the neuroglia.

Fig. 60.—Spastic paraplegia, cross-legged progression. (Dercum.)

Symptoms.—Diplegic cases are half as frequent as the hemiplegic. Freud describes the following forms: (*a*) *General rigidity,* or Little's disease, 33 per cent. of which are caused by asphyxia. The main characteristics are (i) *predominance* of the *rigidity* over the paresis, and (ii) *predominance* of the *leg paresis* over that of the arms, the converse of adult cerebral paralysis (hemorrhage occurs most often over the leg centres). Convulsions often usher in the symptoms, which appear after birth or a few months later. The rigidity is general but most conspicuous in the *flexors and adductors,* leading to unusual postures. The face is slightly involved, the maximum manifestation being spasm of the mouth; in rare cases the child cannot nurse or swallow. Speech is slow in development and stuttering is common. In 33 per cent. the head cannot be lifted or the trunk flexed, due to involvement also of the uncrossed pyramidal tracts. The arms may be but slightly paretic, but the legs suffer extensor spasm on movement and the toes turn down (talipes equinus or equinovarus). The child learns to walk late, if at all, because of the adductor rigidity, which may cause crossing of the legs. The reflexes are greatly increased. The intellect is somewhat affected in 66 per cent. Nutrition

in the paretic members is usually intact. Later epilepsy is uncommon. The paresis may regress and the mind improve, but the legs remain spastic. (*b*) *Paraplegic rigidity* resembles type (*a*) except that the arms are but little or not at all affected and the legs alone are rigid. The name Little's disease is also given to this form. Strabismus is common. Premature delivery is a factor in 50 per cent. of cases. (*c*) *Paraplegia* is rare and is referred to extra-uterine causes or infantile infections. The legs are rigid and paralyzed. In severe types there may be strabismus and imbecility. (*d*) In *bilateral spastic hemiplegia* (spastic diplegia), mental degeneration, nystagmus, convergent strabismus, bulbar symptoms and speech involvement (60 per cent.) are frequent. (*e*) *General infantile chorea* is also called "choreic diplegia"; asphyxia is a factor in 25 per cent. Its clinical characters are paralysis, rigidity and chorea-like movements which are irregular, slight in degree and wide in distribution. (*f*) In *bilateral athetosis* the mind is less involved than in the preceding types. The paralysis may precede it or it may be primary.

In all forms *epilepsy* may (i) replace the regressing paralysis; (ii) overshadow the paralysis; (iii) paralysis may appear only after years of epilepsy, or (iv) may not appear at all, "cerebral paralysis without paralysis."

Diagnosis.—Separation from the family spastic paralysis (*q. v.*) may be difficult or impossible.

Treatment.—In the initial stage convulsions may be treated by chloral. Some regression in the paralysis is frequent, but the hemiplegia, mental changes and epilepsy are incurable. Marked improvement can be expected in the syphilitic cases alone. Orthopedic devices are valuable. The resulting epilepsy is usually intractable and bromides with opium are of little avail. Twenty-five per cent. die from operations, but the surviving cases seem to improve, especially in regard to the convulsions.

BRAIN TUMORS.

Etiology and Pathology.—Eichhorst found brain tumors in $\frac{1}{6}$ of 1 per cent. of his cases. Sixty-six per cent. of tumors occur in the *male sex*, especially glioma and tubercle. *Age.* In the first decade, 18.5 per cent. of cases are observed; in the second, 14; third, 20; fourth, 18.5; and in the fifth, 14 per cent. Trauma may act as the "*agent provocateur*" in glioma, gumma, sarcoma, tubercle or aneurysm. The author has seen endothelioma of the dura develop years after and directly at the seat of an unhealed skull fracture.

Classification.—1. The Infectious Granulomata.—(*a*) *Tubercle* constitutes 50 per cent. of cerebral tumors, 75 per cent. being found in the first and second decades; 33 per cent. occur in the cerebellum, 33 per cent. in the cerebrum and the remainder in the pons, central ganglia, crus, etc. With equal frequency they are single or multiple (even 20 or 80). They are firm, rounded tumors, with grayish-red periphery and yellow, caseated centre. Tubercles range from the size of a pea or walnut to that of an egg or the fist, from fusion of many small growths. They grow along the lymph vessels, cause vascular thrombosis, present the

degenerations of tubercle of other tissues and may be shelled out of the brain tissue, which is compressed and atrophied, but neither infiltrated nor softened. (*b*) *Gummata* (see BRAIN SYPHILIS) in the adult are the most common type of tumor. They are most frequent in the cerebral hemispheres and pons and are rare in the cerebellum and central ganglia; they are most often the size of a pea or hazel-nut. Their centre is yellowish-gray and their periphery is reddish-gray, gelatinous, rich in cells and ultimately cicatricial. Gummata are usually multiple. The adjacent brain tissue is not infiltrated, but commonly softened and compressed. Microscopically, they closely resemble tubercle, but are more nodose, irregular, more distinctly connected with the dura, even when deeply located, more gelatinous and less caseated.

2. NEURO-EPITHELIAL GROWTHS.—(*a*) *Glioma* constitutes 25 per cent. of brain growths; 50 per cent. occur in the hemispheres, especially the cortex; 25 per cent. in the cerebellum and the balance in the central ganglia, pons, medulla, crus, etc. They occur more frequently in the brain than in the cord or retina, to which three structures they are peculiar. Gliomata are, in 90 per cent., single and are seen most frequently in adults, largely in the gray substance, which they so closely resemble in tint that detection is difficult at operation or indeed at autopsy. Their size varies from that of a pea to that of a hemisphere. Glioma infiltrates without sharp demarcation rather than compresses the brain tissue. The cells resemble the neurogliar structure and usually have round or oval nuclei; Klebs found ganglionic cells. Their processes are fine; numerous and branched delicate band-like lines result from fiber degeneration. (*b*) The rare *cerebroma* of Hayem is heterotopic gray matter. (*c*) *Adenoma* of the pineal gland and hypophysis, growths from the plexuses and neuromata may be brought under this heading.

3. CONNECTIVE-TISSUE TUMORS.—(*a*) *Sarcoma* consitutes 20 per cent. of brain tumors; it may develop in the meninges, periosteum or vessels. It is most common in adults. Though it infiltrates more than tubercles or gummata, many are well demarked. Its structure and degenerations are those of sarcoma elsewhere. Though commonly single, diffuse or multiple sarcomatosis is sometimes encountered. Endothelioma, cylindroma, angiosarcoma and melanosarcoma are but varieties. The sand tumors (psammomata) are sometimes described as angiolithic sarcomata. The "pearly tumors" (cholesteatomata) are formed of flat endothelial cells. (*b*) Lipoma, fibroma and osteoma are very infrequent.

4. METASTATIC TUMORS.—Cancer is not infrequent (7 per cent.), especially from mammary or lung carcinoma. It often invades the hemispheres and basal ganglia and may attain huge dimensions. Like sarcoma, it infiltrates. Cancer is very rarely primary.

5. CYSTIC TUMORS.—Cysts may result from hemorrhage or softening, degeneration in neoplasms, porencephalia, trauma or inflammation in the choroid plexus. *Dermoids* and teratomata are most rare. *Echinococcus cysts* may be single or multiple, chiefly on the surface of the brain or in the meninges. *Cysticercus* cysts develop mostly on the pia and arachnoid over the cortex along the sulci or in the ventricles. Diamond could collect but 8 cases from the American literature; they

vary from the size of a pea to that of a hazel-nut, are usually multiple (200 in Snell's case) and contain clear fluid with black dots, the heads of the parasites.

6. Aneurysms.—Aneurysms have been considered.

Localization.—The localization is: cerebrum, 42 per cent.; cerebellum, 25; base, 11; pons, 8; central ganglia, 7; medulla, 4; corp. quadrigemina, 2; and crus, 1 per cent.

Symptoms.—In rare instances, tumor is found at autopsy without clinical symptoms (*latency*), due to location in a "silent" or tolerant brain area. Symptoms result from the following *mechanisms:* (*a*) *Direct pressure* on the brain, which may flatten the convolutions, obliterate the sulci, force open the skull fissures in children, absorb or perforate the cranium, conspicuously in growths of the posterior fossa closed by the rigid tentorium. (*b*) *Internal hydrocephalus,* distention of the ventricles, may follow closure of the aqueduct of Sylvius or compression of the veins (choroid plexus and vena Galeni magna), especially by tumors of the cerebellum and corp. quadrigemina. (*c*) The *brain* may be softened by pressure on its vessels, or hemorrhage may develop (as in glioma). (*d*) Infiltration or destruction of the brain matter. (*e*) *Meningitis,* local as in gumma, or general as in tubercle. (*f*) *Auto-intoxication* from the growth. (*g*) Distortion of the cerebral or spinal nerves may cause their degeneration, or changes in the posterior columns of the cord and its roots.

1. General Symptoms.—General symptoms usually introduce the disease and include the following: (*a*) *Headache* is present in almost all cases, is usually constant, with accessions (especially in the morning), is general, severe, is increased by alcohol, exertion and coughing, and rarely corresponds with the tumor's site except when the pain is persistently local and the tumor near the surface. Headache is best explained by stretching of the dural branches of the fifth nerve. It may be pulsating in aneurysm. (*b*) *Choked disk,* the most important general symptom, consists of a grayish-red swelling of the disk to even two or three times its normal dimensions, which lessens its sharpness of outline and physiological cupping; the arteries become less lustrous and smaller, the veins turgid and tortuous, extravasations of blood and white patches of fatty degeneration or exudation appear. (See Plate IV, Fig. 3). It is due to *increased intracranial tension,* which causes mechanical hydrops of the optic sheath or to inflammation from choking of the veins by toxic products. It is bilateral, although more marked on the side of the lesion, is not an early symptom and does not depend on the size of the tumor. It is constant in growths of the corp. quadrigemina, is present in 90 per cent. of cerebellar growths and in 35 per cent. of those of the pons, medulla and corp. callosum. There is often limitation of the field of vision (scotoma for color). In many cases it develops into atrophy, oftener still into stasis and blindness, perhaps suddenly, while in some it may regress. Transitory *amaurosis* results from compression of the optic tract or chiasm by the distended third ventricle. (*c*) *Convulsions* occur in 50 per cent., are called the "signal symptoms" by Seguin and may be general; general tonic precede clonic convulsions in no special order; the pupils react

poorly, the skin reflexes are increased unless deep coma develops, the tendon reflexes are not suspended and respiration is labored. Convulsions are more frequently late than early. Slight seizures, like the *petit mal* of epilepsy, may occur. Jacksonian epilepsy belongs to the focal symptoms. (*d*) *Vomiting* is usually an early general symptom, indicating irritation of the vomiting centre (in the medulla it is a focal symptom). It is of the cerebral type, occurring without effort, nausea or pain, often on an empty stomach, as in the morning, or at once after eating, regardless of the kind of food ingested. Vomiting is due to increased intracranial pressure or meningeal irritation. It is rarely absent in growths of the cerebellum or medulla, is very frequent in disease of the corp. quadrigemina and occurs in 25 per cent. of tumors in the hemispheres. (*e*) *Sensorium.* The most common change is dulness, which often deepens into stupor and coma. Tumors affecting the association fibers and avoiding the motor centres may cause disturbance of intelligence, judgment or memory only; they are frequently discovered at autopsy in the frontal or temporosphenoidal lobes in subjects committed to asylums for psychoses or general paralysis. (*f*) *Vertigo* is an early symptom; it may be mere "giddiness" or it may affect the labyrinth, pons or cerebellum and be a sense of actual rotation. It is present in 31 per cent. and as a focal symptom when the middle cerebellar peduncle is implicated. (*g*) *Slowed pulse* results more often from indirect pressure on the vagus fibers behind and lateral to the hypoglossal nucleus. The vagus fibers become accustomed to pressure and the heart rate increases. It is often associated with yawning, hiccough, Cheyne-Stokes's breathing, glycosuria or polyuria.

2. Focal Symptoms.—Focal symptoms may be absent when the disease is limited to "silent" or "tolerant" areas, when the tumor does not destroy nerve structures and when vicarious compensation by other parts is possible. Focal symptoms of irritation or paralysis may result directly from actual lesion of a centre or its fibers, or indirectly from "distance pressure" upon them. They are most significant when no particular increase of intracranial tension exists.

(*a*) *Frontal Lobes.*—Early observers claimed that mental changes were no more frequent in frontal localization than in localization elsewhere. A dementia with characteristic irritability and excitement, a tendency to make jokes, apathy and suffering are described. Homolateral tremor and contralateral loss of the abdominal reflexes are described by Granger Stewart. Mental alteration sometimes follows other localizations. Tumors in the frontal lobes may be thought to be in the motor areas from indirect pressure upon the latter. The head and eyes may deviate and the neck and back are sometimes rigid; frontal tumors may induce cerebellar ataxia, caused by unequal innervation with disturbance of the sensorium or involvement of the trunk centres. Bradyphasia is probably psychical and differs from the separation of syllables sometimes seen in tumor of the pons. In "laryngeal aphasia" the patient moves his lips and attempts to speak, but finds the voice lacking.

(*b*) *Motor Cortex.*—(i) *Jacksonian convulsions* (50 per cent.) are often dissociated, and follow a given sequence (see page 751). The convulsions may run into the hundreds (17,000 in eleven months). When the con-

ducting fibers are destroyed they cannot occur, whence they may be observed in the part nearest the seat of greatest damage. As long as convulsions are present the motor centres cannot be wholly destroyed. Tapping the skull may initiate spasms. (ii) *Cortical paralysis* is usually monoplegic and associated with cortical spasm; at first a weakness from exhaustion follows the spasms; later it becomes more marked. Slowly progressive hemiplegia without other symptoms may suggest tumor (Williamson) and paresis may be the first sign of tumor, perhaps narrowly localized, as in the thumb alone, fingers or great toe; paralysis is accompanied by increased reflexes and rigidity. Surgical extirpation has relieved both spasms and paralysis. In infrequent instances the paralysis may be bilateral, as when a cortical tumor near the median line presses on the opposite hemisphere or, when there are bilateral symmetrical growths. (iii) Sensory changes are less frequent, because the sensory is larger than the motor area, extends posterior to it and possibly is bilateral. Hypesthesia, especially for tactile and muscular sensibility and paresthesia, are not rare.

(*c*) *Parietal Lobes.*—Sensory disturbance is fairly common. Incoordinate movements, ataxia, conjugate eye movements and sensory aphasia, hemianopsia, deafness and word-deafness are observed.

(*d*) *Temporal Lobes.*—Hemianopsia, disturbed eye movements toward the opposite side, ptosis and agraphia are recorded. Uncinate "fits" are characterized by subjective—and usually disagreeable—sensations of smell and taste, of which Mills collected 12 cases (1908).

(*e*) *Occipital Lobes.*—Hemianopsia is the most constant finding. Optical hallucinations indicate a cortical occipital localization. Alexia, optic aphasia and psychical blindness are seen.

(*f*) *Pons.*—Focal may precede the general symptoms, as enumerated under Cerebral Localization. *Hemiplegia with alternating paralysis* of some of the first eight cranial nerves is the most common type (50 per cent.). Foci in the substance of the pons involving the sixth nerve usually involve the opposite third nerve; foci near the surface involving the sixth nerve do not involve the third nerve. Sometimes the nerves may be involved without hemiplegia (25 per cent.), but rarely without crossed motor irritation or hemianesthesia; hemiplegia alone occurs in 25 per cent. Unilateral tumor may compress the opposite motor tracts (paraplegia or diplegia) and cranial nerves. Motor irritation is not uncommon, as trismus, facial spasm, trigeminal neuralgia, convulsions in the contralateral limbs, etc. *Sensory disturbance* is usual, as hemianesthesia cruciata (in arm and leg of one side and in the face of the opposite side when the focus is low in the pons), hemianesthesia of face, arm and leg of the same side (tumor in the upper pons) and anesthesia of one or perhaps of both sides. Dysarthria, dysphagia, difficult urination or defecation and disturbed respiration on change of posture have been observed.

(*g*) *Medulla.*—It is remarkable that tumors may produce no symptoms or only such that localization in the pons or cord is first suggested. In a small number of cases only are the symptoms not ambiguous. Bilateral paralysis of the ninth to twelfth nerves with degeneration and atrophy of the lips, tongue, palate, pharynx or larynx is characteristic and presents

a picture like progressive bulbar palsy. Alternating hemiplegia is rare. Sensory, motor, atactic and nerve involvements are usually bilateral. Stern (1907) collected 72 cases of cysticerci of the *fourth ventricle* and 50 of tumors, characterized by suboccipital pain, bowing of head forward, paroxysmal attacks of vomiting, headache and cervical rigidity. Growths *outside the medulla* may cause paralysis of the neck muscles. Slowed or rapid pulse, singultus, embarrassed breathing, glycosuria and polyuria are not uncommon.

(*h*) *Cerebellum.*—Cerebellar ataxia, referred by Nothnagel and Gowers to lesions of the vermis in 85 per cent. of the cases, occurs but half as frequently in disease of the cerebellar hemispheres. Compression of the pons, medulla, corpora quadrigemina and fifth to twelfth cranial nerves is somewhat characteristic. Extra-cerebellar tumors may, in the ponto-medullocerebellar angle, involve hearing and also the eighth, fifth or sixth nerves (disturbance of hearing, paralysis of winking and tardy corneal reflex). Of the general symptoms, choked disk is almost invariable; occipital headache and rigid neck are very frequent. Convulsions are common and opisthotonos with tetanic rigidity of the body and extremities is most suggestive.

(*i*) *Optic Thalamus.*—Moderate hemiplegia (transient and without contractures), hemichorea, hemiathetosis, hemianesthesia (always marked as to deep sensation), slight hemiataxia, marked astereognosis and persistent pains (*hemiplegie douloureux*) constitute the *thalamic syndrome* of Roussy (1907). Less common are Nothnagel's mimetic paralysis, unilateral convulsions, forced movements or postures, contralateral hemianopsia, paralysis of the eye muscles, widening of the opposite pupil and monoplegia.

(*j*) *Corpus Callosum.*—According to Bristowe, dementia (in 90 per cent.), absence of initiative in speech and action, paraplegia, dysarthria and few general symptoms, as choked disk, are characteristic. Oppenheim holds that there are no distinctive symptoms.

(*k*) *Corpora Quadrigemina.*—Paralyses of the third nerve are common, sometimes associated as double ptosis, and bilateral paralysis of the internal recti. The pupils are usually free. There may also be crossed hemiataxia, disturbed hearing, amblyopia and intention tremor. No paralysis, except of the third nerve, is present unless the tumor extends beyond the corp. quadrigemina (Oppenheim).

(*l*) *Pineal Gland.*—The symptoms are the same as when the corp. quadrigemina are affected, save that the fourth and sixth nerves are involved more than the third and compression of the pons or medulla is more common. There may be obesity or genital hypertrophy.

(*m*) *Tumors at the Base.*—These include osseous tumors, aneurysms, hypophysis growths and those from the nerves or meninges. (i) In the *anterior fossa*, hypophysis tumors (see ACROMEGALY) disturb vision and produce bitemporal hemianopsia with amblyopia or sometimes irregular or transverse hemianopsia. Usually there is no choked disk, but simple optic atrophy. The third nerve is paralyzed in most cases, sometimes other ocular trunks. Exophthalmos is occasional and the growth may perforate externally into the eye, pharynx or nose. Loss of

smell (anosmia) and mental symptoms are observed. (ii) In the *middle fossa*, mental symptoms and convulsions are less common. The Gasserian ganglion may be compressed and result in neuroparalytic ophthalmia. Other cerebral nerves, as the third and sixth, sometimes suffer. Syphilis and sarcomatosis may be diffusely basal. (iii) Lesions in the *posterior fossa* may simulate bulbar palsy. The lower eight cranial nerves usually suffer more readily and earlier than the pyramidal tracts; the grouping of the cranial nerve implication is different; thus paralysis of the sixth nerve is rarely associated with the conjugate fourth nerve, as occurs when the lesion is central. The seventh and eighth nerves are often conjointly paralyzed in disease of the posterior fossa and but rarely so in central tumors. The symptoms often resemble those of lesions of the pons, medulla, etc.

Diagnosis.—Four general groups of cases are encountered: (1) Absolute clinical latency; (2) general and focal symptoms, the typical case; (3) general but not focal symptoms; (4) definite symptoms of some brain disease but of equivocal significance. According to Bruns, 80 per cent. of cases can be detected.

Analysis of the General Symptoms.—(*a*) *Headache* is suggestive when it is severe and when the skull is tender to percussion. Organic, irritative brain disease, as abscess or inflammation, may cause headache. Headache may result from migraine, neurasthenia, hypochondriasis, eye disease, intoxications (uremia, gastro-intestinal disorder and poisoning by nicotine, opium, alcohol or lead), and from arteriosclerosis. (*b*) *Choked disk* in 85 per cent. of cases indicates brain tumor (Oppenheim) and if very marked it is seldom due to other causes. It is less frequent in abscess and meningitis, in which neuritis optica is more frequent. It is most rare in embolism, softening, hemorrhage, hematoma duræ matris, arteriosclerosis, poliencephalitis, encephalitis, the acute fevers, lead poisoning, neuritis multiplex or alcoholism. *Uremia* and tumor have, in common, headache, epileptiform or apoplectiform attacks, hemiplegia, aphasia and slow pulse, yet neuroretinitis is more common in nephritis. The rare choked disk of chlorosis responds to iron. (*c*) General epileptiform *convulsions* occur in meningitis, pachymeningitis, abscess, vascular disease, syphilis, alcoholism, plumbism, multiple sclerosis, paretic dementia, uremia, etc.

Epileptic Convulsions.	Convulsions resulting from organic brain disease.
More coarse; more biting of tongue; more frequent cry; more sudden, complete loss of consciousness, frequently an epigastric aura; usually longer interval between attacks.	Convulsions more orderly, slow, clonic, broken by tonic contractions; often with gradual local commencement, without loss of consciousness, and with paresis in the Jacksonian type.

(*d*) *Vomiting* is probably cerebral and due to tumor, if hysteria, migraine, toxemia and gastric disease can be excluded.

Combination of General Symptoms.—Headache and choked disk occur in chlorosis, plumbism (also with delirium, convulsions and coma,

but distinguished by the lead line on the gums) or in uremia, in which special examination for albumin, casts and cardiovascular changes are definitive. The same combination may prevail in hypermetropia in anemic girls, in amenorrhea and in injuries of the head, but care usually distinguishes them from the slow onset and focal signs of brain tumor.

Topical Diagnosis.—*Focal symptoms usually follow the general,* whereas, in apoplexy, for example, the focal hemiplegia is early. In tumor with hemiplegia, contracture or spasticity accompanies rather than follows the paralysis, as usually occurs in hemorrhage or embolism. Hemiplegia from a tumor is less pure, less circumscribed, less systematic (Fournier). A tumor at the base may cause hemiplegia from pressure on the middle cerebral artery with encephalomalacia. Jacksonian epilepsy with paresis is due to cortical tumors in 50 per cent. of cases. It is also produced by hemorrhage, softening and inflammation and is then earlier, is not confined to as small an area and is not as progressive as in neoplasm.

Nature of the Growth.—(*a*) *Location. Cortical* growths are probably gumma or tubercle (possibly glioma, cysts, cysticercus or sarcoma); *subcortical,* glioma or sarcoma; *pons,* tubercle, gumma, glioma; *cerebellum,* tubercle or glioma; *extracerebral,* sarcoma; *multiple occurrence,* tubercles or gummata. (*b*) *Course.* A rapidly growing tumor is probably glioma or sarcoma. An apoplectic attack indicates glioma or ruptured aneurysm. Growth with remissions is characteristic of tubercle or glioma. A very slow course suggests fibroma or osteoma. Coincident meningitis indicates tubercle or syphilis.

Differentiation.—*Brain abscess:* abscess and tumor may result from trauma, but brain abscess is almost always traumatic, otitic or metastatic; unilateral optic neuritis is more common than choked disk, and abscess is more prone to fever, chills, rapid course and apoplectic seizures than is tumor, while involvement of the cranial nerves is less frequent. Differentiation from paretic dementia, pachymeningitis, meningitis, syphilis, hysteria and hydrocephalus will be considered under each disease.

Multiple Sclerosis. The pressure symptoms of tumor are absent and in the disk there is an incomplete optic atrophy. The tremor may be confused with incoördination tremor of tumor in the optic thalamus, pons or cerebellum.

Course and Prognosis.—The first symptoms are usually general; the later are focal. The disease runs a fatal course in *one-half to two years,* in rare cases four to fourteen years. *Exacerbations* result from aneurysmal rupture, hemorrhage into a glioma, cyst formation, ventricular effusions, meningitis or local neuritis. *Remissions* are not uncommon; healing of parasitic and simple cysts, aneurysms, tubercles (in the young) and gummata is possible. *Unfavorable* symptoms are pronounced mental change, incoercible vomiting, irregular, rapid pulse, repeated fits, apoplectiform seizures or festinating neuritis optica. The *mechanism of death* is exhaustion from vomiting, pain or cachexia; inhalation pneumonia; coma with disturbed respiration, circulation or other bulbar signs;

sudden death in tumors of the base; meningitis or arterial disease; and intercurrent infections.

Treatment.—Medical treatment is not encouraging save in syphilis, but every patient should be given a long course of mercury and iodide. Tubercle is sometimes arrested by increasing the physiological resistance by forced feeding, fresh air, cod-liver oil and arsenic. The headache is often relieved by iodide, the ice-bag, morphine, atropine, coal tars and decompression operations. Convulsions should be treated by chloral. Brain congestion from alcoholism, excitement, exertion, overeating or obstipation should be avoided.

Surgical Interference.—Macewen, Horsley, Bennett, Ferrier and Godle are the pioneers in brain surgery. (*a*) In *radical operation* the outlook is not encouraging. Allan Starr estimates that but 18 per cent. of tumors are accessible, and but 7 per cent. removable. The best results are obtained in meningeal and cortical growths, especially of the motor and occipital zones. Duret found that 18 per cent. died, 64 per cent. improved and vision was completely restored in 60 and partially in 18 per cent. Recurrence is common. Death occurs from hemorrhage, sepsis, dislocation of the brain, basal compression, brain edema and shock. Horsley and Macewen suggested two steps; first exposing the tumor and later removing it after the brain had accommodated itself to the change of relations. (*b*) *Palliative decompression* may be beneficial, often greatly relieving headache and blindness and permitting the tumor to grow outward when it cannot be removed. From *lumbar puncture* 38 cases of sudden death are reported, the withdrawal of the supporting column of cerebrospinal fluid accentuating a hernia of the medulla into the foramen magnum.

INFLAMMATION OF THE BRAIN.

I. Encephalitis.—"Inflammation of the brain" as understood by the laity, is meningitis. Coswell and Hasse (1850) definitely distinguished between encephalomalacia and encephalitis.

Etiology.—The three main factors are: (*a*) *Intoxications*, from alcohol, illuminating gas, sulphuric acid, ptomaines in decayed meat, nicotine and sunstroke. (*b*) *Infections*, as influenza, scarlatina, hydrophobia, etc. Some relation to chlorosis, chorea insaniens or maniacal forms of exophthalmic goitre is possible. (*c*) *Trauma.*

Pathology.—The process begins as multiple disseminated foci, principally in the gray matter; these usually are the small "flea-bite" areas of capillary hemorrhage, which glisten and stain the adjacent tissues, show above the cut section and later become foci of softening. Microscopically, hyperemia, perivascular exudation of red and white cells, serous effusion, aggregations of mononuclear cells somewhat resembling a tubercle, and degenerated, fatty and swollen ganglion cells are observed; the medullary sheaths and later the axis-cylinders degenerate. Its issues are: (*a*) regression (recovery); (*b*) necrobiosis with softening; (*c*) cyst formation; (*d*) cicatrix formation or (*e*) possibly diffuse brain sclerosis

or multiple sclerosis. *Associated nervous changes* are optic neuritis, multiple neuritis and poliomyelitis, and *general changes* are enlarged spleen, general hemorrhages and parenchymatous degeneration, etc.

Special Pathology and Symptomatology.—1. Poliencephalitis Acuta Superior Hemorrhagica or Ophthalmoplegica.—Wernicke (1881) described this affection in *alcoholic* adults; it attacks the gray matter about the third and fourth ventricles and aqueduct of Sylvius, whence it may spread upward or downward; it is analogous to acute poliomyelitis. The *general symptoms* are delirium tremens, or somnolence in non-alcoholics, headache, vomiting and rigid neck. The *local symptoms* consist of an early extensive ophthalmoplegia, which involves associated eye muscles, as the two internal recti, or the internal rectus of one eye and the external of the other and usually evades the pupil and levator palpebræ superior. The gait is atactic from involvement of the corpora quadrigemina. Speech is frequently difficult. Neuritis is observed in the optic disk and sometimes in the peripheral nerves, as evidenced by the absent patellars, ataxia, tender muscles, etc. Sometimes there is hemiparesis or facial paresis. The temperature is usually normal or subnormal, though respiration and pulse are increased. Death occurs within two weeks after these violent symptoms. Some cases thought to be nuclear have been proved polyneuritic.

2. Encephalitis or Poliencephalitis Hemorrhagica.—Strümpell (1884) described cases causing infantile paralysis (*q. v.*). In 1885 Leichtenstern saw cases following certain infections. Foci in the cortex, subcortex or central ganglia, contained pus cocci and influenza bacilli. The disease occurs mostly between the fifteenth and thirtieth years. After an influenza (*q. v.*) there may be (*a*) *general symptoms*, as headache, vomiting, delirium, mental obscuration, convulsions, high fever, slow pulse, irregular breathing, stiff neck or optic neuritis; (*b*) *focal symptoms* appear after a few days, as hemi- or monoplegia, aphasia, conjugate deviation, focal convulsions or hemichorea. The reflexes are affected as in apoplexy. The course lasts one-half to three weeks and is often fatal. This form may resemble hemorrhage, syphilis or softening, but the paralysis is preceded by fever, enlarged spleen, somnolence or convulsions. Meningitis is usually differentiated with ease.

3. Subtypes.—These consist of the following: (*a*) *poliencephalitis inferior acuta*, involving the pons or medulla and giving acute bulbar symptoms; there is paralysis of the sixth or seventh to twelfth nerves when it occurs in various combinations or with types (1) and (2). Confusion is possible with softening, hemorrhage, the late hemorrhage of trauma or tumors. (*b*) With *poliencephalomyelitis*, inflammation and paralysis of the anterior spinal roots may coexist. Hutchinson's facies is observed, viz., fixed eyes, fallen lids, prominent eyebrows, obliterated nasolabial folds and atrophic paralysis of the arms. Differentiation from multiple neuritis is usually possible.

Prognosis.—The peracute cases may die in two days; acute cases last one-half to three weeks and death is most frequent; Oppenheim instances recoveries. Much involvement of the mind, decubitus, alcoholic delirium, chlorosis, sinus thrombosis and weak pulse are unfavorable elements.

In children recovery is common, but epilepsy, paralysis and mental alteration are sequences.

Treatment.—This is symptomatic. The causal factor must be eliminated, cerebral congestion avoided and the skin, bowels and bladder, delirium and convulsions, diet and patient's rest must be cared for as in brain hemorrhage. Mercurial inunctions and potassium iodide give the best results. Electricity is indicated in the chronic stage.

II. Abscess of the Brain.—Encephalitis apostematosa is not a common disease. Eichhorst found three among 13,563 medical cases.

General Etiology.—The streptococcus is the most frequent microorganism, the staphylococcus coming next, then the pneumococcus and other bacteria. Mixed infections are common; old abscesses are often sterile. Sixty-six to 75 per cent. occur in men. Twenty per cent. occur in the second and 33 per cent. in the third decade. The cerebrum is involved in 75 and the cerebellum in 25 per cent.

General Pathology.—The focus is single in 80 per cent. of cases. The average size is from one to two inches, but it may occupy nearly the entire hemisphere. The surrounding brain may be inflamed, or edematous. The right side of the brain is more often affected. Pressure is less common and less intense than in tumor; very large abscesses may produce internal hydrocephalus. The abscess consists of pus of variable consistency, yellow or green, acid; flocculi of disorganized brain tissue are common. The pus cells are often disintegrated and fatty needles, cholesterin crystals and sometimes corpora amylacea are found. Putridity is due to communication with the nose or ear or embolism from putrid lung foci. Encapsulation occurs in 50 per cent., chiefly in old cases, but rarely completely or permanently limits the abscess, which usually grows and often ruptures.

Special Etiology and Pathology.—1. Infective Foci near the Brain.—Infective foci near the brain are its usual causes, and for this reason brain abscess is seen by internalists, neurologists, surgeons and otologists. (*a*) *Ear disease* explains at least 25 per cent. of brain abscess. Insurance companies recognize the risk in these cases. Jansen found one abscess to 500 cases of chronic and one to 2650 of acute otitis. The ear disease may date back thirty or forty-five years and is especially dangerous when it recurs and when granulations and polypi exist. The acute infections thus indirectly cause brain abscess. Infection traverses the bone to the dura by way of the thin tegmentum tympani, by the roof of the antrum, sometimes through the petrosquamous suture (in children), mastoid cells, lateral sinus, labyrinth or aqueduct along the sheaths of the seventh and eighth nerves; in 42 per cent. there is a fistule between the abscess and the causal focus; in 15 per cent. the dura interposes between the two foci; in 15 per cent. there are dural adhesions and in 17 per cent. the intervening substance is softened brain tissue. Toynbee remarked that disease of the roof caused temporosphenoidal abscess, but disease of the mastoid cells chiefly produced cerebellar abscess. Of otitic abscess, temporosphenoidal constitutes 67 per cent. (75 per cent. under ten years) and cerebellar abscess 33 per cent.; the latter is rarely due to any cause other than otitis and almost invariably

occurs in the anterior part of its hemispheres. Otitic abscess infrequently involves the occipital, frontal (two personal observations) or parietal lobes. The smaller the abscess the nearer it lies to the cortex. (*b*) *Traumatism* produces 25 per cent. of cases. Punctured wounds are more dangerous than open, compound fractures. Fractures of the base (by infecting the middle ear), splintering of the inner table (areas becoming infected through the blood) and operations on the brain are possible causes; 80 per cent. occur over ten years of age. Ninety-three per cent. are solitary abscesses. Infection travels along the perivascular and perineural lymph sheaths but may pass "retrograde" by the valveless veins. (*c*) *Other contiguous disease* is rarer, causing but 4 per cent. of brain abscess. Disease of the nose and communicating sinuses may cause abscess of the frontal lobes and in antrum disease sometimes of the parietal lobes. Disease of the orbit, as phlegmon or penetrating wounds, and still more rarely cranial osteomyelitis, ulcerative tuberculosis or syphilis may cause abscess by contiguity.

2. Metastatic Abscesses.—Metastatic abscesses constitute 25 per cent. of brain abscess. The abscesses may number from 2 to over 100; they are multiple in 66 per cent. They are cortical or subcortical, less frequently in the central ganglia and almost never in the cerebellum alone. The causal embolus is seldom found. (*a*) *Lung lesions:* "pulmonal cerebral abscesses" are due to empyema, putrid bronchitis and bronchiectasis (45 per cent.), gangrene, abscess and sometimes tuberculous cavities. This form rarely encapsulates. (*b*) *Ulcerative endocarditis*, pyemia, osteomyelitis, etc., are less common causes; paradoxical embolism may occur through a patent foramen ovale, whereby the clot escapes the lungs.

Symptoms.—Symptoms are absent in 10 per cent. of cases.

1. Symptoms of Sepsis.—(*a*) *Fever* is the most important general symptom; Eichhorst describes cases closely resembling typhoid; it is present in acute cases and during the last stages when the abscess spreads or meningitis or sinus thrombosis develops. Macewen's cases showed normal or subnormal temperature. (*b*) Chills are not frequent. (*c*) The pulse is often accelerated, but may later become slowed to 30 or 40, the compression pulse.

2. Diffuse Brain Symptoms.—These are due to increased intracranial tension, inflammation, meningitis or internal hydrocephalus and include (*a*) *headache*, which is most common and severe, though less so than in brain tumor; even the stiff, painful neck and headache are general rather than focal symptoms. It is frequently associated with (*b*) *vomiting* and (*c*) *vertigo*. (*d*) *Mental symptoms* are those of slow cerebration. (*e*) *General convulsions* are frequent only in the last stages, in cases of extensive abscess or rupture into the ventricle. (*f*) The *pulse* is often slowed, even with coexistent fever, to 40 (even 10) and sometimes with slow or irregular respiration. (*g*) *Optic neuritis* is more common than choked disk; it occurs in 53 per cent. and may produce amaurosis and atrophy.

3. Focalizing Symptoms.—Focalizing symptoms are often absent and when present are less conspicuous than in tumor because they are

obscured by the primary affection or the general symptoms and occur in areas of less physiological dignity. They usually signalize advance of the abscess, softening or increased brain tension.

Frontal or mental symptoms may occur (see TUMOR), but foci as large as the fist may remain latent. They may impinge on the *motor cortex.* The symptoms are those of a cortical lesion, which extends more rapidly than tumor and is attended by Jacksonian fits, rigidity and increased tendon and decreased skin reflexes on the contralateral side; in subcortical lesions from traumatic and metastatic abscess, hemiparesis occurs in 50 per cent., is sometimes progressive, usually incomplete and at times is apoplectiform in onset (three personal observations). If hemiparesis is early it indicates involvement of the internal capsule, which also causes hemihypesthesia. Conjugate deviation is not common. *Temporosphenoidal* lesions are largely otitic, frequently latent and subcortical, whence the centres are less involved than the deeper association fibers. The more important left-sided lesions lead (in 42 per cent.) to *sensory aphasia,* partial word-deafness, alexia, agraphia, amnestic and optic aphasia. The right side concerns the memory for letters and figures. *Occipital* lesions or those deeper in Gratiolet's optic radiation, produce hemianopsia (more in traumatic or metastatic than in otitic abscess). *Cerebellar abscess* results almost exclusively from ear disease and is often latent. Occipital headache and stiff neck occur; there may be a tendency to fall forward or backward and hemiparesis or yawning may result from compression; cranial nerve paralyses are not frequent and result from coincident meningitis. Paralyses of the seventh and eighth nerves are often due to bone disease. Abscess of the pons, medulla (of which Cassier, in 1903, found only 16 cases) and crus is exceptional. (See CEREBRAL LOCALIZATION.)

SYMPTOMS BY STAGES.—(i) The *initial stage* is observed mostly in acute traumatic or metastatic abscess. Pus formation usually requires ten to thirty days. The causal septic and general brain symptoms appear rapidly; death may occur from coma, fever and meningitis or the next stages appear. (ii) The *latent stage* has no fever, is seen in chronic abscess, probably corresponds with encapsulation and lasts for months or even decades. (iii) In the "*manifest*" *stage* septic, general or focal brain symptoms appear continuously or remittently, with evidence of extension. Most cases are seen in this stage. Gowers estimates that after nervous symptoms once develop, (iv) *the next or terminal stage,* appears in five days (20 per cent.), in ten days (33 per cent.), fourteen days (50 per cent.) and in thirty days (75 per cent.).

Diagnosis.—Recognition is impossible in latent cases affecting "silent" areas, whence "no brain disease of equal severity so often escapes recognition." Few cases can be detected without consideration of the etiology, which is definitive in 75 per cent., the sepsis, general and focal brain symptoms.

Otitic abscess may be confused with the otitis itself, especially in children; otitis may produce headache, vomiting, convulsions, optic neuritis, nystagmus and diplopia, but all symptoms disappear under surgical care; otitic brain abscess runs an acute course; temperature

is often lower than in other forms, the headache may be local and edema may be seen over the temporal region, with tenderness on percussion and a higher percussion-note (Macewen).

Serous meningitis is often mistaken for the suppurative form and is very important in ear disease. It is attended by slight temperature, internal hydrocephalus, paralysis of the basal nerves and gives a serous fluid on lumbar puncture. *Extradural abscess* was found in 78 per cent. of cases of brain suppuration by Jansen, sinus thrombosis in 18 per cent. and brain abscess in barely 3 per cent. Local symptoms, as edema and tenderness, are commoner than diffuse and focal symptoms. *Sinus thrombosis* (*q. v.*) is more often attended by pyemic symptoms, foci in the lungs, choked disk and external signs of altered brain circulation.

Traumatic abscess is often suspected because of delayed healing of the wound. It is usually cortical (whence its focal symptoms) and often provokes fever and other general symptoms. It must be differentiated from traumatic hemorrhage, which occurs days or weeks after the injury and often in the corp. quadrigemina, pons or medulla; from acute encephalitis, whose course is more rapid; from concussion, in which the symptoms are more diffuse and may involve the brain nerves or arouse fever, though cortical symptoms are absent. The *late traumatic abscess* is subcortical (affecting the conduction or association fibers). *Cerebral tumor* (*q. v.*).

Abscess by metastasis; localization is largely in the hemispheres, thus obeying the topography of embolism. The Sylvian district and occipital lobes are most often affected and the multiplicity of localization should be remembered. The course is usually precipitate.

Prognosis.—*Spontaneous recovery* by demarcation, inspissation, calcification or external rupture is very unusual. Without operation, *death* is practically inevitable, from (*a*) rupture into the lateral ventricles, chiefly in metastatic and otitic abscess and into the membranes (cerebellar abscess), (*b*) hydrocephalus internus, (*c*) sinus thrombosis, (*d*) pressure on the medulla, or (*e*) brain edema. Sudden death may occur without adequate postmortem explanation.

Treatment.—(*a*) *Prophylactic* therapy concerns the etiological factor. Adequate drainage is always necessary, although granulations in the middle ear are protective and should not be curetted in any routine manner (precisely as uterine curettage is discountenanced in puerperal sepsis). (*b*) *Palliative* treatment is that of brain tumor and meningitis. Special care regarding vomiting is indicated, lest it rupture the abscess. (*c*) *Surgical* intervention. Successful cases were those of Morand 1868, Roux 1848, and Schede 1866. The contra-indications are metastatic abscess, a fatal etiological disease, ventricular rupture and diffuse meningitis, although in the last 2 cases isolated recoveries are recorded. Sinus thrombosis, circumscribed meningitis and incipient pyemia do not contra-indicate operation. In Körner's otitic abscesses, 51.6 per cent. recovered, Wheeler's series gave 87.5 per cent. and Macewen's 96 per cent.

III. Dementia Paralytica.—It is also known as paretic dementia, progressive paralysis of the insane and among the laity as softening of the

brain. Bayle, 1822, and Calmeil, 1826, first accurately described the affection. It is so important in every-day practice and constitutes such a large proportion (10 to 25 per cent.) of all insanities that its description is in place.

Definition.—Dementia paralytica is a chronic inflammation or degeneration of the brain, characterized by progressive vasomotor, motor and psychical disturbances.

Etiology.—(*a*) Syphilis is the cause, Noguchi and Moore finding the spirochete in 25 per cent. of cases; dementia is syphilis and not a parasyphilitic affection. (*b*) Other causes are *modern life*, with its strenuousness, competition, hurry, worry, short sleep, dissipation, overwork and city life; Krafft-Ebbing summarized the etiology as "syphilization and civilization." These causes may coöperate with heredity and alcoholism. Generally speaking, the higher classes and *certain occupations* (officers, artists and actors) are more subject to it. Krafft-Ebbing found no clergymen among his 2000 patients. It occurs between the *thirtieth and fiftieth years*, and is 7 to 10 times as frequent *in men* as in women.

Pathogenesis.—Lues probably produces (*a*) vasomotor paresis in the forebrain, followed by (*b*) lymph stasis in the cortex and pia, (*c*) degeneration and atrophy of the brain and cord, and (*d*) interstitial inflammation. All cases end in brain atrophy but some regard chronic meningo-encephalitis as primary, and others hold that it is secondary to encephalitis parenchymatosa.

Pathology.—(*a*) The calvarium is thickened in 33 per cent. (*b*) The dura in 50 per cent. is thick, opaque, vascular and adherent to the skull (*pachymeningitis*, which is often hemorrhagic). In attempting to strip off the pia it is found adherent to the brain cortex (in 80 per cent.), especially over the frontal and motor areas, supplied by the internal carotid artery. (*c*) *Gross brain changes* are constant. The brain in the early stages is swollen and hyperemic, in the later stages, *wasted;* it may weigh but a third or quarter of the normal (1260 gm.); atrophy affects largely the zones supplied by the internal carotid artery, especially the frontomotor cortex and island of Reil. The gray matter is yellowish-gray, soft and wasted, but the atrophied white substance is firm. The *convolutions* are *greatly narrowed* and the sulci are widened, the degree of atrophy corresponding to the 6 or 8 ounces of serum which fills the deficit (hydrops ex vacuo). In 80 per cent. granulations are seen on the ependyma of the lateral ventricles, resembling the back of a "cat's tongue," and the ventricles contain hydrops ex vacuo. (*d*) The *minute alterations* in the brain are as follows: the ganglion cells are early swollen and cloudy; the later findings are vacuolated and atrophied ganglion cells; *wasting of the medullary sheaths* and axis-cylinders, especially the *association* or tangential nerve fibers running parallel to the cortex which waste before any inflammation can be seen; corpora amylacea represent what is left of them; the same changes occur in the cranial nerves; the bloodvessels show mural proliferation, *exudation*, colloid or hyaline metamorphosis, thickening, atrophy and obliteration; the lymph vessels show proliferation, transudation and compression of the cortex, where cystic degeneration occurs from snaring off of lymph vessels; the neuroglia

increases even sevenfold (sclerosis). (*e*) Alteration of the *spinal cord* is present in 90 per cent.; the lateral columns often show descending *degeneration*, but more commonly changes *like those of tabes* (*q. v.*); the cerebrospinal fluid contains globulin and mononuclear cells and exhibits the Wassermann reaction.

General Symptoms.—As a rule, the symptoms are vasomotor, psychical and motor, in the order of their usually slow development. The leading symptoms are referable to alteration in the frontomotor cortex. (1) In the *prodromal stage*, the first symptoms are equivocal or point to (*a*) *neurasthenia* because of the "irritable weakness," ready fatigue in mental processes, headache, cephalic pressure, introspection and "rheumatic" pains. Again, the initial symptoms are (*b*) *congestive*, as headache, migraine or attacks similar to *petit mal*. (*c*) Most important are *mental changes*. The patient is "not himself" but "he does not remark his own alteration" (Schule). "The patient is confused as to time, and comes too early to work or remains after time; he confuses places and may smoke in church; his observation is faulty as well as his judgment; he confuses dreams with actualities and he forgets entire epochs, especially recent impressions. Esthetic and social lapses are common; the subject becomes vulgar, careless, neglects duties, disregards amenities and from lack of will-power may steal or dissipate most openly. Emotional outbreaks occur, as violent or brutal conduct or maudlin sentiment over soon-forgotten incidents" (Krafft-Ebbing). The patient is often perniciously active. (*d*) Focal symptoms, especially *amnestic aphasia*, may appear. (*e*) General findings are noted after a time, as small and unequal pupils or other tabes-like findings, double vision, fibrillary contractions of the tongue, tremor, pains, headache, vertigo, congestive attacks, slow pulse, unmodulated voice, skipping of words in reading and incorrect writing. This stage covers months or years. In rare instances the symptoms follow tabes, "the ascending form of general paralysis." (2) At the *height* of the disease, (*a*) the psychical symptoms take various types, as maniacal exaltation, melancholia or simple dementia. (*b*) Congestive, apoplectiform attacks are frequent and immobile and unequal pupils, hesitant speech with elision of syllables, altered expression, trembling or paresis of the face, incoordination of the hands and gait, disturbances in writing and reading become more distinct. (3) *The terminal stage* is characterized by total dementia (the final type of all initial varieties), complete disorder of speech, incoördination so extreme as to necessitate feeding the patient and keeping him in bed, sensory, vasomotor and trophic complications and death from pyelonephritis, bed-sores or bulbar symptoms.

Symptoms in Detail.—1. Psychical Symptoms.—The weakness, *alteration in character*, loss of memory and reason and the ethical, esthetic and moral deficit have been described. During the height of the disease there is one of three main mental manifestations: (*a*) *megalomania*, which classical expansive type occurs in 66 per cent., associated anatomically with the characteristic meningo-encephalitis, and characterized by ideas of grandeur, the patient believing he is a king, God, a millionaire, an athlete, etc. He makes ridiculously large plans, buys extravagantly, lacks will-power, indulges himself excessively, changes with great "facil-

ity" from one thing to another, is optimistic, friendly, even philanthropic and rapidly runs through his capital. He loses himself, forgets names and confuses the real with the unreal. Periods of acute mania, with fever and grinding of the teeth are expressions of brain congestion; complete dementia is the final outcome, though remissions occur. (*b*) The *melancholic* or hypochondriacal form is thought to occur with cystic degeneration of the cortex. It may be of the dull or agitated type. The patient may dwell on the condition of his bowels and think they would "burst and flood the world." There is often *micromania*, characterized by "self-belittlement," in which the individual fears everything and thinks himself a "dot, nothing, or denies his existence." These cases may commit suicide. (*c*) *Simple primary dementia* may be present from the beginning; this type is increasing in frequency, while megalomania is decreasing. It is anatomically simple cortical atrophy in which inflammation is absent or late. These subjects are often deceptive in the early stage, being quiet, urbane and optimistic. In the ultimate dementia of all types, "there is no sense of time, place or identity," and the patient babbles or is speechless.

2. Motor Disorder.—This usually occurs with mental changes, is constant, though variable in its expression, incomplete in degree, extensive as to parts involved and progressive. (*a*) *Speech* is pathognomonically disturbed as a result of mental change, wasting of association fibers and later bulbar atrophy. It is incoördinate, involving letters (*"literal" ataxia*) or syllables. In pronouncing such words as "electricity" the patient characteristically stutters over syllables. Loss of word-memory is the primary trouble; the patient forgets expressions, speaks ungrammatically and "does not notice it." Speech becomes atactic, there is paraphasia, the voice is unmodulated, gives out suddenly, is sometimes hoarse, nasal or slow, but never scanning. Later, speech is disturbed from bulbar alteration, causing dysarthria from disease of the facial and hypoglossal nuclei; it is ultimately unintelligible (absolute aphasia). Closely associated with speech disturbance is (*b*) *disordered reading* (paralexia) (*c*) *Writing* is disturbed first because of the mental alteration; the patient has difficulty in collecting his thoughts, although he does not realize the cause (the ataxia by which he drops syllables, repeats words or sentences—*paragraphia*); and it is disturbed secondly because of tremor. The writing is uneven, up and down or zig-zag. These changes are diagnostic. (*d*) *Paralysis of the eye muscles* (in 6 per cent.) is usually partial and transitory. When total, syphilis or tabes is the probable cause. (*e*) The *pupils:* Myosis may occur, often just before maniacal outbursts. Mydriasis is frequently unilateral and results from sympathetic irritation. In 60 per cent. of cases the pupils are now large, now small, which is considered especially sinister. They are often irregular or triangular. The Argyll-Robertson pupil, responding to accommodation but not to light, is marked in 47 per cent., partial in 20 per cent. and absent in 33 per cent.; it is due to degeneration in Bechterew's column (between that of Goll and of Burdach). (*f*) The *facial nerve:* The paresis of the lips is cortical, as are fibrillary twitchings, automatic chewing movements and almost constant grinding of the teeth.

The "fatuous" or "masked" expression is psychical. Salivation results from cortical irritation. There may be difficulty in swallowing. (*g*) *The limbs:* (i) The cortical changes cause the small-waved, rapid, constant tremor, the ataxia, loss of muscle sense and the uncertain, awkward, tripping paralytic gait. (ii) The gait may be tabetic, with absent knee-jerks, immobile pupils and vesical symptoms; or it is more often spastic with early increased patellars, ankle-clonus, contractures and arthropathies. (iii) Neuritic atrophy of the cranial nerves may occur as in tabes. (*h*) *Apoplectiform and epileptiform attacks.* (i) Apoplectiform seizures result from vasomotor paralysis and brain edema and differ from apoplexy in their incompleteness, evanescence, increased temperature and increased reflexes. They last part of an hour and may produce temporary hemiplegia, but especially aphasia with paresis of the right arm. (ii) Epileptiform attacks occur later, result directly from cortical disease, are usually partial (involving the face or arm), and either Jacksonian or attended by partial loss of consciousness.

3. Sensation and Special Senses.—Coincident tabes and pachymeningitis may emphasize the sensory manifestations, as lancinating pains. Sensation is often dulled and, like the analgesia, is cortical in origin. Perhaps the hypochondriacal complaint that certain organs, as the bowels, are absent, is due to visceral anesthesia. Migraine, first appearing late in life, may indicate an organic origin; Charcot described a form with hemianopsia and symptoms like glaucoma. Migraine is cortical when associated with paresis of the face, tongue or arm. There may develop optic neuritis and atrophy (4 per cent.), optic hallucinations and psychical blindness.

4. Other Symptoms.—(*a*) *Vasomotor* changes occur in the skin (lividity, edema and sweating) and in the brain, lungs, bladder, intestines and stomach (resembling the so-called bilious attacks). (*b*) *Trophic* symptoms are sweating of blood, rough skin, phosphaturia, perforating ulcer of the foot (3 per cent.), bed-sores and friability of the bones, which may be broken without the patient knowing it. The "insane ear" is frequent.

5. General Symptoms.—The pulse is often slow and monocrotic, the temperature low, although the heat centres may be involved in congestive seizures with great febrile elevation, and the body weight is often reduced in the early and late stages, though at the height of the disease it often increases, especially before the congestive seizures. The sexual instinct, at first increased and sometimes perverted, later declines. Insomnia may be severe, protracted or absolute.

Diagnosis.—The diagnosis depends upon (*a*) the *etiology,* as syphilis with mental strain or excesses; (*b*) the grouping of the *psychical symptoms,* as character, esthetic or ethical changes with (*c*) *motor* symptoms, as the speech, writing or tremor; (*d*) *vasomotor* symptoms, as migraine or apoplectiform insults, and (*e*) the *progressive course.*

The Wassermann test (*v.* page 219) of the blood is positive in 80 to 98 per cent.; the spinal fluid shows lymphocytosis, globulin and the Wassermann reaction.

Differentiation.—1. Diffuse Brain Disease.—(*a*) Cerebral *syphilis* may be confused with diffuse meningo-encephalitis. (*b*) *Senile dementia* seldom occurs under sixty years of age. Its course is longer, the symptoms more gradual and less intense, memory is maintained longer and there is less megalomania than fear of persecution and poisoning. (*c*) *Multiple sclerosis* occurs earlier, is complicated by late and mild mental symptoms and an intention tremor, which is wider in amplitude and ceases during repose. The speech is scanning, staccato, and there is no literal ataxia. Nystagmus is common, while it is rare in general paralysis. Motor symptoms are largely referable to lateral column disease.

2. Focal Brain Disease.—(*a*) Although migraine, headache, apoplectiform attacks and sensory or motor irritation of the Jacksonian type may occur in *brain tumor*, it does not cause difficulty in pronouncing syllables, peculiar mental alteration or immobile pupils; it is characterized by general symptoms, as choked disk or vomiting and focal symptoms, which are foreign to general paralysis. In tumor there is dementia only with marked internal hydrocephalus. (*b*) Hemorrhagic pachymeningitis complicates 20 per cent. of cases of dementia paralytica; when it exists as an independent affection, it often leads to intense local headache, vomiting, recurrent hemiplegia or monoplegia, cortical irritation and more rapid course. (*c*) Softening usually produces focal symptoms.

3. Psychoses and Neuroses.—(*a*) Every case of *mania or hypochondriasis* between the twenty-fifth and forty-fifth years, not due to alcoholism or to acute somatic disease should arouse suspicion of general paralysis (Mendel). (*b*) In *neurasthenia* the onset is more acute; the memory is fatigued but never lost and the patient recognizes his incapacity. In dementia paralytica there are hypochondriasis with absurd conceptions, changes in the pupils, disk and field of vision, alterations in character, speech and intellect which are never seen in neurasthenia.

4. Intoxications.—These may cause the forms known as general pseudoparalysis. (*a*) Chronic *alcoholism* is not progressive, recovery is frequent, delirium tremens and convulsions are common, the course is acute and the alcoholic tremor, headache, hallucinations and neuritis are found. (*b*) Chronic *plumbism* (saturnine encephalopathy) is often progressive, and in its last stages almost indistinguishable from dementia paralytica. It is more acute and 80 per cent. of cases recover. There are headache, cardiac distress, anemia and a gingival lead line. (*c*) Chronic *bromism* is distinguished by its dementia, bromide acne, fetid breath, coated tongue, pharyngeal anesthesia, weak heart and acute and usually favorable course.

Prognosis.—The disease is fatal in 66 per cent. of cases within two years, though remissions may occur. Krafft-Ebbing saw no recoveries in 2500 cases; recovery indicates an incorrect diagnosis, pseudoparalysis generalis. The following courses are distinguished: (*a*) *acute* or galloping form, which lasts months to a year; (*b*) *classical*, expansive type, three or four years; (*c*) the depressive type, two or three years; (*d*) dementia forms, four or five years; (*e*) circular form, in which depression alternates with mania; (*f*) female variety which has a longer course than in males;

(*g*) spinal form, with a relatively long course; (*h*) juvenile or adolescent; and (*i*) senile form. Death results in 50 per cent. from the disease itself, by apoplectiform seizures, suicide, inanition or bulbar symptoms. In 50 per cent. it results from complications, tuberculosis (18 per cent.), bed-sores, with sepsis (10 per cent.), lung gangrene (6 per cent.), pneumonia (5 per cent.), choking (10 per cent.) and cystitis.

Treatment.—*Hygienic* treatment, as rest, protection from heat, and quiet surroundings, is indicated. The patient should be committed to an asylum, for suicide or other violence is always possible. In simple dementia alone, may the patient be treated at home. *Hydrotherapy* is valuable within limits; cold rubs lessen excitation; full cold baths are contra-indicated. *Medicinal* treatment (*v.* Syphilis).

CHRONIC BULBAR PARALYSIS.

First thoroughly described by Duchenne (1860), as labioglossopharyngeal (laryngeal) paralysis, Trousseau made the first autopsies, Charcot and v. Leyden demonstrated alteration in the bulbar nuclei.

Etiology.—Most cases occur *in males* between *fifty and seventy years* of age. Heredity is a factor only when the disease is associated with progressive muscular atrophy.

Pathology.—To the naked eye the medulla usually appears normal. Microscopically, (*a*) the *motor nuclei* are symmetrically and bilaterally wasted. The hypoglossal nucleus suffers most and few normal cells remain; its accessory nucleus is usually normal. The spinal accessory nucleus is next most frequently affected, but the vagus nucleus suffers less. Atrophy may invade the glossopharyngeal or exceptionally the facial and fifth nuclei. In some cases perivascular exudation occurs within the nuclei and the nerve elements are degenerated (the parenchymatous form), but in others increase of connective-tissue or vascular thickening may be seen. (*b*) The respective *nerve trunks* in their course or in their terminal endings show parenchymatous and interstitial change, even within the medulla, and the posterior longitudinal fibers waste in direct ratio to the hypoglossal nuclear atrophy, especially those which form the anterior fundamental fibers of the spinal cord. Sometimes the loop of the seventh nerve wastes. (*c*) The *muscles* supplied by the above nerves waste, as those of the lips, tongue, palate and larynx (the process sometimes extends to the neck and arms). The greatest change is in the tip of the tongue. The changes are those of progressive muscular atrophy (*q. v.*). (*d*) In all cases *pyramidal tract degeneration* may be traced down into the cord or up into the crus.

Symptoms.—Labioglossopharyngeal paralysis, the name proposed by Duchenne, designates the parts paralyzed by the wasting of the motor nuclei. It is bilateral, but one case showing unilateral disease. It is slow in onset, afebrile and painless. The first symptom is usually *paresis of the tongue* (hypoglossus) which first shows fatigue and much later actual paralysis. It is difficult to protrude or move up, down or transversely. The linguals, L, N, R and S are formed with difficulty and also

later the lingual palatines, as T and D. The tongue is wrinkled and wasted. The *lips* become weak and whistling and formation of the labials (O, U, P, B, M) become impossible. The tongue and lips are more closely associated than any other muscles of the body; anatomically the facial fibers for the orbicularis oris probably originate in the hypoglossus nucleus. Later the mouth cannot be closed, the lower lip sags and the zygomatic muscles overcome the weak fibres of the upper lip and accentuate the nasolabial furrow. The lips are frequently thin. The expression is peculiar, the forehead is corrugated, the eyebrows somewhat lifted, but the mouth is "dead" and laughing and emotional displays produce a strange effect, which Trousseau compared to the mask of the Greek actors. The original descriptions picture the patient constantly holding a handkerchief before the lips to collect the free flow of saliva from the open mouth; the salivary secretion is probably also increased from disease of its bulbar centre. *Swallowing becomes difficult* from the inability of the tongue to propel the food to the pharynx so that the subject must push the food back with the finger, and again the palate (controlled by the spinal accessory and vagus nerves) is weakened, producing a nasal voice and allowing food to regurgitate into the nose. The *pharynx is paretic* (glossopharyngeal and vagus nerves). Semisolid food can best be swallowed, because fluid more easily regurgitates into the nose and solids more easily reach the larynx. The *larynx* is often involved (spinal accessory and vagus nerves). The adductors are more often affected than the abductors. Talking and coughing are difficult or impossible. *Fibrillary twitchings* are very common, sensation is normal, but the reflexes of the skin and mucosa are usually lost. There is sometimes a partial reaction of degeneration. The intellect is normal. Forced laughing and weeping are not uncommon. The general nutrition suffers from the dysphagia. In some cases the *eyes* may be involved, as in progressive nuclear ophthalmoplegia (an identical process in the pons); occasionally the cervical, facial, masseteric, temporal and pterygoid muscles are involved. Bulbar paralysis, frequently coexists with progressive muscular atrophy of spinal origin, affecting the limbs or trunk, and Kussmaul pointed out that the two processes are identical in nature. The spinal disease may progress upward to the medulla, causing secondary bulbar palsy, or the bulbar palsy may be primary. In typical forms, there are wasting, paralysis and absence of the reflexes; in some atypical forms there is no wasting; at times the reflexes are increased (clonus in the muscles of mastication), probably from a supranuclear lesion, "the bulbar analogue of amyotrophic lateral sclerosis of the cord."

In the *progressive course* of the malady the pulse becomes rapid and irregular and the heart tones run together, which Duchenne compared to the "heart beating in water." Dyspnea develops and the expiration is weak.

Diagnosis.—This is based on (*a*) the distribution of the process in the motor nuclei; (*b*) its bilateral symmetry; (*c*) gradual onset, and (*d*) chronic progression. *Neuritis* of the bulbar nerves is very rare; the lips escape, because the nerve fibers to the orbicularis oris, arising in or near the hypoglossus nuclei run to the pons and leave it by the

facial nerve fibers; there is more atrophy, more rapid course, more sensory disturbance and the reaction of degeneration is present. It occurs in acute infections. *Brain tumor within the medulla,* growths *without the medulla* and multiple sclerosis and syringomyelia rarely produce bilateral or symmetrical paralyses. *Pseudobulbar paralysis;* bilateral disease, more often in or near the internal capsule, especially when due to arterial lesions, may cause dysarthria (Joffroy, 1872). One lesion may be in the cortex and the other in the lenticulostriate area. Pseudobulbar palsy causes mental change, aphasia, hemiplegia, double facial paralysis and hemianopsia; one side is involved after the other and it is attended by no wasting, no loss of reflex action and no reaction of degeneration.

Prognosis.—The disease is fatal in one to three years, especially in advanced life or with coexistent spinal muscular atrophy. In the rarest instances the disease may be arrested. Death results from inanition, inhalation pneumonia, intercurrent disease or paralysis of the cardiac or respiratory centre.

Treatment.—Treatment is discouraging. Nutrition must be maintained by feeding with the nasal catheter. Iron and strychnine may be given. Electrotherapy is of little value.

Asthenic Bulbar Palsy; Myasthenia Gravis.—Chronic bulbar palsy *without anatomical lesion* was first described by Wilkes (1877), Erb (1879), Goldflam (1891) and Oppenheim. It is also known as the Erb-Goldflam syndrome. Starr collected 250 cases. It most often occurs in person's under the thirtieth year. The cardinal feature is *extreme exhaustion (myasthenia) after the slightest exertion.* Weakness develops in the eyelids, face, palate, masseters, pharynx, vocal cords and tongue, although but slightly expressed by ptosis (the first symptom in 33 per cent. of cases), dysphagia, dysarthria and difficult mastication, and disappears after rest, to return anew after a few muscular efforts. Myasthenia may be confined to the eyes. From these bulbar symptoms the affection is called *asthenic bulbar paralysis.* In some cases the arms, legs and trunk are similarly involved, so that walking or arm movements become impossible after short exertion—*myasthenia gravis pseudoparalytica.* The symptoms are symmetrical. The *myasthenic reaction of Jolly* consists of normal tetanic response to faradic stimulation, becoming weaker with each repetition, until at last no reaction is elicited. The reflexes soon become exhausted. Some atonicity of the digestive tract is common. Collins noted a rapid exhaustion of the special senses and Buzzard re-remarked sensory alterations. Remissions are usual, sixteen years being the maximum clinical duration. *About 40 per cent. of cases die* from exhaustion of the cardiac and respiratory centres but the *autopsy* findings are *negative* (in 66 per cent.) or doubtful; Kalisher found hemorrhages in the medulla, and C. Mayer found vascular and nuclear alterations. Lymphoid cells were found in the heart and between the muscle bundles (Weigert, 1901), and Weigert, Hun and Blumer describe lymphoid and glandular hyperplasia in the thymus gland. The *diagnosis* is based on (*a*) absence of atrophy, of the reaction of degeneration and of twitchings and sensory phenomena; (*b*) the presence of muscular adynamia

recurring remittently; (*c*) Jolly's myasthenic reaction and (*d*) the slight involvement of the hypoglossus.

Treatment.—Rest and massage are helpful; iodides, calcium, adrenalin and strychnine are less useful. Galvanization is recommended by Goldflam, who reported recovery under its use.

Apoplectiform Bulbar Paralysis.—Sudden bulbar paralysis deserves discussion in this place for the sake of topical differentiation. Its usual *causes* are hemorrhage, embolism, thrombosis and less frequently bulbar encephalitis or neuritic trauma.

Symptoms.—(*a*) The *onset* is apoplectiform with symptoms which are rarely focal, as vertigo, vomiting, dyspnea, yawning, slowed heart, and often without coma. (*b*) *Motor symptoms* are frequent, as *double hemiplegia;* monoplegia is possible, or if the lesion occurs at the pyramidal decussation there may be hemiplegia cruciata, *i. e.*, paralysis of one arm and the opposite leg. A unilateral focus may produce bilateral symptoms rarely, however, symmetrical. The pyramidal tracts in the medulla are supplied by the inferior cerebellar artery. (*c*) *Sensory symptoms* may develop, as hemiataxia or hemianesthesia (*d*) The *bulbar nerves* may be affected; paralysis of the tongue, pharynx, masseters, larynx and, because of the vascular supply, frequently of the eyes, face and other pons centres may result. The reflexes of these parts are often suspended, though in the extremities they are increased. The lesion may be at the level of the nuclei or supranuclear. The hypoglossal and spinal accessory nerves are supplied by the anterior spinal and vertebral arteries; the vagus, glossopharyngeal and auditory nerves by the vertebral; the fifth, seventh and eye nerves (third, fourth and sixth) by the basilar artery. There may be hemiplegia with paralysis of the opposite side of the tongue if the lesion is in the anterior part of the medulla.

Diagnosis.—*Chronic bulbar palsy* is excluded by the acuity of onset and the asymmetry and irregularity of the bulbar symptoms, as double hemiplegia, which is unequal on the two sides, or dysphagia without paralysis of the tongue or lip. Differentiation between hemorrhage, embolism and thrombosis is frequently impracticable. *Pseudobulbar paralysis of cerebral origin* (*v. s.*).

Prognosis and Treatment.—The *prognosis* is most grave at the onset. Improvement and even recovery are possible. *Treatment* concerns chiefly the underlying vascular disease.

Progressive Nuclear Ophthalmoplegia.—An atrophy of the oculomotor nuclei. (*v.* Affections of the Third Nerve.)

HYDROCEPHALUS.

Acute hydrocephalus is an acute accumulation of fluid between the dura and the brain (acute external hydrocephalus) or in the ventricles (acute internal hydrocephalus). In some cases the fluid is a *transudate*, being part of a general edema, resulting from cardiac or renal disease, or due to local causes, as sinus thrombosis. In most cases it is an *exudate*. Acute meningitis, the only certain cause, is tuberculous in 80 per cent.;

it is less often serous meningitis. Quincke thinks some cases are angio-neurotic.

I. Chronic External Hydrocephalus.—Fluid between the dura and brain is caused by (*a*) compensatory *hydrops ex vacuo*, occurring where brain development is arrested (congenital form) or when the brain wastes (paretic dementia, softening, hemorrhage, senile atrophy, porencephaly). (*b*) *Stasis*, as in sinus thrombosis; and (*c*) *chronic diseases*, as cancer, nephritis or alcoholism. The symptoms are indeterminate.

II. Chronic Congenital Internal Hydrocephalus.—Etiology.—Possible causes are fetal meningitis, obstruction of the choroid plexus, branches of the vena Galeni, lymphatics, lateral apertures of the fourth ventricle near the glossopharyngeal roots, aqueduct of Sylvius or in the foramen of Magendie or Luschka.

Symptoms and Pathology.—The large head may be an obstacle to labor. It may measure 167 cm. in circumference. If it appears after the seventh year the head is not enlarged, and in cretins the head may be undersized. Often the head cannot be held upright. The skull is even membranous and translucent to the candle test. If the disease halts, the bones may thicken and Wormian bones develop. The head projects beyond the base, the sphenoid is luxated forward and downward, the ear meatus points downward, the occiput is forced back, the sella turcica is flattened and widened, the roof of the orbit may be pushed down so as to become palpable, the fontanelles are large, the fissures gape and fluctuation may be felt. The hair is thin, the veins are large and a systolic murmur may be heard, the cause of which is doubtful. The *brain is pale* and may be flattened almost beyond recognition. The ependyma are thick, granular and sclerotic. The average amount of fluid is 1 quart, the maximum 13 quarts. The ventricles, especially the lateral, are enlarged; the enlarged third ventricle easily compresses the optic chiasm. The foramen of Monroe is often very large. The fluid is usually clear, and rarely blood-stained. It is neutral or alkaline, its specific gravity is 1.001 to 1.009; and the albumin is 0.2 to 2 per mille; more than 1.009 or 2.5 per mille of albumin indicates inflammation.

The *face is small*, triangular, and the eyes are prominent. Much of the sclera is shown and the upper lid is small. The brain may be reduced to a small fraction of an inch in thickness and is a mere flask for the fluid. The demarcation between gray and white matter is lost and in marked cases the convolutions and sulci are obliterated, the basal ganglia flattened, the cerebellum forced into the spinal canal and many structures, such as the fornix or corpus callosum have disappeared. *Mentality*, as a rule, is impaired or abolished, speaking is learned late if at all, and irritability, epileptiform attacks, psychoses, headache and vomiting are fairly common. Eye symptoms are frequent, as diminished vision or blindness from optic atrophy, strabismus, nystagmus and wide, possibly reactionless, pupils. The limbs often present spasticity, contractures, paresis and increased reflexes. The child walks late, if at all. Sensation is normal. Puberty may be delayed or the infantile testes may persist. Lumbar puncture shows increased intraspinal pressure (36 mm. of mercury).

Complications.—Complications are largely anomalies of development, as syringomyelia, hydromyelia, meningocele, microcephaly, porencephaly, anencephaly and encephalocele.

Course.—Some subjects die during or after birth. Many die in the first three years from increase of brain pressure with coma or intercurrent disease. In some cases temporary remissions, and in still rarer instances permanent arrest of the process occurs, the mind improves, the speech shows some defects or the gait remains paretic and spastic. In very exceptional cases rupture externally is observed after head trauma or spontaneous breaking into the ear or nose occurs, as in Lebert's case, which "leaked" for five years. Gölis reported a case which lived seventy-one years.

III. Acquired Chronic Hydrocephalus.—Etiology.—The causes are (*a*) *stasis*, general (cardiac or pulmonary) or local (tumor or sinus thrombosis); (*b*) *cachectic* transudation, as in carcinoma or nephritis; and (*c*) *inflammation*, beginning as acute meningitis of the sporadic or epidemic type, or as slowly beginning chronic basal meningitis, especially in young children. The same points of obstruction may exist as in the congenital variety. (*d*) Rickets is a frequent associate. Some forms may be angioneurotic.

Symptoms.—Meningitic symptoms usher in some cases in which after weeks new evidences of brain pressure appear, as alteration in the pulse and breathing, unequal pupils or coma. Headache, vomiting, visual disturbance, spastic paresis, epilepsy, altered mentality, idiocy and involvement of the cranial nerves are seen, as in the chronic congenital form. The shape of the head, however, is not altered.

Diagnosis.—Differentiation between chronic congenital and acquired forms is very difficult, without an accurate history. (*a*) From *rhachitis:* The two affections are often associated. The rhachitic head is square rather than globular, it protrudes more in front and laterally than behind, the sutures are less widely separated, the fontanelles are wide but do not bulge, and there are no signs of brain tension. The rhachitic head should be measured, because it seems larger than it actually is. (*b*) From *brain tumor:* The two conditions may be coincident. The congenital form is the more easily distinguished by the history and its slower course, but focal symptoms are less common, and spinal symptoms are more common than in tumor. (*c*) *Hyperostosis* of the skull, brain hypertrophy and acromegaly enlarge the head, but are separable by the same arguments as in rhachitis.

Treatment.—Few therapeutic results can be obtained. *Bandaging* the head is sometimes helpful, but it may aggravate the brain compression. *Puncture* of the *lateral ventricles* dates from the time of Hippocrates. Isolated recoveries are recorded, but the evacuation must be slow. Permanent drainage is usually fatal. Attempts to effect anastomosis between the ventricles and the subdural space are sometimes successful. Lumbar puncture in acquired forms may be useful, but may cause death.

DISEASES OF THE CEREBRAL MENINGES.

Inflammation of the dura is termed pachymeningitis, and inflammation of the arachnoid and pia, leptomeningitis or meningitis.

PACHYMENINGITIS.

External pachymeningitis is secondary to skull disease, infections and cerebral atrophy. Its symptoms are indefinite, as pain, headache, possibly hemiplegia, or are those of the primary lesion. Surgery in Macewen's 22 cases gave 100 per cent. recovery.

Internal pachymeningitis is divided into (*a*) the purulent form, which follows bone disease or leptomeningitis; and (*b*) the pseudomembranous or serous type, seen chiefly in general paralysis or in children with perisplenitis. More important is (*c*) *pachymeningitis hemorrhagica interna*, described by Heschl (1855).

Etiology.—(*a*) Fifty per cent. occur in persons over fifty years of age and 40 per cent. over sixty. (*b*) Seventy-seven per cent. of cases occur in *males*. (*c*) *Paretic dementia* causes 19 per cent. Pachymeningitis is common in *other dementias* and chronic psychoses, whence its great frequency in asylums and poor-houses. It also occurs in arteriosclerosis and encephalomalacia. (*d*) *Chronic alcoholism*, frequently with cardiac and renal disease, is a frequent cause. (*e*) *Trauma*, as during birth, from forceps delivery or from small pelvis. (*f*) *Hemorrhagic diathesis* (scurvy, hemophilia, pernicious anemia), chronic nephritis. (*g*) *Infective diseases*, as typhoid, puerperal fever (9 per cent.), heart disease (18 per cent.), hereditary syphilis (11 per cent.), and tuberculosis (23 per cent.) are also causes.

Pathology.—1. Gross Pathology.—The inner surface of the dura is cloudy, yellow or brown from punctate hemorrhages or hematoidin deposits. In pronounced cases the dura shows membranous laminæ, maybe a quarter of an inch thick, which are adherent by proliferation of bloodvessels and by organization. Those layers nearest the brain are the most newly formed. Repeated hemorrhage into the layers of the newly formed tissue or between them may be an inch thick or reach the size of an egg. The free hemorrhage may spread over the entire surface of the brain, even reaching the retina. Corresponding to the most frequent seat of pachymeningitis, the convexity of the brain is compressed, especially over the motor areas and bilaterally in 56 per cent.

2. Minute Examination.—The fresh fibrin and the exudation contain red cells and, nearest the brain, a covering of endothelial cells. Later the exuded fibrin develops capillaries, organizes and again hemorrhage occurs from the newly formed vessels. This organizes in turn, so that successive laminæ develop.

3. Pathogenesis.—Jores proved that two classes of cases exist: (*a*) The hemorrhage is primary and the simple clot organizes into firm connective tissue; this variety is called *regressive* and occurs in cases due to trauma, blood diseases, infections and arteriosclerosis. (*b*) In

chronic pachymeningitis proper, the inflammation is primary, the process is *progressive*, the connective tissue formed in layers is looser, more vascular, and its inner, successively formed layers are the seat of repeated hemorrhages.

Symptoms.—Generally there are no symptoms, because the hemorrhage is slight or overshadowed by the original disease.

1. General Symptoms.—The most frequent is headache, which is of little differential value, diffuse and most marked in alcoholics. There may be prodromal delirium, slow and irregular pulse, choked disk, moderate albuminuria or some fever.

2. Focal Signs.—The most important is *hemiplegia*, which is preceded for some time by coma (Fürstner). The paralysis is contralateral, rarely complete, varies with increase or decrease of the clot, often involves the cortical facial and hypoglossal centres, is sometimes bilateral and associated with aphasia (33 per cent.), hemihypesthesia (hemiataxia), hemiamblyopia, contractures in the paretic members and Jacksonian epilepsy. Conjugate deviation of the head and eyes, nystagmus and forced postures may be observed. The patient may grasp at his clothes or at his hair or beard. The pupil contralateral to the lesion is usually larger; the pupils react poorly and later both are wide. The tendon reflexes are increased, but the skin reflexes are decreased or abolished during the coma. Basal nerve involvement, trismus and rigidity of the neck are uncommon.

Course and Prognosis.—Death may occur in the first seizure or the symptoms may subside for months, possibly permanently. Residual signs include headache, ataxia, psychical alteration, paresis and aphasia. Recurrence is frequent, with stupor, coma and hemiplegia until death occurs from the fundamental disease, hemorrhage or intercurrent affections.

Diagnosis.—A correct diagnosis is rare. A patient with paretic dementia may die from a pseudo-apoplectic shock or pachymeningitis; a nephritic may succumb to pachymeningitis, apoplexy, uremia, brain edema or heart failure. A presumptive diagnosis can be made from the age, causal disease, preceding dulness or coma, hemiplegia, small pupils and convulsions, and recurrences.

In pachymeningitis there is more cortical irritation than in cerebral hemorrhage, the coma and paralysis are more variable and temperature, narrow pupils and optic neuritis are more common. In encephalomalacia, the hemiplegia is more often attended by aphasia and hemianesthesia, and there is less cerebral irritation. Brain tumor is easily distinguished.

Treatment.—Treatment relates to prophylaxis and the seizure. The use of the ice-cap, quiet, elevation of the head, application of leeches back of the ear, and possibly venesection are indicated. Nourishment should be given by rectum. Morphine and chloral relieve the headache, excitement or convulsions. Lumbar puncture has not proved beneficial. Operation was first performed by Ceci in traumatic and Annandale, Jaboulai, Monroe and Ballard in other varieties.

MENINGEAL HEMORRHAGE.

Etiology and Pathology.—(*a*) *Trauma* may rupture the anterior, middle or posterior meningeal arteries, with or without fracture of the skull, usually at the site of trauma, but sometimes by *contre-coup*. The corresponding veins may rupture, also the sinuses or the pial vessels, from the latter of which blood may percolate to the base of the brain or cord. The internal carotid may be ruptured and death then occurs. External hemorrhage results most frequently from middle meningeal rupture. The blood clot may weigh 3 to 17 ounces or reach the size of the fist, compressing the brain and dislocating the falx. *During birth* (see INFANTILE CEREBRAL PARALYSIS) trauma may be a factor; large symmetrical hemorrhage of the convexity is common. (*b*) *Arteriosclerosis* and rupture of cerebral aneurysms; (*c*) acute infections, hemorrhagic diathesis, sinus thrombosis and rarely heart and lung disease; (*d*) 50 per cent. of cases occur under forty years of age. Small clots may be absorbed or microgyria, porencephalia, cysts or brain sclerosis may result.

Symptoms.—Clinical latency is not rare in the newborn. Small hemorrhage in the adult may produce no symptoms. Very large effusions result in early, sudden death. In rupture of the *middle meningeal* artery there are (*a*) symptoms of brain pressure with those of brain concussion or contusion, as loss of consciousness, irritative symptoms, increased breathing, slowed pulse, pallor, vomiting and fever of 100° to 101°. Either death or improvement may result. (*b*) In other cases improvement from the concussion and contusion occurs, but after an interval of hours or days, *hemorrhage occurs or recurs,* with coma, pressure symptoms, mono- or hemiplegia and convulsions, as in pachymeningitis. Rigidity is less frequent than in meningitis. Hemorrhage of the sinuses and pia are said to cause monoplegia.

Prognosis and Treatment.—Ninety per cent. of cases die from expectant treatment; 33 per cent. die in twenty-four hours and, when the hemorrhage is due to aneurysmal rupture, 30 per cent. die in ten hours. Early surgical treatment is indicated.

ACUTE SUPPURATIVE LEPTOMENINGITIS.

In 1768, Whytt noted the acute hydrocephalic symptoms of meningitis, but failed to recognize the meningitis, which Quinn (1779) described. In 1855 Rilliet and Barthez distinguished the suppurative, secondary, tuberculous and other forms.

Etiology.—Suppurative meningitis is generally a secondary process.

1. TRAUMA.—Microörganisms from the ear or nose may find ready access to the brain when its resistance is lessened by trauma.

2. REGIONAL EXTENSION.—(*a*) Disease of the *middle ear*, antrum or petrous bone may result in suppurative or serous inflammation (see BRAIN ABSCESS). (*b*) Extension from the nose is less common. The disease may be spontaneous or operative, syphilitic, tuberculous, menin-

gococcic, neoplastic or suppurative, of the nasal mucosa or of the ethmoidal, sphenoidal or antral cells. Infection may travel through the bone, along the veins to the dura or along the olfactory nerve endings. (*c*) From the *eye*, the avenues to the brain are numerous, and yet disease of the eye itself, even panophthalmitis is less important than orbital cellulitis or enucleation, which exposes the sheath of the optic nerve. (*d*) Extension may result from *parotitis*, carbuncles of the face, whose veins communicate with the cavernous sinus; scalp affections; erysipelas, etc.

3. METASTATIC OR SECONDARY EXTENSION.—(*a*) *Septicopyemia*, from lung suppuration, empyema, arthritis, endocarditis and kindred affections; (*b*) *pneumonia* and rarely exanthemata may produce it; in this group there is an intermediate link, as otitis media in scarlatina and empyema or lung infarct in typhoid.

Bacteriology.—(*a*) The *pneumococcic* is the most frequent variety, being primary or secondary to pneumonia. (*b*) *Pyogenic organisms*, especially the Streptococcus pyogenes and the Staphylococcus pyogenes aureus; (*c*) the *typhoid, colon* and *influenza* bacilli, pneumobacillus, actinomycomata, and many other organisms have been found.

Pathology.—1. *Macroscopically* a purulent exudate effuses between the dura and leptomeninges and in the meshes of the arachnoid. The dura is tense. The leptomeninges are reddish, cloudy and often dotted with punctate hemorrhages. The exudate is purulent, yellow or whitish-green and succulent. The infection spreads rapidly, follows the perivascular lymphatics, as streaks of pus, imbeds the vessels and nerves in its meshes, obliterates the sulci and involves the brain and cord membranes. The brain is always swollen, springs out as the membranes are cut, is vascular and sometimes locally anemic from compression by the exudate; its surface is most affected by small abscesses, punctate hemorrhages or minute islets of encephalitis, particularly in the metastatic form (convexity meningitis): the base is most frequently concerned in ear disease. There may be primary or secondary sinus thrombosis. The ventricular fluid is increased, cloudy, purulent or sometimes serous. The apertures of the ventricles may be occluded; inflammation may be noted in the velum interpositum, choroid plexus or ependyma. In the spinal membranes the changes are similar and are most marked over the posterior part of the dorsal region. The process is usually diffuse. On incising the membranes the swollen cord bulges out and the same changes are seen as in the brain.

2. *Microscopically* there are perivascular exudation of red and white cells and fibrin formation, even in those cases in which the membranes appear normal to the naked eye. Exudation is most marked over the cortex and near the ventricles, but may also occur deeper in the medullary substance. The ganglionic cells show degeneration. Analogous findings are present in the cord, on its surface or about the central canal. In the optic nerve, exudation, hemorrhage and edema are seen, possibly also in other cranial and spinal nerves. Various bacteria are found, as the pyogenic organisms, the pneumococcus and the meningococcus; pathologically (though not bacteriologically), the suppurative and epidemic forms are identical.

Symptoms.—Prodromal phenomena are ambiguous; otitis, pneumonia or pyemia may overshadow or totally obscure meningeal manifestations. Complete *latency* is possible, as in a convalescent pneumonia patient, who while dining, felt dizzy and died in a few minutes. The autopsy revealed a massive, purulent cerebrospinal meningitis, which had not produced a single sign or symptom. The *onset* may be sudden with chill and fever. *Headache* is usually the first and most prominent symptom, and is often associated with vomiting. The *sensorium* is disturbed, delirium is succeeded by stupor and finally by coma, in which headache still persists. *Convulsions* are particularly equivocal in children. *Optic neuritis* may develop if the course of the disease is not too precipitate. *Rigidity* of the *neck and spine* is soon noted, as well as *hyperesthesia*, hyperalgesia and muscular rigidity. *Fever* is usually present. It is usually higher than in other types, perhaps 104° to 105°, and more continuous. Toward the end it may be 107° or may drop to subnormal. The pulse is faster and irregular. *Irritation* and *paralysis* of the *cerebral nerves* develop and are manifested by ocular paralysis, pupillary inequality and tardy reaction to light, if the inflammation is basal. If the process invade the convexity, *cortical irritation* may be manifested by Jacksonian epilepsy and mono- or hemiplegia. *Lumbar puncture* reveals purulent fluid and the organisms of suppuration. *Kernig's sign* is present. The abdomen is often retracted, there may be vomiting, the urine is febrile and frequently contains albumin and peptone. The course is generally rapid; it lasts a few hours or more, usually two to ten days, and almost invariably results fatally in coma. The diagnosis, differentiation and treatment are considered under Cerebrospinal Fever.

SEROUS MENINGITIS.

This is more frequently seen at the bedside than at autopsy. It is often tuberculous. The membranes are glistening, clear or flocculent fluid is seen in the arachnoid meshes and ventricles, which may be the sole seat of the disease, and the pia especially shows exudation. It is often mistaken for edema of the brain. The microscope may be necessary to demonstrate inflammation.

Symptoms.—No definite symptoms can be outlined. The disease is too frequently diagnosticated. Headache is common; vomiting, rigidity, fever, slow and labile pulse, optic neuritis, hyperesthesia, spasms and paralyses of the cerebral nerves are much less frequent than in the other forms of meningitis. The symptoms resemble those of brain tumor. Lumbar puncture in some instances gives absolute relief; the prognosis is most favorable in this type.

CHRONIC LEPTOMENINGITIS.

Etiology.—It may result from (*a*) acute leptomeningitis; (*b*) trauma, atrophic and sclerosing brain diseases, as paretic dementia, hydrocephalus and softening and in alcoholism, nephritis or plumbism or (*c*) suppuration, syphilis and tuberculosis.

Pathology and Symptoms.—The leptomeninges are diffusely or locally dense, opaque and fibrous. In the chronic suppurative form the pus loculi are situated in a firm, fibrous tissue. The symptoms, if cortical, are like those of the chronic tuberculous form; if basal, nerve paralysis and optic neuritis are common. In the *chronic simple form* (Gee and Barlow) the process may begin acutely and may persist, especially at the base and in the posterior fossa. Possibly it is a sequel of a localized epidemic or tuberculous type. This "simple meningitis of children" occurs in 76 per cent. in the first year of life. Rigid neck is the leading symptom. Paralyses, convulsions and rigidity are rare. Fifty per cent. of patients die. The *alcoholic form* is slight, diffuse and cortical; meningitic symptoms are obscured by alcoholic disease of the liver, kidneys, heart or peripheral nerves. *Diagnosis* is difficult, and *therapy* wholly etiological.

DISEASES OF THE SPINAL CORD.

GENERAL ANATOMICAL, PHYSIOLOGICAL AND SYMPTOMATIC CONSIDERATIONS.

The nervous system is made up of numerous similarly constituted units, called *neurones* (Waldeyer). Each neurone comprises (*a*) a *central nerve cell;* (*b*) protoplasmic processes or *dendrites* from the cell which conduct (cellulipital) impulses to the cell; (*c*) an axis-cylinder or *axone* arising from the cell and conducting impulses (cellulifugal) from the cell; and (*d*) the terminal ramifications of the axis-cylinder, known as *arborizations*. The axis-cylinder often gives off collateral branches or *paraxones*. The *nutrition of the nerve cell* probably depends upon the integrity of its nucleus, and the nutrition of the cell governs that of its neurone. Disease of the cell causes degeneration of the entire neurone; division of a process or axis-cylinder results in its degeneration below the point of separation from the nutrient cell. The neurones are mostly independent of each other; at times, especially in the retina, some degree of anastomosis between the dendrites of different systems exists (Dogiel). The nerve cells are closely grouped in the gray cortex, gray substance of the cord and ganglia of the peripheral nerves. The axis-cylinders largely course through their white substance. To comprehend localization in spinal diseases, we must recognize that pathological processes are in general of two varieties: (*a*) those involving certain neurones, or *system diseases* and (*b*) those not involving given neurones, *non-system diseases*.

The *functions of the cord* are (1) conduction of motor impulses to the muscles; (2) conduction of sensory impressions to the brain; (3) certain reflexes; (4) centres governing the bladder, rectum, etc., and (5) trophic influences.

1. The *motor tract* is composed of *two sets of neurones or segments*. The *upper segment* has its origin in the cells of the motor cortex (Fig. 61, *A*), runs through the corona radiata, internal capsule, crus, pons

and anterior pyramids of the medulla (whence the name pyramidal tracts), and crosses to the opposite side (Fig. 61, *B*), running down in the lateral columns of the cord (*C*) and giving off branches (*D, D*) to the anterior horns, where the tract ends in fine terminal filaments, which surround the motor ganglion cells in the anterior horn. (See Fig. 56.) The *lower segment* begins with the motor cell in the anterior horn (*E*), the dendrites of which connect with the terminal ramifications of the

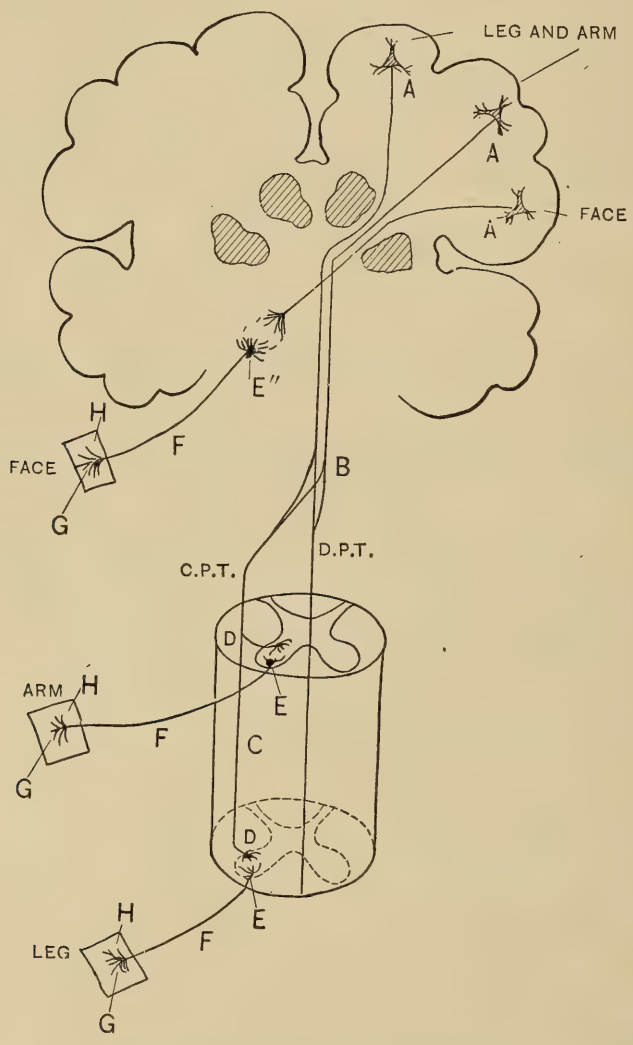


Fig. 61.—Illustrating the course of the two motor neurones: *A*, cortical cell of origin of motor tract; *B*, decussation in medulla; *C*, course in lateral columns of cord: *D, D*, branches to anterior horns in cord; in second (lower) neurone, *E*, cells of anterior horns, *E″*, cells in pons; *F*, nerve trunks; *G*, motor end plates; *H*, muscles.

axis-cylinder of the upper segment by contact (Ramón y Cajal) or by concrescence (Held); it extends along its own axis-cylinder into the anterior nerve roots and nerve (*F*), and ends in terminal ramifications (*G*, motor end plates) in a muscle, *e. g.*, of the arm or leg, which is on the side opposite to the origin of the cortical motor centre. From the cells of both segments the motor impulse travels downward (cellulifugal conduction). About 75 per cent. of the upper motor neurones cross as above described, are called the *crossed pyramidal tracts* (*C.P.T.*, in Figs.

61 and 62) and lie in the *lateral columns* of the cords; 25 per cent. of the motor fibers do *not cross* in the medulla, but pass down in the *anterior columns* of the same side—the anterior or *direct pyramidal tracts* (D.P.T.,

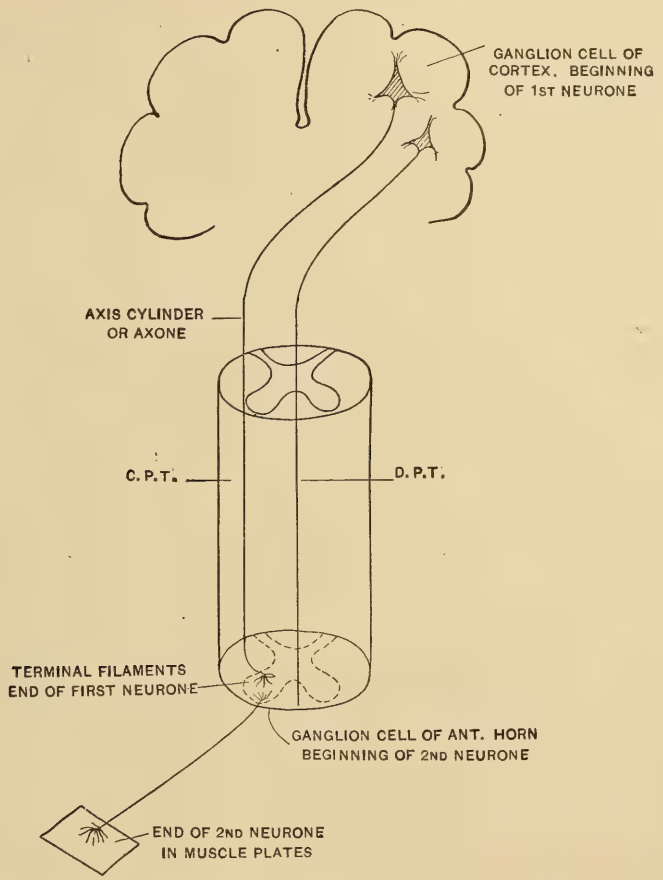


FIG. 62.—Illustrating the general outlines of the upper and lower neurones and of the direct (*D.P.T.*), and crossed (*C.P.T.*), pyramidal tracts.

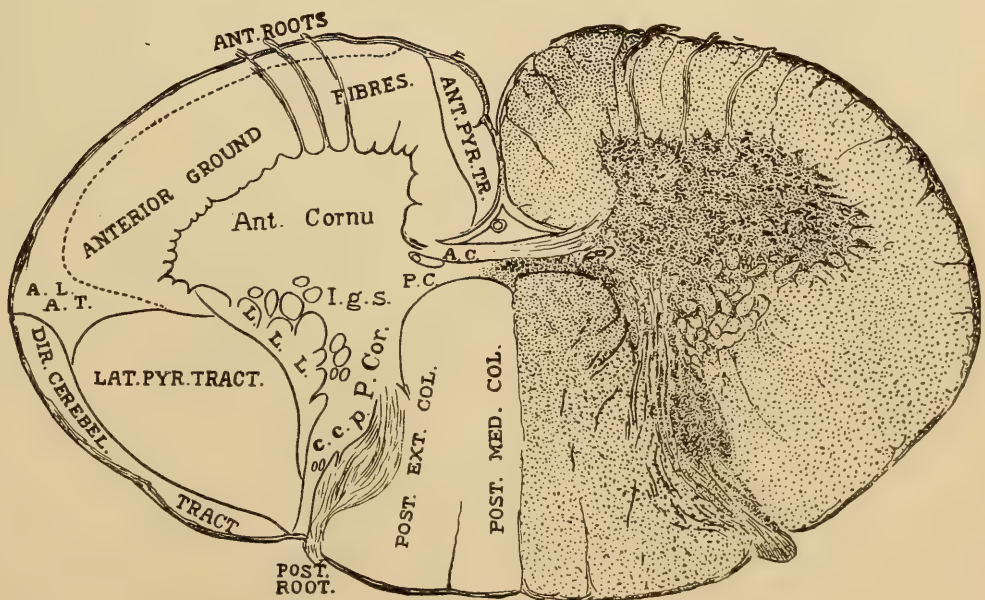


FIG. 63.—Showing the different tracts of the cord. (Gowers.)

in Figs. 61 and 62). The direct pyramidal tract probably crosses to the anterior horn of the opposite side by way of the white portion of the commissure of the cord. The upper segment is largely crossed, but the lower segment (or neurone) is a direct tract, the cells in the anterior horns supplying muscles of the same side of the body.

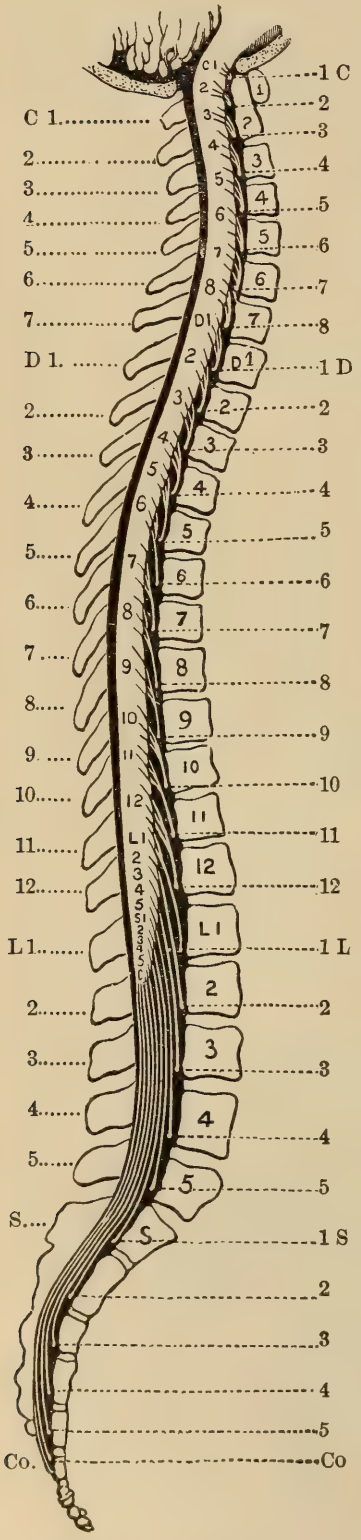


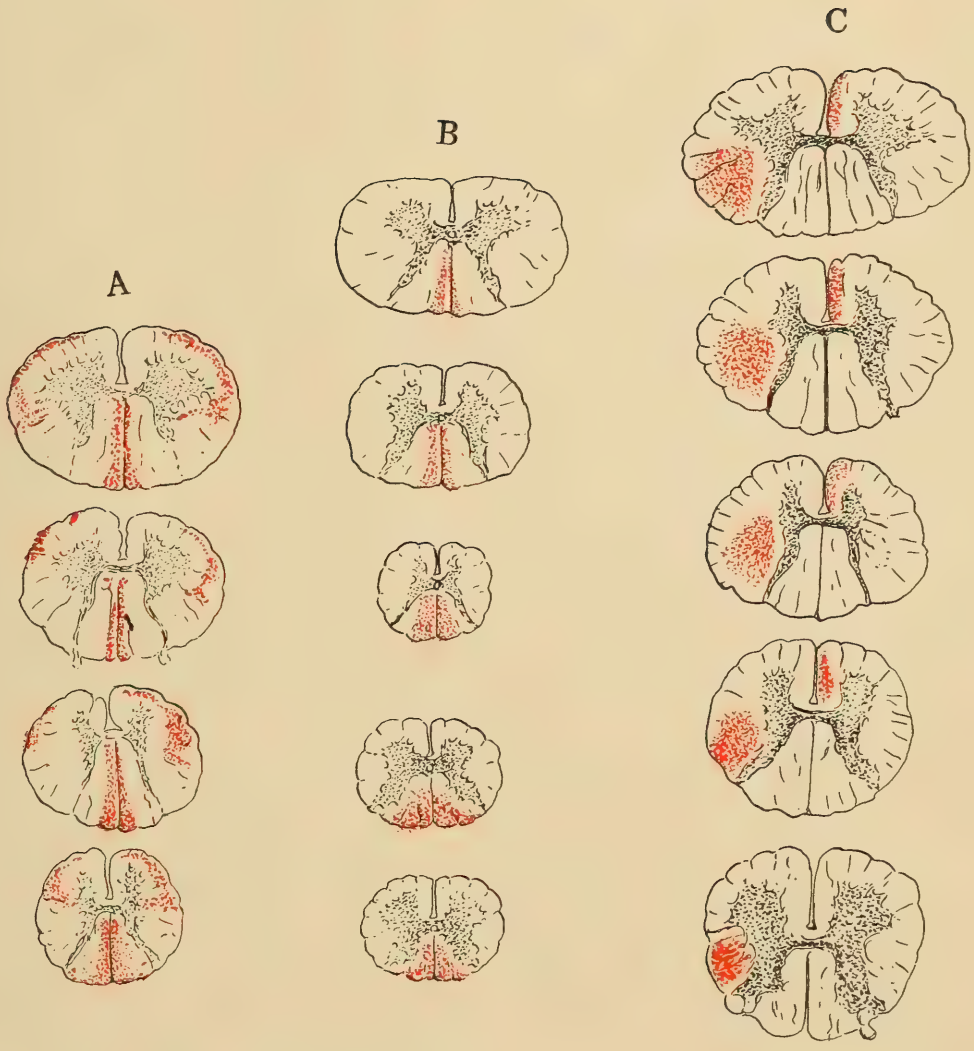
FIG. 64. — Diagram from Gowers, showing relation of vertebral spines to their bodies and to the nerve roots.

The cord therefore contains parts of both neurones, the course of which has been ascertained by the *secondary descending degeneration* which follows neurone lesions. Türck first fully described its occurrence and Flechsig and Bechterew added information from embryological studies. After a lesion, as hemorrhage in the internal capsule, the fibers of the upper neurone below the lesion degenerate; granule-bearing cells appear and the axis-cylinders swell, degenerate and stain poorly by the Marchi method, because the axones are separated from the nutrient cortical cells. The neuroglia increases and corpora amylacea develop. Degeneration is seen in the opposite lateral pyramidal tract and in the anterior column on the same side as the brain lesion. Sometimes slight degeneration occurs in the lateral pyramidal tract of the same side, which probably has some connection with the anterior uncrossed pyramidal tract of the same side (see Broadbent's theory, page 770). Since the vitality of the neurone fibers decreases as their distance from their trophic cell increases, the degeneration is greatest in the most peripheral part of the axone. The degeneration usually stops at the end ramifications of the axone (see page 766 and Plate XX).

Degeneration in the Lower Segment.—Disease of the anterior horns or the nerve trunk causes degeneration of the nerve toward the periphery and trophic muscle changes. The *reaction of degeneration* is elicited. (See NEURITIS.)

The two neurones do not correspond in number, because one neurone of the upper segment is connected with several lower neurones, *i. e.*, various cells in the anterior horns at different levels are probably excited by a single pyramidal fiber. For complex movements, as of the hands, there are more pyramidal fibers than for simple movements, as of the intercostal muscles. The anterior spinal root nerves join with fibers from

PLATE XX



A. Ascending degeneration in the posteromedian column and anterolateral ascending tracts from lumbar lesion. (After Gowers.)

B. Ascending degeneration after injury to cauda. (After Schultze.)

C. Descending degeneration of pyramidal tracts from right-sided cerebral hemorrhage. (After Mott.)

the posterior roots, and passing downward, leave the spinal cord between the vertebræ, thus forming the spinal nerves, whose area of origin in the cord is called a *segment.* The peripheral nerves often arise from several segments, which are sometimes considerably separated; muscles with similar function have similar spinal localization, because *movements rather than muscles are localized in the cord,* as well as in the brain. The table on page 824 gives the localization of the muscles, with special regard to their cells (the beginning of the second neurone), as well as of the skin and reflexes. The cord is shorter than the spinal canal, reaching only to the second lumbar vertebra. The nerve roots descend and therefore their level at the point of exit does not correspond with their level of origin. Fig. 64 shows this relation.

2. *The sensory tract* conducts upward and is more complicated and less clearly understood than the motor tract, because the interpretation of experiments on animals is confusing. It is composed of three or more neurones. The cells of the *first neurone* are in the intervertebral ganglia, whose cells end in an axis-cylinder dividing into a branch to the skin and another to the cord by the posterior root; the branches from the skin (peripheral sensory nerves) represent the dendrites of the ganglion cells and the branch to the cord represents the axis-cylinder. All peripheral sensory fibers originate in the ganglia and almost all fibers of the posterior nerve roots come from these ganglia. From the posterior roots the fibers reach the cord, where (*a*) some pass into the posterior column, (*b*) some into the gray matter and (*c*) others run to the cells of the anterior horns (reflex arcs).

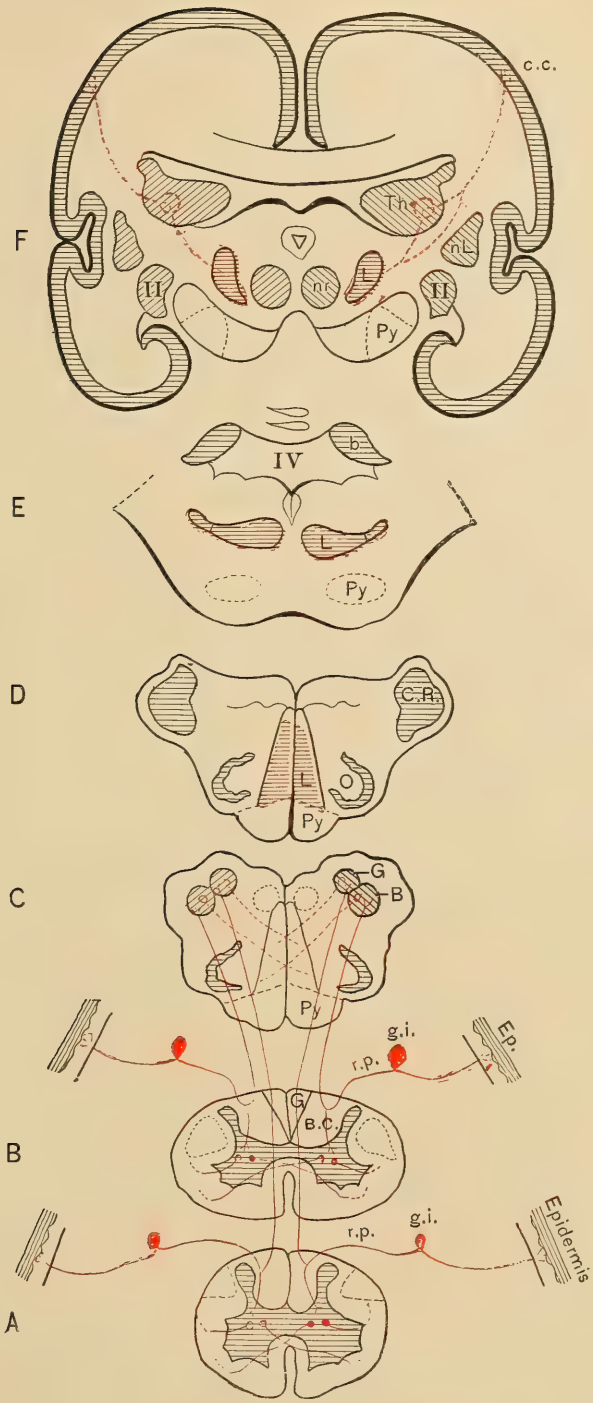
(*a*) The *fibers entering the white substance* (the *posterior columns*) divide into a short descending branch (probably concerned in reflex processes, giving off some collaterals and ending in the gray matter) and into a more important long ascending branch; by the entrance of new fibers at higher levels they become more centrally located in Goll's posterior median column in which the lowest fibers (*e. g.*, the sciatic) are most posterior and the highest (*e. g.*, the cervical) are most anterior. The *posterior columns* convey fibers for *muscle sense* (sense of posture and movement association) and possibly also for simple tactile sensation. "Sensations we do not feel" travel in these columns (Gowers). Goll's posterior median column ends in the nucleus gracilis of Goll (postero-median nucleus) in the medulla. It is uncrossed, *i. e.*, direct. Burdach's posterior external column ends in the nucleus cuneatus of Burdach (posterior external nucleus) in the medulla. It is likewise uncrossed or direct. The first neurone ends in these nuclei. The *second neurone crosses* above the motor decussation in the medulla (interior arcuate fibers) to the opposite side and unites with fibers that have already crossed in the cord, thus forming the fillet (see below, *b*). This neurone is crossed. The posterior nuclei communicate with the opposite cerebral cortex, with the cerebellum of the same and opposite side and with the external arcuate fibers.

(*b*) The *fibers entering the gray matter of the posterior horns* also divide into ascending and descending branches. Some lie at the zona terminalis, the most posterior part (Lissauer's zone) of the posterior horn. All these

Segment.	Muscles.	Skin: Sensation.	Reflexes.
2–3 Cervical.	Sternocleidomastoid, trapezius, scaleni, neck muscles. Diaphragm (3, 4, 5, 6 cerv.).	Neck and occiput.	Sudden inspiration by sharp pressure below the ribs.
4 Cervical.	Lev. ang. scapulæ, rhomboideus, supra- and infraspinatus, deltoid, supinator longus, biceps.	Shoulder (anterior part, Dana).	Widening of pupil from irritation of neck, 4–7 cervical.
5 Cervical.	Supinator brevis, serrat. mag., clavicular portion of pectoralis maj., teres minor.	Radial side of arm (volar and dorsal surfaces) to the insertion of deltoid. Post. surface of shoulder?	Scapular reflex (fifth cerv. to first dorsal). Tendon reflexes of this group of muscles.
6 Cervical.	Pronators, coracobrachialis, brachialis ant., triceps, long extensors of hand and fingers. Lower neck muscles and middle part trapezius, 6, 7, 8, C and 1 D.	Dorsal and volar surfaces of radial side of hand to midline of middle finger, and up to base of hand, narrow strip on volar and dorsal surfaces up to axilla, connecting with above area.	Tendon reflexes of muscles named.
7 Cervical.	Costal portion of pectoralis maj., latissimus dorsi, teres maj.	Ulnar part of hand (dorsal and volar) from middle of the fourth to middle of third finger and connecting with it, a moderate strip on volar and dorsal surfaces of arm.	Volar reflex of hand, periosteal reflexes of radius and ulna. Tendon reflexes of muscles named.
8 Cervical.	Long flexors of the hand and fingers.		
1 Dorsal.	Extens, poll. long. et brev. Small muscles of hand.	Dorsal and volar surfaces of hand to middle of fourth finger, narrow strip on dorsal and volar surfaces of arm up to axilla.	Corresponding tendon reflexes.
		Narrow strip on ulnar surface of arm and forearm down to base of hand. (The upper part, perhaps, belongs to the second dorsal segment.)	Tendon reflexes.
2–12 Dorsal.	Muscles of back (also lowest part of trapezius) and abdomen. Intercostals D, 1–10.	Chest, back, abdomen and upper gluteal region. (Umbilicus, tenth dorsal; ensiform, sixth to seventh dorsal).	Abdominal reflex. Epigastric reflex (according to Dinkler, is at the ninth dorsal, the middle and lower abdominal at the tenth and twelfth, respectively).
1 Lumbar.	Abdominal muscles, quadratus lumborum. Ileopsoas.	Pubic area, anterior aspect of scrotum.	Cremasteric reflex (1–3 lumbar).
2 Lumbar.	Sartorius (or third lumbar), flexors of hip. Quadriceps femoris (extensors of knee), cremaster.	Anterior and inner side of hip, inner side of leg to malleolus, inner side of foot, external surface of hip, lumbar regions.	Patellar reflex (2–4 lumbar). Erection (lumbar cord). Uterus (lumbar cord).
3 Lumbar.	Internal rotators of thigh.		
4 Lumbar.	Adductors of hip. Sartorius (?). Abductors of hip. Tibialis anticus. Calf muscles.	Posterior surface of hip, thigh, external surface of leg and foot.	Gluteal reflex (4–5 lumbar).
	Flexors of knee (?), gluteals (extensors of hip).	Back of foot.	
5 Lumbar.	External rotators of hip. Flexors of foot (?). Extensors of toes. Peronei.		
1–2 Sacral.	Flexors of foot and toes. Small muscles of foot.	Skin of sacrum, anus, perineum, genitalia.	Plantar reflex; ejaculation (3–4 sacral). Achilles tendon reflex.
3–5 Sacral.	Muscles of perineum.		Bladder and rectum reflex.

The brackets to the left concern only the muscles, and indicate the origin of the muscles from several nuclei.

PLATE XXI



Scheme of Sensory Conduction. (Strümpell.)

A.—Entrance of posterior sensory roots into lumbar cord: *g.i.*, intervertebral ganglion; *r.p.*, posterior root; part of the fibers end in the posterior horns, from whose cells other fibers arise and enter the lateral columns, partly on the same and partly on the opposite side; other fibers from the posterior roots, course upward in the posterior columns and form in:

B (the cervical cord).—Goll's columns (*G*); *B.C.*, Burdach's columns.

C.—Medulla oblongata, *G* and *B* are the nuclei wherein Goll's and Burdach's columns end. From these, new fibers arise which decussate and form the fillet (lemniscus, *L*) in:

D.—*L*, lemniscus; *Py*, pyramidal tract; *O*, the olive; *C.R.*, corpus restiforme.

E.—Pons; *IV*, fourth ventricle.

F.—*L*, lemniscus of fillet; *n.r.*, nucleus ruber; *Py*, pyramidal tract in crus cerebri; *nL*, nucleus lenticularis; *Th*, optic thalamus (beginning of new neurone to cortex?).

fibers end with terminal ramifications about ganglion cells in the gray matter; they form the beginning of a second neurone which crosses in the commissure of the cord and courses upward in the anterolateral column and anterior ground fibers of the opposite side (just anterior to the lateral pyramidal tracts) and they unite in the medulla with the fibers of the second neurone of (*a*) (*v. s.*). (See Plate XXI.) The fibers entering the posterior horn convey *temperature and pain conduction* and travel between the posterior median column and the central canal. *Tactile sensation* is probably conveyed in the anterolateral column, which conveys most of the upward sensory impulses, since their division causes anesthesia (Gowers). Both (*a*) and (*b*) then run in the median lemniscus of the fillet and in part of the longitudinal bundles of the formatio reticularis of the pons, tegmentum of the crus and posterior limb of the internal capsule, beyond which some fibers run directly to the motor cortex or parietal lobes, while others end in the optic thalamus, whence perhaps by a *third neurone* to the cortex.

The *sensory tracts degenerate upward* (ascending degeneration). Plate XX, Fig. *A*, shows degeneration in the posterior and anterolateral columns. Most ascending degenerations stop in the medulla. The *direct cerebellar tract*, which begins at the first lumbar nerve, also degenerates upward; most of its fibers come from the lower dorsal and first lumbar nerves; some of them come from the gray substance of Clark's columns to which collaterals are sent from the posterior fibers; Flechsig believes that it conveys muscular impressions from the lower part of the trunk and the legs; its destination is the middle lobe of the cerebellum by way of the restiform body.

The *spinal sensory nerves* descend lower than do the corresponding motor nerves and skin sensation for a given area is often supplied by two, three or more nerves, whence anesthesia results only when all of them are diseased (Sherrington). (See Plate XXII.)

Lesions of the *conus medullaris* down to the fourth sacral nerve produce paralysis of the bladder and rectum and simultaneously saddle-shaped anesthesia on the anus, gluteal region, perineum, genitalia, undersurface of the thigh (pudendal and coccygeal plexuses), but the scrotum escapes because the spermatic plexus and external spermatic nerve connect with the lumbar plexus. Motor paralysis is absent because the limbs receive their supply from the lumbar and upper sacral segments. The cremaster reflex is normal. If sexual desire and erection are preserved with loss of vesical and rectal function, a conus localization is probable. Sensory dissociation, trophic changes and rapid advance of the disease argue for conus disease. Lesions of the *cauda equina* (where the roots lie closely together) are usually symmetrical. The onset is insidious and the progression slow; pain is prominent and usually the first symptom; then the leg reflexes are lost, followed considerably later by paralysis, atrophy and anesthesia (especially in the perineum, ext. genitalia and a narrow zone about the anus). For caudal lesions speak remission and improvement in the vesical and rectal functions and the slow appearance of muscular wasting, reaction of degeneration and trophic alteration. In its upper part, the above symptoms with paralysis of both legs are

noted; paraplegia dolorosa is frequent (see Plate XX, Fig. *B*). A lesion below the third lumbar vertebra is usually limited to the pudendal and sciatic distributions.

3. In the gray matter there are *reflex centres* enumerated in the table above. The reflex arc comprises the afferent sensory fiber with its termination in the gray matter and the motor nucleus with its efferent motor fiber. The fibers of the *oculopupillary reflex* run by way of the sympathetic nerve (rami communicantes) to the cord. Irritation of its centre produces mydriasis, especially in spinal caries. Paralysis from disease of the centre produces myosis, narrowing of the eye fissure, sometimes retraction of the eye-ball and flattening of the cheek; this paralysis is produced by section of the eighth cervical and first dorsal segments, *i. e.*, the same localization as for paralysis of the forearm and

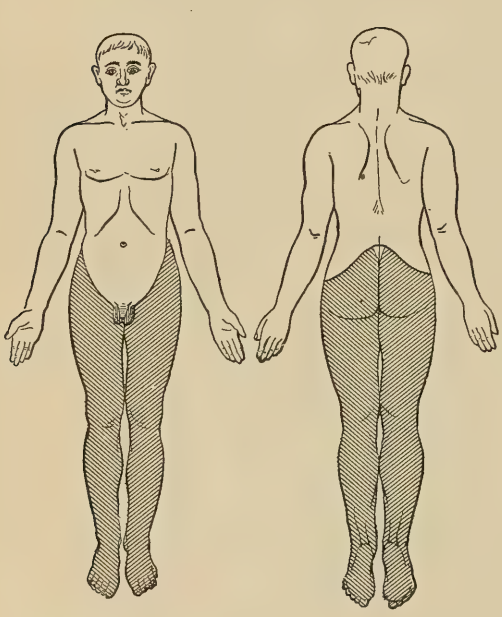


Fig. 65.—Lesion at the level of the second lumbar segment (Strümpell and Müller, from whom also following figures are borrowed).

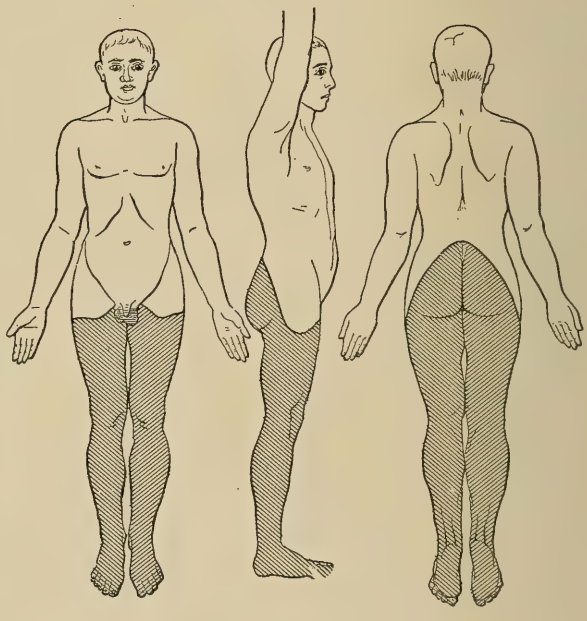


Fig. 66.—Lesion at third lumbar segment.

anesthesia of the ulnar surfaces of the hand and arm. It occurs more often in disease of the nerve roots than of the cord itself. The *tendon reflexes* will be considered under the individual cord diseases. They are inhibited from the brain and probably also from the cord. There are muscular fibers to evacuate the *bladder and rectum* and sphincters to retain their contents. The former are stimulated by distention and the latter relax; they are controlled normally by the will. If inhibition fails, the bladder or rectum is evacuated, sometimes with the knowledge of the patient, as in disease of the pyramidal tracts, or unconsciously, as in coma or when the sensory nerves of these parts are diseased. As to the rectum, disease of the centre discloses itself by continuous evacuation and relaxation of the sphincter on digital examination; disease above the centre, by normal tonicity of the sphincter.

In the above considerations, the leading points are given regarding the *level of the lesion* and the *course of the chief systems*. *Total transverse disease* of the cord produces paralysis of the muscles at and below that

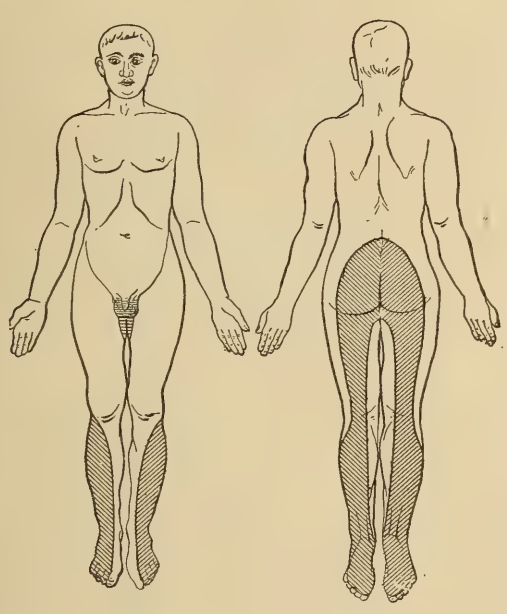


FIG. 67.—Lesion at fifth lumbar segment.

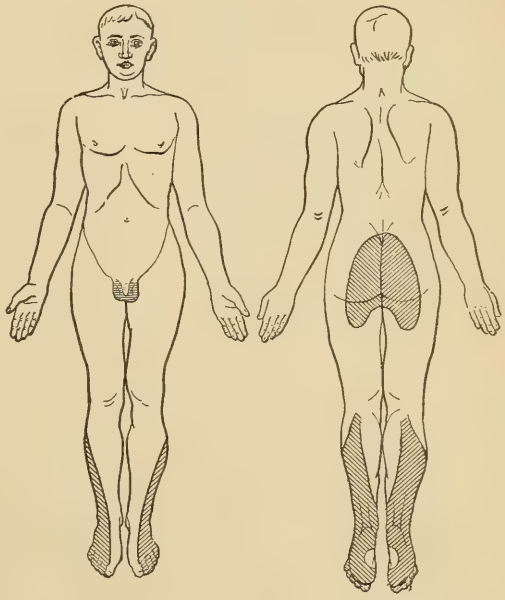


FIG. 68.—Lesion at first sacral segment.

level, and sensory interruption; it might be difficult to decide whether the cord were affected through a narrow horizontal level, or whether all the cord below it were diseased, unless the reflexes below were noted. Hyperesthesia usually discloses the level of the lesion. *Lesions of the*

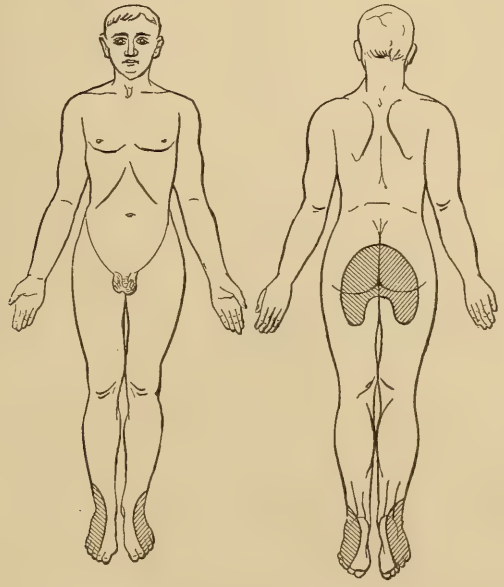


FIG. 69.—Lesion at second sacral segment.

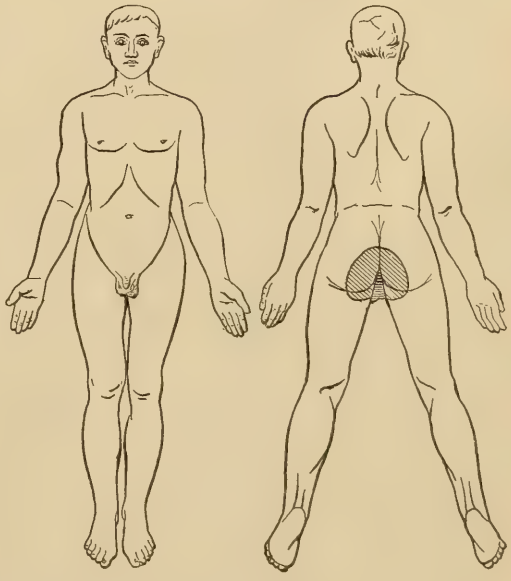


FIG. 70.—Lesion at third sacral segment.

pyramidal tract (first neurone) produce motor paralysis at the level of the lesion, increased reflexes and spasticity (hypertonic muscular rigidity) but with no muscular atrophy, reaction of degeneration or sensory dis-

turbance. Lesions of the *anterior horns or anterior roots* produce a flaccid paralysis, atrophy and the reaction of degeneration if the process is acute. (If it is chronic the atrophy is more marked than the paralysis and the reaction of degeneration is absent or atypical. The trophic cells are not specialized in the horn, but all cells are probably trophic.) The motor nerve (Nasse, 1839) and the muscle degenerate; the reflexes are abolished; the muscle tension (myotatic irritability) is lost, there is vasomotor paralysis (since the fine fibers of the anterior roots are vasomotor) and contractures result (from sensory irritation). If the lesion is in the anterior horn there may be fibrillary muscular contractions. Irritation of the anterior horns may increase the reflexes and induce contractures. In lesions of the *posterior columns* the chief alteration is sensory disturbance, especially in the muscle sense, and therefore inco-

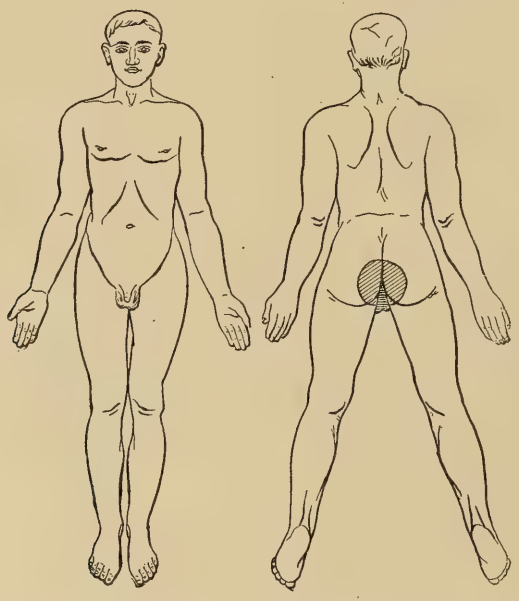
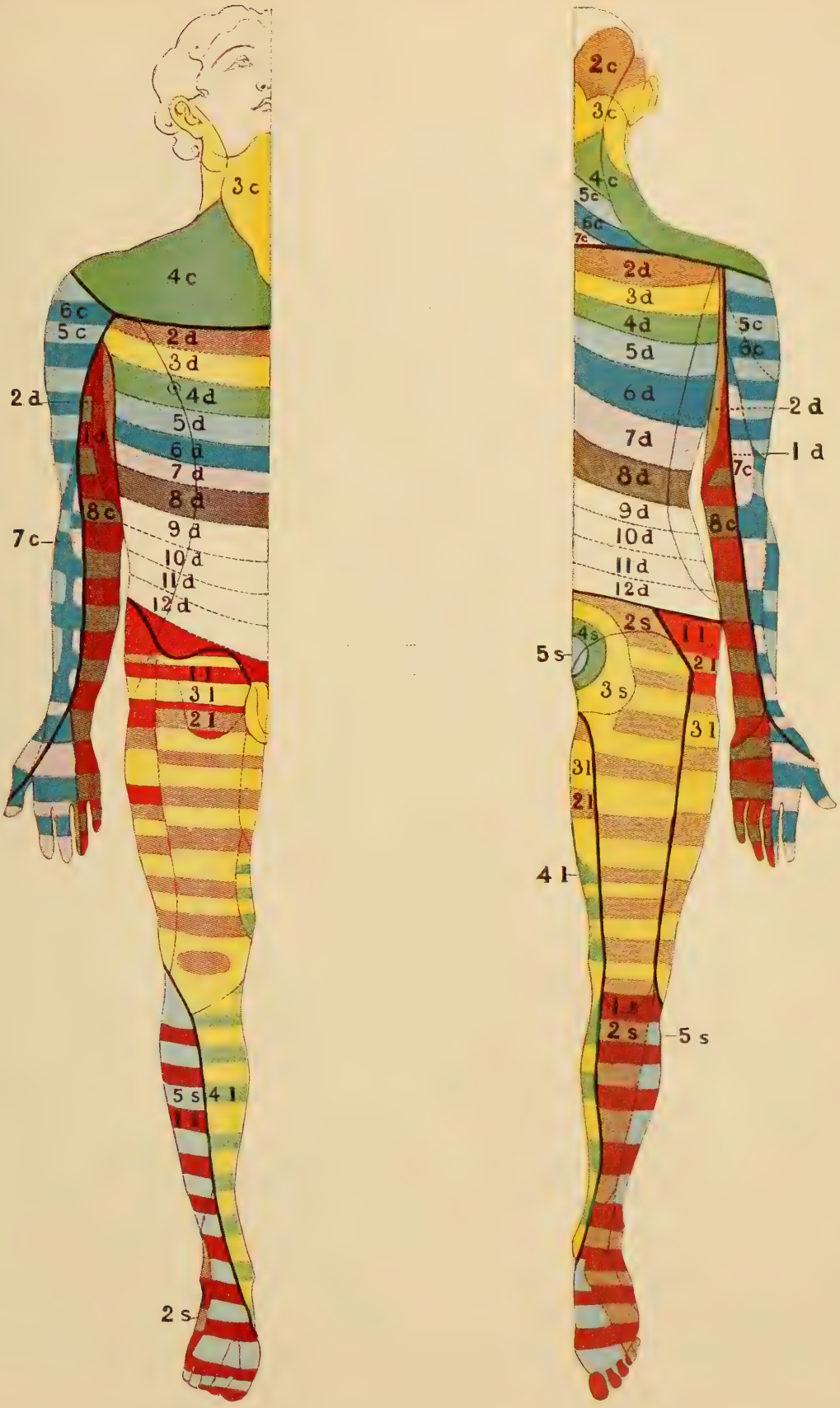


Fig. 71.—Lesion at fourth sacral segment.

ordination or ataxia. The reflex arc may be interrupted. In lesions *of the posterior horns* there are disturbance of the temperature and pain sense and some alteration of the tactile sense; the reflexes may be disturbed. In lesions of the *posterior roots* the reflexes are disturbed and there are anesthesia and ataxia. Irritation of these roots causes pain, hyperesthesia in the form of a girdle sensation around the trunk or lancinating pains in the extremities and reflex contractures. The importance of the spinal ganglion as the trophic centre must be remembered (Waller, 1852). *Vasomotor fibers* leave the cord through the anterior roots by the rami communicantes and course down in the lateral columns or in the intermediolateral tract from the centre in the medulla. Most of the vasoconstrictor fibers leave the cord between the third dorsal and second lumbar segments, while the vasodilator fibers are much more diffusely scattered through the cord.

PLATE XXII



These figures have been devised by Wichmann in order to show the distribution of the sensory areas corresponding to the segments of the spinal cord. The colors used correspond to those of the normal spectrum, red, orange, yellow, green, blue, indigo, and violet, with brown; respectively—the first, second, third, fourth, etc., segments in each portion of the spinal cord—cervical, dorsal, lumbar, and sacral. The last four segments in the dorsal region are left white. The advantage of the plate is that it shows very clearly the overlapping of the segments that has been demonstrated by Sherrington. The dark heavy black lines on the arms indicate the division between the two sides of innervation—that from the upper portion of the cervical enlargement and that from the lower portion. The heavy black lines of the legs indicate the divisions between the sacral and lumbar areas of innervation. The figures and letters indicate the segments in which the supply has been drawn and have been introduced for the sake of making the diagram more available for ready reference. C—cervical, D—dorsal, L—lumbar, and S—sacral. The horizontal bands of color without dividing lines between them indicate that both segments innervate the areas involved. (Musser.)

DISEASES OF THE SPINAL MENINGES.

Most diseases of the vertebræ and cord affect the meninges, but generally, meningeal symptoms excite little clinical interest.

I. Hemorrhage.—(*a*) Between the vertebræ and dura, hemorrhage is frequent from rupture of the subdural plexus of veins. It surrounds the nerve roots and is most severe over the posterior cord; it results mostly from trauma and to a lesser extent from vertebral disease or aneurysmal rupture. (*b*) Arachnoid hemorrhage (hematorrhachis) results from the above causes, dystocia, or hemorrhagic diseases. The entire spinal canal may be filled with blood from brain hemorrhage, especially at the base.

Symptoms.—Small hemorrhages cause no symptoms and meningeal apoplexy may be overshadowed by brain or traumatic cord symptoms. Pain in the back, rigidity, radiating pains from root compression, hyperesthesia, painful reflex spasms, motor weakness, anesthesia, sphincter disturbance and abolition of the tendon reflexes are the common symptoms. They affect the lower more than the upper cord, increase for a day or so, produce inflammatory reaction for a few days and subside in from four to eight weeks. Physical signs vary with the location; brachial paraplegia is present in cervical, and involvement of the legs in lumbar or dorsal, hemorrhage.

Diagnosis and Treatment.—In *diagnosis* the most important symptom is irritation, while in hemorrhage of the cord (hematomyelia) paralysis prevails and pain is less frequent. In non-traumatic cases, diagnosis is impossible. The *treatment* is symptomatic. Absolute rest in the lateral decubitus or on the face should be enjoined.

II. Pachymeningitis.—(*a*) This may be *external* (peripachymeningitis) and follows caries, penetrating bed-sores, deep cellulitis, psoas or retropharyngeal abscess. Its symptoms are those of compression. Root symptoms and fever are frequent. The prognosis is unfavorable and the treatment is etiological, expectant or surgical. (*b*) *Internal pachymeningitis hemorrhagica.* Its causes are those of the cerebral form (*q. v.*). (*c*) Charcot (1871) and Joffroy described a *pachymeningitis cervicalis hypertrophica,* in which the inner surface of the dura is thickened by annular concentric fibrous deposits. The dura adheres to the vertebræ and leptomeninges and compresses the nerve roots, which indurate. Several cervical segments are involved. The process is most marked on the dorsal aspect of the cord, which is flattened anteroposteriorly and shows peripheral induration by extension of the process to it through the pial vessels (meningomyelitis); this causes ascending and descending degeneration. Cold, syphilis, alcoholism and trauma are probable causes.

Symptoms.—Charcot described *three stages:* (*a*) the *neuralgic stage,* which corresponds to involvement of the posterior roots, in which pains radiate from the neck into the ulnar and median nerve supply. They are continuous, with paroxysmal exacerbations, with localization at times in the arm-joints, with rigidity, paresthesia in the arms and some motor weakness. (*b*) The *second stage* is *atrophic paralysis* of the arms, the

process involving the anterior roots. The pains are replaced by atrophy and paralysis in the ulnar and median nerve areas, while the radial area is usually unaffected; this produces flexor paralysis in the forearms and hands, and the claw-hand (*main en griffe*) results from contracture; the wrist is overextended, the first phalanges are extended, the second and third are flexed; ulnar and median anesthesia and the reaction of degeneration are found. The shoulder and elbow muscles may be involved. Deviations from Charcot's complex are at times noted, as absence of atrophy and contracture. (*c*) In the *third stage* the legs exhibit *spastic paralysis* without atrophy; the bladder and rectal functions and sometimes the sensory conduction are disturbed from secondary changes in the cord.

Diagnosis.—The process is easily localized to the cervical region. Marked pain also occurs in tumors, cervical spondylitis, syphilitic meningomyelitis and at times in syringomyelia. Amyotrophic lateral sclerosis (*q. v.*) and progressive spinal muscular atrophy cause no such severe pains and syringomyelia (*q. v.*) is characterized by dissociated anesthesia, muscular atrophy, painless felons and trophic changes in the joints. Tumors (*q. v.*) are difficult to differentiate. Neuritis causes symptoms limited to the limbs.

Prognosis.—This is usually unfavorable and chronic progression, cystitis, etc., usually develop. Instances of recovery are recorded.

Treatment.—Warm baths, counter-irritation, sedatives, narcotics, mercury, iodides and care of the skin and bladder are indicated.

TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

Of Schlesinger's 400 cases 126 were intramedullary and 239 extramedullary (151 intradural and 88 extradural).

I. Tumors of the Membranes.—These may arise from the dura or leptomeninges. In Horsley's *extradural tumors* there were sarcoma, lipoma, tubercle, echinococcus cysts, myxoma, fibrochondroma, carcinoma and fibrosarcoma. Cysticercus often causes no symptoms. *Intradural tumors* include myxoma, sarcoma, fibroma, psammoma, tubercle, parasitic and syphilitic tumors. Lipomata, echinococci, cysticerci, neuromata and angiomata are rarer. Usually single, multiple occurrence may be noted in sarcomata, parasitic tumors and neuromata. This group compresses the cord or its nerve roots. There are edema of the cord, flattening, peripheral hardening, central softening and ascending and descending degenerations. The nerve roots atrophy.

Symptoms.—Extradural growths produce more vertebral than cord symptoms. The first symptoms are usually those of compression of the *nerve roots*, *e. g.*, radiating pains, first on one side, then on both, which run into the arms, trunk or legs and advance from one root to another; hyperesthesia; paresthesia; anesthesia, which is less common since it implies involvement of at least three roots; atrophic paralysis; rigidity; and spontaneous muscular contractions. Local tenderness is rare. The symptoms recall those of carcinoma of the spine aside from its

vertebral signs. Later, evidences of *compression* of the cord appear, as paresis, perhaps of one side first (Brown-Séquard's type, *v. i.*) or paraparesis with atrophy if the anterior horn is involved at that level, with increased reflexes (unless the arc is broken), with spasticity and contractures; and with sensory changes. As in carcinoma of the vertebræ, the paraplegia dolorosa of Cruveilhier is common, as is anesthesia dolorosa (the "eccentric projection" or reference of pain to the anesthetic areas). Finally, there are total paraplegia, anesthesia and bladder and rectal paralysis.

II. Tumors in the Cord Substance.—They are less common than tumors in the membranes, and include *glioma* (cervical and upper dorsal regions), sometimes gumma, sarcoma (pia), tubercle (gray substance or posterior horn) and cysticercus. Most are circumscribed, though glioma tends to vertical diffusion. Unlike meningeal tumors, they are rarely metastatic.

Symptoms.—In intramedullary growths, root symptoms are absent, especially in gliomata and, since there is gradual compression of the cord, the clinical picture may resemble chronic myelitis and syringomyelia. The cord symptoms are disturbed conduction; spastic paraplegia; marked increase of reflexes (unless the arc is broken or absolute cord compression develops); bilateral symptoms, or if unilateral, they soon reach the other side; extensive muscular atrophy, if the cervical or lumbar enlargement is diseased, often then with the reaction of degeneration; and sensory changes involving all varieties of sensation or certain varieties only, as pain or temperature (dissociated or partial anesthesia). Bruns maintains that the lesion and symptoms are at first unilateral, followed by the Brown-Séquard complex (*q. v.*). As the tumor grows, total paraplegia and anesthesia may, in rare cases, develop below the level of the lesion.

Localization.—Accurate localization is most important but rarely possible until the cord itself is compressed. The most common error is in locating the tumor too low. Atrophic paralysis, in the cervical or lumbar region, is the most certain aid. Sensory changes are less reliable; the actual lesion is two, three or four segments higher than the anesthesia because anesthesia results only from involvement of at least three roots (Sherrington's law). The hyperesthesia usually represents the level of the lesion and local spinal tenderness, though rare, is very important. Finally, the spinal segments involved must be considered in terms of the corresponding vertebræ (*v. s.*, page 822). (*a*) In a tumor of the *upper cervical* cord there are pains in the cervical plexus and perhaps anesthesia; at first there may be a spinal hemiplegia differing from cerebral hemiplegia in that the face and hypoglossus escape and the anesthesia is contralateral to the paralysis; then there is spastic paralysis of all four extremities, which rarely lasts long, since affection of the phrenic nerve causes early death. (*b*) In tumor of the *cervical enlargement* there are atrophy, flaccid paralysis and pain in one arm, spastic paresis in the leg of the same side, anesthesia of the entire opposite half of the body and opposite limbs; and then paralysis of both arms and legs with anesthesia. (*c*) Localization in the *dorsal cord* often produces

unilateral signs, as paresis of one leg with its half of the trunk, and anesthesia of the corresponding parts of the other side; spastic paraplegia of legs and abdomen, retention of urine and later incontinence; and then girdle pains. (*d*) In tumors of the *lumbar enlargement* there are unilateral pains in the lumbar plexus and atrophic paralysis in the ileopsoas, quadriceps and adductors; there is total anesthesia when the cord is involved in the region of the lumbar plexus; later partial anesthesia (affecting the sense of temperature and pain) develops in the region of the sacral plexus; still later there is complete paralysis and anesthesia in the region of the lumbar plexus, with atrophy, absence of the patellar reflexes, the reaction of degeneration, spasticity of the legs and feet, and Achilles clonus. (*e*) If the localization is in the *sacral cord* there is atrophic paralysis of the leg and foot, posterior muscles of the thigh, gluteal and perineal muscles, anesthesia, total paralysis of the bladder and rectum, impotence, loss of the Achilles reflex (while the patellars may persist), early decubitus and cystitis. (*f*) If the tumor is in the *cauda*, the symptoms are usually bilateral from the beginning; paraplegia is rarely as symmetrical as in cord lesions; there is the reaction of degeneration; pain is violent, since many sensory fibers are in close contact; Minor described bilateral and even unilateral sciatica as an early symptom. The sacral plexus is chiefly or first involved, while growths of the lumbar enlargement affect both the sacral and lumbar plexuses.

Diagnosis.—The diagnosis of meningeal tumor, always difficult, is based on (*a*) local pain and rigidity, which are especially marked in the extradural type; (*b*) hyperalgesia; (*c*) radiating pains, *i. e.*, root symptoms which later gradually give way to (*d*) cord symptoms, often unilateral at first and then bilateral, as interrupted sensory and motor conduction and increased reflexes. *Carcinoma* of the vertebræ produces more pain on movement than other tumors. In *caries* there is less pain, local vertebral changes eventually appear, and the course is usually more rapid. In *myelitis* the arms are less often paralyzed; radiating pains are much rarer than in tumor and a girdle sensation is more common. The local pain sometimes suggests *aneurysm*, lung disease or peripheral neuralgias but the tender points of Valleix are absent. Root symptoms, early interruption of cordal conduction and absence of trophic disturbance in the bones and of dissociated anesthesia argue against *syringomyelia*. A diagnosis of *tuberculosis* or syphilis is fortified by its presence in other organs. In youth, tubercle; at puberty, sarcoma and glioma; in the third and fourth decades, glioma, tubercle or sarcoma; and in advanced years gummata are most frequent. *Diffuse sarcomatosis* is usually cerebrospinal, rarely invades the cord substance, affects the posterior cordal surface most frequently and generally in the lumbar and lower dorsal regions; it occurs largely in the young and its course is rapid. *Tabes* in one remarkable case was simulated by multiple tumors of the posterior roots. *Tumors of the cord* are more difficult of differentiation, for root symptoms are often lacking.

Course and Prognosis.—Slow development, progression either steady or "by starts," sometimes remissions and eventual compression of the roots or cord are usual, with death from cystitis or decubitus. Sudden

paralysis is less common. The average duration is one to three years, but a longer course is possible (even fifteen years).

Treatment.—1. THERAPEUTICS.—Narcotics are employed sparingly in the early stages, lest their effect fail later if the course is protracted. Syphilis is always a possible factor.

2. SURGICAL TREATMENT.—Gowers first, 1887, localized a fibromyxoma which Horsley removed successfully. Tumors of the cord are less favorable than those of the meninges. Intramedullary tumors may only crowd the nerve paths, but in their removal the attendant laceration or acute myelitis results in damage equal to that of the tumor. Accurate localization is most important. Death may result from shock, escape of cerebrospinal fluid and infection. The operative mortality is 47 per cent. In Stursberg's 126 cases, 30 per cent. recovered.

CIRCULATORY DISTURBANCES. HEMORRHAGE. TRAUMA OF THE CORD.

I. Anemia of the Spinal Cord.—Paralyses referred to anemia of the cord are mostly due to neuritis, hemorrhages in the cord or nerve sheaths, and to degeneration. Paralysis following profuse hemorrhage from the stomach or uterus may be properly referred to cord anemia. Stenson (1667) demonstrated experimentally that compression of the aorta produced paraplegia due to anemia of the cord, the motor cells of which appear to be especially susceptible to ischemia.

II. Embolism and Thrombosis (Myelomalacia).—Embolism of the spinal arteries is extremely rare, though observed in endocarditis. No separate clinical symptoms can be recognized. Thrombosis is very rare. Venous thrombosis is secondary to other lesions of the cord. Arterial thrombosis is rather more common; it has been found in syphilis, multiple sclerosis, senility and perhaps bears some relation to acute disseminated myelitis. It is often impossible to decide whether an area of softening is ischemic (myelomalacia) or inflammatory (myelitis).

III. Hemorrhage (Hematomyelia).—Etiology and Pathology.—Most cases of this rare lesion occur in men between 20 and 40 years of age. Trauma causes 90 per cent. (*a*) Capillary hemorrhages occur in foci of softening, inflammation, etc. (*b*) The larger hemorrhagic focus from trauma, muscular effort, caisson-disease, etc., compresses and destroys the cord substance; it rarely exceeds the size of a hazel-nut but may extend the entire length of the cord (tubal form of Levier), following exactly the less resistant gray matter, while the firmer pyramidal tracts usually resist its lateral diffusion; if it invades the white matter, hemorrhage usually occurs in the posterior columns; it occurs mostly in the cervical cord; all large non-traumatic hemorrhages occur in the gray matter. The nervous tissue is disorganized by large hemorrhages, which result eventually in a pigmented apoplectic scar or cyst.

Symptoms.—(*a*) In the accessory form there is bleeding into a tumor, cavity, softening or inflammation; punctate hemorrhage may accompany purpura, stasis or convulsive disorders, and few or no symptoms develop.

(*b*) The traumatic form is less important in spinal fracture than concussion (see TRAUMA); it may develop during dystocia or fetal extraction. (*c*) The *spontaneous* form is rarer than it is in the brain, because the cord is firmer, subject to lower blood-pressure, and most rarely the seat of miliary aneurysm; it results from sudden exertion in man (and more often in the horse), repeatd coitus or rarely arteriosclerosis.

The onset is sudden, without prodromes or loss of consciousness. Local symptoms must vary with the structures involved and their level; they embrace paraplegia, monoplegia, the Brown-Séquard syndrome, paralysis with or without atrophy or spasticity, anesthesia either total or partial; pain at the level of the lesion; less often eccentric pain, girdle sensation and muscular rigidity. In general the reflexes are increased in small and abolished in large hemorrhages. The symptoms advance rapidly and may be aggravated by secondary myelitis. Late death from cystitis or decubitus is more common than early death. Recovery is rare compared with cerebral apoplexy; it occurs most often in the punctate type; it is rarely absolute.

Diagnosis.—The diagnosis is usually only probable, based on the sudden paresis and anesthesia and their rapid advance. For differentiation from *hematorrhachis*, see page 829. From *acute myelitis*, differentiation is often uncertain and of only academic interest; in myelitis and poliomyelitis elevation of temperature is distinctive.

IV. Caisson or Divers' Paralysis.—According to Pol and Watelle (1854), this occurs in workers subject to pressure of at least 2, and usually 3 or 4, atmospheres. Symptoms appear when the individual comes suddenly into the open air. They include transient pain in the ears, sometimes deafness, *vertigo*, headache and *pains in the limbs*, joints and epigastrium; in 35 per cent. there is *paraparesis* or paraplegia, sometimes with anesthesia, and usually with urinary retention; much less often there is hemiplegia or involvement of both arms; epistaxis, mental symptoms; loss of consciousness and symptoms of brain-pressure, as vomiting or slowed pulse. Symptoms are not severe or are absent if the interval of work in the caisson does not exceed five hours.

Pathology and Pathogenesis.—Hoppe-Seyler (1885) and Bert found that lessened atmospheric pressure liberated nitrogen in the blood and tissues, which ruptured the vessels and tissues. In the first autopsy v. Leyden found clots particularly in the posterior and lateral columns, surrounded by round cells (reactive disseminated myelitis); most changes were in the less compact, dorsal cord; secondary degeneration is found and changes in the gray substance from hemorrhage; Catsaris saw gas bubbles in the blood. Under moderate pressure the gas is given off to the lungs, while in extreme instances it is given off to the tissues (air embolism).

Prognosis.—A few subjects die at once; 3–16 per cent. die from cystitis, as in myelitis; about 50 per cent. recover completely and the balance partially, with spastic paresis.

Treatment.—The return to normal atmospheric pressure should be made gradually; recompression is successfully employed. Ergot and strychnine are recommended.

V. Brown-Séquard's Paralysis.—According to Brown-Séquard's original statement (1850), *on the side of section* (cervical cord) there occur (*a*) paralysis of voluntary motion, muscle sensibility and vasomotor tonus; (*b*) hyperesthesia of trunk and limbs, to touch, pain, heat and cold; (*c*) vasomotor paralysis of face and eyes (higher temperature, narrow pupils and contracture of certain facial muscles). *On the contralateral* side there is anesthesia of all varieties of sensation, except muscle sensibility.

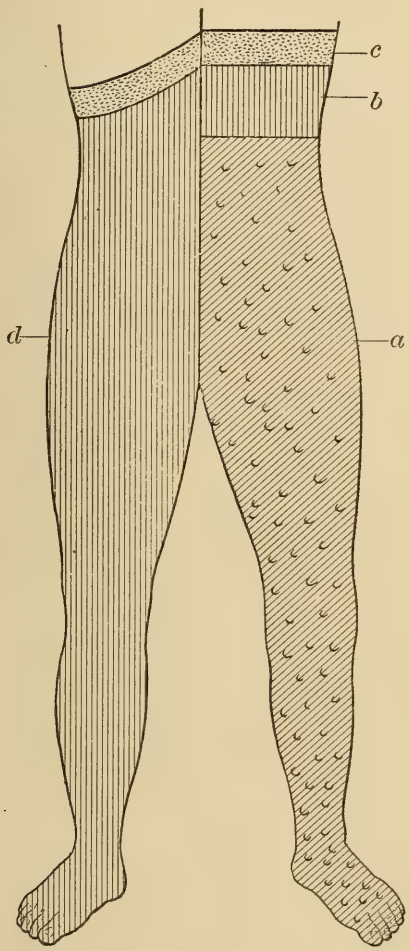


FIG. 72.—Brown-Séquard's paralysis from a left-sided focus: *a*, vasomotor and motor paralysis; *b* and *d*, cutaneous anesthesia; *c*, hyperesthetic zone. (Erb.)

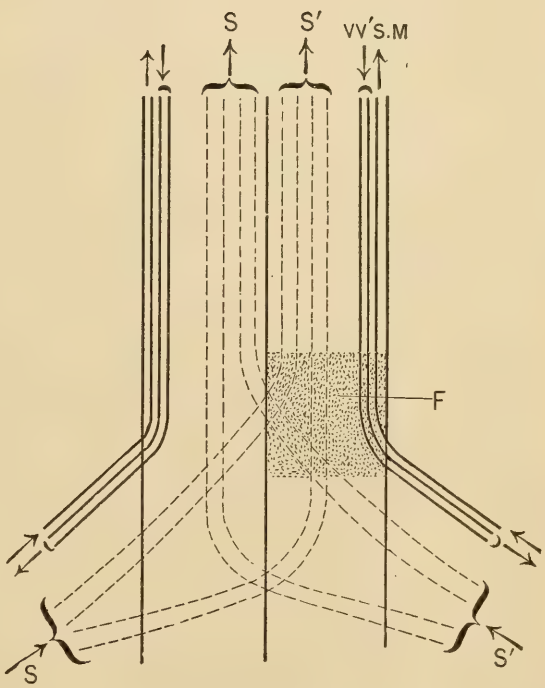


FIG. 73.—(Combined from Brown-Séquard.) *F*, focus in left side of cord; *V*, pyramidal fibers having crossed in medulla; *V'*, vasomotor fibers, not crossing in cord; *S.M*, fibers for muscle sense, not crossing in cord; *S S S' S'*, other sensory fibers crossing in the cord. The focus explains the classical signs and also how few of the crossed sensory fibers to the left side are involved (merely a zone of anesthesia), while all sensory fibers to the right side are severed (hemianesthesia).

Symptoms.—Exact *hemisection* of the *cervical cord* produces a *spinal hemiplegia* on the side of the section, but in injuries and experiments the trauma is rarely complete or strictly unilateral. (In some cases decussation of the uncrossed pyramidal tracts may occur in the cord lower down than the usual crossing point in the medulla (Flechsig) and spinal hemiplegia with contralateral monoplegia may result from unilateral lesion.) The characteristic clinical type is the *mid-dorsal* lesion, but in cervical section or in pathological foci, paresis of the arm with paralysis of the leg is more usual, because the cervical motor tracts

are less compact than they are in the lower cord; paralysis of the leg may be incomplete, while that of the arm is complete, owing to the escape of fibers for the leg which cross lower down in the cord. The paralysis often decreases with surprising rapidity, and if the anterior cells are intact it gradually becomes simple weakness. There is an inactivity atrophy of the muscles without abolition of faradic irritability and without reaction of degeneration. Respiration is rarely affected. Involvement of the thoracic and abdominal muscles usually indicates a bilateral lesion. Swelling and edema in the paralyzed members and enlarged joints have been observed. The *deep reflexes* are exaggerated on the side of lesion; they are abolished in a sudden total transverse lesion of the lower cervical or upper dorsal cord. The skin reflexes are abolished or decreased on the side of paralysis and on the opposite anesthetic side they may be normal, absent or increased.

Hyperesthesia occurs on the paralyzed side and also as a zone above the anesthetic area, near the level of the lesion. Hyperesthesia or hyperalgesia concerns tactile, pain and heat and cold sensation and their absence infers incomplete section. It usually disappears rapidly. The fibers involved lie in the lateral tracts.

The *zonular anesthesia* on the side of the injury and of motor paralysis is explained since the lesion involves not only the sensory fibers (which are ready to decussate to the other side and produce the crossed hemianesthesia), but also sensory fibers from the contralateral side, which have just decussated to the side of section. Abolition of *muscle sensation* on the side of the lesion is in accord with the uncrossed course of the muscle sense fibers in Burdach's column, entering Goll's column higher up. Muscle sense is preserved on the side opposite to the lesion. Vasomotor paralysis occurs on the side of section, as the vasomotor fibers course in the anterolateral columns. "*Sensation is affected on the opposite side,* but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate but occurs somewhat above the entrance of the nerve" (Gowers). Complete crossed hemianesthesia occurs in 60 per cent. and partial anesthesia (analgesia and thermoanesthesia) in 40 per cent. of cases.

Prognosis.—Almost complete restitution to normal is possible; it is not due to reëstablishment of conduction in the severed tracts, in which ascending and descending degeneration occurs, but to assumption of function by the uninjured side of the cord. The prognosis and *treatment* of Brown-Séquard's paralysis vary with the etiology of this symptomatic syndrome, which may be syphilis, tumor, sclerosis, myelitis, tuberculosis, trauma, hematomyelia, fracture or luxation.

INFLAMMATION OF THE CORD.

Myelitis, inflammation of the cord, was described by Oliver and Abercrombie a century ago; the term has been made to cover many affections not inflammatory, as compression paralysis. Oppenheim and Marie dispute the idea that myelitis is a frequent affection.

I. Acute Myelitis.—Etiology.—The affection occurs largely in males between ten and forty years of age.

1. **Acute Infections.**—Experimental myelitis has been produced by the B. pyocyaneus, B. diphtheriæ, B. coli, B. typhosus, strepto- and staphylococci, etc. It has followed epidemic meningitis, variola, measles, rheumatism and other infections, caused (*a*) by the germs themselves or secondary infection (focal myelitis resulting) or (*b*) by their toxins (disseminated myelitis resulting).

2. **Intoxications.**—These include poisoning by alcohol, ergot and lead and perhaps auto-intoxications, as in cancer or severe anemia.

All other causes are doubtful; trauma or compression by caries or cancer causes softening or hemorrhage, to which infection may be added; exposure to cold and dampness reduces the physiological resistance, thus indirectly favoring bacterial localization; syphilis (*q. v.*) may initiate meningomyelitis, but syphilitic myelitis is usually ischemic softening from endarteritis.

Pathology.—On *macroscopic examination* we find redness and swelling; on cross-section, blurring of the cord tissues and decreased consistency, even to fluidity; "red softening" (hemorrhagic myelitis) and in older cases "yellow softening" from fatty change, or "white softening." After hardening in Müller's solution, the normal tissue appears green and the diseased, yellow. In the ultimate stages, atrophy, cyst formation, induration and very rarely abscesses are found. *Microscopically* there are, *in fresh cases*, degenerated, swollen axis-cylinders; disintegrating medullary sheaths from which myelin drops form; granule corpuscles (leukocytes or vessel endothelial cells); extravasated red and white cells; turgid vessels the nuclei of which proliferate; choking of the perivascular lymph spaces with cells; degeneration, blurring and vacuole formation of the nerve cells, the processes of which shrink and the nuclei vanish; amorphous deposits of coagulated albumin in and about the cells; in some cases the parenchymatous far exceeds the interstitial alteration (parenchymatous myelitis, or better, parenchymatous degeneration). The gray matter (poliomyelitis) is involved much more often than the white (leukomyelitis). In *older cases* there are corpora amylacea, empty spaces corresponding to destroyed nerve fibers; Deiters's large "spider" cells with many processes; and interstitial proliferation is substituted for the destroyed tissue. In rare instances bacteria are found, but their toxins are more important. The microscope differentiates between inflammation and softening. The *degree and the extent* of inflammation vary greatly; *transverse myelitis* may destroy an entire cross-section; or *disseminated* foci may involve at random, various structures at different levels. *Meningomyelitis* (perimyelitis, annular or cortical myelitis) chiefly affects the periphery of the cord, whence concentric invasion along the septa may split up the cord structures, particularly in the syphilitic form. *Poliomyelitis* (inflammation of the gray substance) is a type of myelitis classified by itself. Degeneration and inflammation of the motor and sensory *roots* may follow myelitis in the cervical or lumbar enlargement.

Symptoms.—The symptoms vary with the extent, form and localization. The localized form, limited to a small segment, is called *transverse*, while the diffused form is known as *disseminated myelitis*.

Acute transverse myelitis is the prototype. The cord symptoms are usually first in time and always first in importance and constitutional symptoms, as fever, chills, malaise and in children convulsions sometimes precede but usually attend the cord symptoms. Symptoms resulting from *involvement of the cord* are (*a*) *paralysis*, which is usually early, rapid but not apoplectic in onset and is prominent, paraplegic, progressive, flaccid and complete. The flexors are usually weaker than the extensors. The toes alone may be moved; twitching of the paretic muscles or clonic muscular spasms are sometimes present. The paralysis is often preceded for a short time by numbness, or moderate darting pains in the limbs, joints or back. The paralyzed limbs show a transient rise of temperature, followed by somewhat subnormal temperature. The limbs are often dry and may not sweat even after injection of pilocarpin. (*b*) *Sensation* is frequently involved. The initial tingling pain has been considered. Anesthesia may be complete but more often incomplete (hypesthesia) and rarely partial. It is higher anteriorly than posteriorly; a zone of hyperesthesia or "girdle pain" marks the upper level, caused by early irritation or late cicatrization. Severe pain suggests vertebral or root disease rather than myelitis; gastric crises like those of tabes, dysesthesia, *i. e.*, diffuse vibrating sensations over the entire limb from local stimulation, and ataxia (usually obscured by the paraplegia) are much less common. (*c*) At the onset all *reflexes* may be lost, but they are usually increased below the focus, from lessening of cerebral inhibition (lateral pyramidal tracts). (See table.) (*d*) The *sphincters* are affected almost constantly and early; ischuria or urinary retention and later incontinence are usual; when the bladder is full it may dribble from relaxation of the sphincter, known as ischuria paradoxa or retention with incontinence; the patient may or may not be conscious of the retention, depending on whether the afferent sensory fibers are involved or not. The urine is at first alkaline and later ammoniacal on the advent of cystitis. The sphincter ani is spasmodically contracted or paralyzed (see table); the paresis of the bowel and abdomen are factors in constipation. (*e*) *Trophic changes.* Muscular atrophy and the reaction of degeneration are caused by disease in the anterior horns and the muscles also waste from inactivity. The skin is usually dry, sometimes glossy or vesiculated. The joints are sometimes swollen and the limbs edematous. Bed-sores are frequent over the sacrum, trochanters, heels, between or even over the knees, from the bed-covers; they result from neglect or in lumbar myelitis from trophic disturbance (disease in the gray matter or posterior horns). The same factors cause cystitis, ulceration in the bladder or urethra and the tendency to cellulitis.

Course and Prognosis.—The onset covers a few hours (the apoplectic form), or several days to a week (the subacute form). *Four stages* are described: the acute *initial stage;* the *advancing degeneration;* the *restitution;* and the *stationary* stage. Complete paraplegia and anesthesia may persist, but more often sensation returns after some weeks, while

movement returns slowly, incompletely and usually with some residual spastic paraplegia. Contractures may develop, especially in the adductors and knee flexors, which indicates profound disease. Contractures, muscular contractions and increased reflexes are caused by secondary

CORD SYMPTOMS GROUPED ACCORDING TO LOCALIZATION.

In order of frequency.	*Dorsal myelitis* most frequent.	Then *lumbar myelitis.*	Lastly, *cervical myelitis.*
Paralysis.	Trunk, back, intercostal and abdominal muscles (favoring mucous stagnation in lungs). Arms normal. Paraplegia, first flaccid then spastic (lateral columns involved).	Trunk and arms normal. Paraplegia flaccid throughout (ant. horns involved).	Neck muscles, diaphragm, arms, trunk and legs paralyzed. Typically but rarely, atrophic flaccid paralysis of arms (ant. horns involved), with flaccid, later spastic paralysis of legs (lat. columns involved).
Atrophy.	In trunk; difficult to elicit. In legs; only from inaction.	None in arms or trunk. Atrophy in legs (ant. horns involved).	Atrophy of neck muscles if high lesion (rare). Usually of arms only (ant. horns involved at level of lesion). In legs only from inaction.
Reaction of degeneration.	Present in trunk muscles; absent in legs (or only quantitative reduction).	Present in legs or in mild cases quantitative reduction.	Present in arms (rarely in neck).
Sensation.	Girdle pain and hyperesthesia, between ensiform and navel. Anesthesia corresponding with motor paralysis.	Pain in loins or legs; hyperesthesia in loins. Anesthesia of of legs.	Pain and hyperesthesia in arm nerves (with highest localization); anesthesia in arms (lower localization); and the trunk and legs.
Reflexes.	Superficial reflexes, initial loss, rapid return and increase.	Lost.	Superficial reflexes; initial loss, rapid return and increase.
	Deep reflexes, initial loss, slow return and increase.	Lost.	Deep reflexes; initial loss, slow return and increase.
Sphincters.	Bladder, initial retention, later intermittent overflow or incontinence. Cystitis common.	Incontinence from beginning (sphincter paralysis).	Same as in dorsal myelitis.
	Bowels; usually spasm of sphincter ani; constipation.	Incontinence (sphincter paralysis) disguised, perhaps, by constipation.	Same as in dorsal myelitis.
Trophic changes, etc.	Decubitus from neglect, etc.	Decubitus, cystitis, etc., from neglect and trophic alteration.	As in dorsal lesions. (Occasionally very high temperature.)
Pupils.	Klumpke's paralysis of pupil (if in upper dorsal).	Absent.	Paralysis of pupil (lowest cervical region). Optic neuritis in isolated cases.
Priapism.	Often present and painful.	None. Impotence.	Priapism often present.
Mode of death.	Cystitis, decubitus.	Decubitus, cystitis.	Medulla symptoms; involvement of phrenic nerve with early death.

degeneration. The immediate outlook is uncertain and depends (*a*) on the etiology, the prognosis being best in acute infections; (*b*) on the intensity of inflammation; (*c*) on its transverse or vertical dimensions, and (*d*) on the rapidity or tardiness of improvement. Complete recovery in circumscribed forms is possible, since indirect symptoms, due to collateral edema, may disappear. Death results from extension to the medulla, phrenic nerve phenomena, and sepsis following cystitis, pyelitis or pyelonephritis.

Diagnosis.—The early onset of retention of urine, paraplegia and anesthesia, is especially significant after acute infections. The upper level of the myelitis corresponds to that of the paralysis or anesthesia. The lower level may be determined by the condition of the reflex centres. *Anterior poliomyelitis* (*q. v.*) does not involve sensation as it rarely extends back of the anterior horns. In compression by *caries* or cancer local vertebral and root symptoms usually precede cord symptoms. The so-called *syphilitic myelitis* (myelomalacia) begins acutely without pain and is rarely relieved by antisyphilitic remedies. *Hemorrhage* in the cord produces most sudden symptoms and acute spinal pain without prodromes. For differentiation from *Landry's paralysis*, see page 842, and from *multiple neuritis*, see page 872. *Meningeal hemorrhage* and meningitis produce unmistakable root symptoms. Of *abscess of the cord*, few more than a dozen cases are reported; its symptoms are those of myelitis plus meningeal symptoms. *Hysterical paraplegia* is characterized by motiveless variability of the symptoms, little myotatic irritability, little extensor spasm (and then both legs move together when one spasmodically extended leg is lifted); rarely by incontinence of urine or feces, no trophic changes, no cystitis, no reaction of degeneration and no decubitus.

In the *chronic stage* the diagnosis is usually provisional and pachymeningitis, tumor and multiple sclerosis should be considered.

Treatment.—In the *acute stage* the indications are (*a*) absolute rest in bed for at least two weeks, keeping the patient on one side or on the face. Blisters should be avoided lest they develop decubitus. (*b*) Gowers recommended spt. etheris nitrosi with digitalis to equalize the circulation and promote diuresis. (*c*) Mercury and iodides are usually without effect in syphilitic myelitis (softening), although their use is rational. (*d*) Care of the skin. Though bed-sores may be trophic, care greatly reduces their frequency and extension. Pressure and heat should be avoided by change of posture, rubber cushions, soft, thick pads of cotton or the water bed. As in typhoid, the use of two beds is very beneficial and the sheets should be stretched smooth. Alcohol rubs are antiseptic and harden the skin. Moisture and filth are most dangerous; the bowels should be irrigated if they move involuntarily; incontinence of urine is more dangerous, especially in women; hard urinals, if left between the legs, may cause equal damage; the urine should be gathered in bags of oiled silk filled with cotton; in women mild antiseptic gauze and impalpable boric acid should be disposed about the genitalia. (*e*) Cystitis develops in most cases in which the catheter is used; residual urine is easily infected, infection ascending the dribbling, patent urethra;

the urethra should be irrigated before the absolutely clean catheter is introduced; lavage with Thiersch's solution in cystitis with retention or incontinence and grs. v–x, each of urotropin and salol, t. i. d., are indicated.

In the *subacute stage*, tonics, massage after four weeks, mild faradism of the bladder and rectum are indicated. Contractures are modified by warm baths and massage. Hot baths are dangerous.

II. Acute Multiple Disseminated Myelitis.—This relatively infrequent affection is subacute in onset; foci develop irregularly, together or successively in different parts of the cord or perhaps of the brain (*disseminated encephalomyelitis*), with acute ataxia, scanning speech, dysarthria, tremor, nystagmus, optic neuritis, spasticity or weakness of the limbs. Sensation and the sphincters are usually not affected. The clinical picture is necessarily irregular, because the inflammation may occur anywhere; von Leyden distinguishes the atactic and the paraplegic forms. Myelitis which spreads after the first two or three days is of the disseminated type; once thought very fatal, numbers of recoveries have been reported. Treatment is as in the acute transverse variety.

III. Chronic Myelitis.—This may be the stationary stage of acute myelitis or in very rare cases myelitis may be chronic from the beginning, *i. e.*, a gradual onset and slow progression. Many deny the existence of this form and classify it under the paraplegic stage, multiple sclerosis or chronic system disease.

IV. Subacute and Chronic Poliomyelitis.—**Etiology.**—Metallic poisons and syphilis are important factors. *Pathologically*, two main types are distinguished; in the larger group there is no inflammation but a primary atrophy of the ganglionic cells, which heightens its resemblance to spinal progressive muscular atrophy; in a smaller group of cases there is early perivascular exudation in the anterior horn and later sclerosis, wherein it resembles acute poliomyelitis. The anterior roots are involved and in most cases there is degeneration in both pyramidal tracts, possibly also in the posterior columns or roots, in Clarke's columns or rarely in the medulla. The *course* is gradual; one extremity becomes slowly affected (in one to three years), sometimes with extension to others after months or years. There are paresis with atrophy, fibrillary tremors, partial or complete reaction of degeneration, decrease or absence of the reflexes and normal sensation. The ultimate outcome is unfavorable; if recovery occurs, neuritis, not poliomyelitis, is the actual lesion. *Differentiation:* (*a*) Multiple neuritis (see page 872); (*b*) from *progressive spinal muscular atrophy*, even an anatomical differentiation is not easily made. As a rule, in progressive muscular atrophy, atrophy precedes and exceeds the muscular weakness, fiber after fiber of muscle very slowly wastes, the atrophy is especially marked in the distal parts, and the reaction of degeneration is less complete. In chronic poliomyelitis, an outright atrophy (*atrophie en masse*) rapidly follows paralysis; it occurs more often in the proximal parts, as in the shoulder, and the reaction of degeneration is marked. (*c*) In amyotrophic lateral sclerosis the paralysis is spastic with increased reflexes, contractures and often with bulbar symptoms. *Treatment* is that of the chronic stage of acute poliomyelitis.

V. Landry's Paralysis.—Acute ascending paralysis was described by Landry and Kussmaul in 1859. Cuvier died of this disease in 1832. Acute ascending paralysis is a symptom-complex rather than a disease and may be conveniently classified under inflammatory affections of the cord. The *etiology* is vague. The affection occurs mostly between twenty and forty years of age and three times more frequently in males. It has followed acute infections, as diphtheria, typhoid or influenza and also chronic infections.

Pathology.—In many cases no anatomical findings existed, upon which Westphal (1876) founded his three criteria: (i) that the disease is a progressive, ascending fatal affection, (ii) with normal electrical reaction and (iii) negative findings at autopsy. Recent cases have given definite though varying postmortem results, as structureless exudation in the cord, disseminated foci of inflammation in the cord, medulla and pons, changes in the pyramidal tracts, anterior horns and in the spinal or cerebral peripheral nerves. Two forms are distinguished: (*a*) the *bulbar or medullary form,* in which sensation and electrical reactions are normal, and (*b*) the *peripheral neuritic form,* with sensory and electrical alterations. The parenchymatous swelling of the liver, spleen, kidneys and lymphatics indicates a general infection or toxemia. Bacteriologically, an anthrax-like bacillus, the pneumococcus, typhoid bacillus, bacilli in all the peripheral nerves and streptococci have been found.

Symptoms.—The onset is acute with paralysis in one foot, then in the other, and in the legs, thighs, abdomen, back, arms, even in the face or the eyes, possibly with optic neuritis; the *paralysis is progressive and ascending;* when the medulla is invaded, dysarthria, dysphagia and paralysis of the tongue occur, finally with death from respiratory paralysis. The proximal parts of the limbs are more involved than the distal and some muscles may escape. Fever is usually absent; the sensorium, sensation, sphincters and electrical reaction are usually normal. There are no tremors, no twitchings, no convulsions, no contractures, no trophic disturbance. The reflexes are usually decreased or absent. In rare cases there may be partial reaction of degeneration (which usually has no time to develop), atrophy and moderate disturbance of sensation or the sphincters. The paralysis may be of the descending type, in which event the bulbar development is fatal before the paralysis in the legs is well developed.

Course and Prognosis.—The disease lasts two days to two weeks. Bulbar symptoms are ominous, but not necessarily fatal. The central form of paralysis is fatal and the peripheral form includes most of the recoveries. In rare cases, death may occur in a few hours or only after months. In still rarer cases of recovery, some chronic paralysis may remain. If recovery ensue, the paralysis regresses in an order inverse to that of development. A *diagnosis* is easily made, because of the ascending motor paralysis with loss of reflexes and without marked mental, sensory, sphincter or electrical alteration.

Treatment.—The treatment is that of myelitis or multiple neuritis. Strychnine and other stimulation are indicated if the bulbar symptoms are manifested. C. L. Greene reports a case which was kept alive by artificial respiration forty-one days after respiratory failure set in.

MULTIPLE SCLEROSIS.

Insular or disseminated sclerosis, *sclérose en plaques disseminées*, was described pathologically by Cruveilhier and Carswell (1838); Frerichs, in 1849, made the first diagnosis, and Charcot (and Vulpian) in 1863–64, gave a description of the typical case.

Etiology.—In fully 50 per cent. of the cases no cause is found. It occurs equally in either sex and 90 per cent. occur under the fortieth year; pseudosclerosis occurs after the fortieth year and is considered arteriosclerotic. Three main factors are thought to exist: (*a*) *Acute infections*, as typhoid, pneumonia, measles, smallpox, etc. (*b*) Chronic intoxications with alcohol, lead, etc. (*c*) The developmental theory (Strümpell).

Pathology.—Macroscopically, the *sclerotic foci* are seen as irregular grayish-red areas as large as a pea or walnut; they are multiple (numbering even over 100), distributed at random and largely *in the white substance* but also in the gray matter. Their distribution is *cerebrospinal*, in rarer cases in the cord alone, and most rarely in the brain only; the cortex is less frequently involved than the centrum ovale, central ganglia, corpus callosum and the undersurface of the pons and crus. The medulla is quite often, and the cerebellum rarely, involved. The white matter in the cord is more frequently affected than in the brain. *On section*, the focus is sunken and transparent, well demarked, firm, save in the freshest foci, and on hardening in Müller's solution, appears light green while the normal tissue is a darker green color. Microscopically, the *medullary sheaths* in the white substance are *thinner* or absent and though the *axis-cylinders* are somewhat wasted and at times varicose, their *remarkable persistence*, first noticed by Charcot, causes *the incomplete character of the symptoms* and the usual *absence of ascending and descending degenerations*. The ganglion cells suffer some slight atrophy and pigmentation. The foci show corpora amylacea, granule-bearing cells, increase of the fine-meshed neuroglia fibrils and nuclear proliferation. In contrast with the system-degenerations, the *primary change* is *interstitial* with secondary changes in the nervous tissue. The cerebral nerves suffer often, especially the optic nerve, in which proliferation of connective tissue is followed by loss of the medullary sheaths, while the axis-cylinders are unaffected. The nuclei, roots and nerve trunks of the other cranial nerves may suffer similar alteration. The spinal roots are not infrequently involved.

Symptoms.—The clinical picture is so variable that a definite description cannot be made to cover all cases. Charcot's description included the intention tremor, the nystagmus, scanning speech, motor weakness and rigidity and apoplectic insults.

1. Motor Symptoms.—These are the most important. (*a*) *Muscular weakness* is very common. Paresis is more frequent than paralysis, since some axis-cylinders remain intact. The movements are slow and fatigue rapidly follows their repetition, more so in isolated than synergistic movements. The paresis is most apparent in the legs, usual

in the arms and frequent in the eyes, head and speech. It sometimes does not develop until late, and is associated with increased reflexes and spasticity. The gait is spastic, scuffling and the feet stick to the ground; to this is sometimes added a cerebellar, atactic or paretic element. Muscular atrophy is rare. The electrical reactions are practically normal. (*b*) *Muscular rigidity, the most frequent finding,* is referred to disease of the lateral columns and is most developed in the legs. It increases with successive movements, each step becoming more stiff in walking. The spasticity may be so extreme and the muscles so hard that the limb cannot be moved. Contractures develop, which usually affect the adductors and extensors first and the flexors later and draw the heels up to the buttocks. Contractures often affect the muscles of the neck. (*c*) *Tremor* is one of the most striking phases of the disease, being present in 75 per cent. of cases. It is caused, as is the paresis, by the loss of the medullary sheaths which allows diffusion of nerve impulses, by foci in the pons or thalamus or by lack of coördination between antagonistic muscles. There is no tremor during rest, but it appears when voluntary efforts are attempted (intention tremor). The hand carrying a glass of water to the mouth shakes so much that all the water may be spilled by the rhythmic tremor, which possesses a wide range, and occurs five or six times to the second. It is apparent in the head when the patient sits, because the muscles of the neck are in constant action to support the head; it disappears in the recumbent posture. Tremor occurs in the face, larynx, trunk or legs on changing posture, as rising; in the fingers it produces handwriting which becomes more erratic with each word written; and in the thoracic muscles it causes a species of cog-wheel respiration. (*d*) *Nystagmus* occurs in 50 per cent. of cases. These short, jerking lateral movements of the eyes are most common on lateral or vertical movements. It is considered a tremor or an incoördination. (*e*) *The speech* is *scanning* (54 per cent.), *syllabic* or *staccato;* it is slow, monotonous, unmodulated and sometimes explosive, especially for the letters c, p, g and d. The speech muscles fatigue easily and the end of a sentence is often elided; the sounds are often nasal, interrupted by yawning inspiration or expiration. The cause of the peculiar speech may be tremor or ataxia from foci in the pons or medulla. (*f*) *Paresis of the eye muscles* (in 17 per cent.) affects the third or sixth nerve, convergence or associated movements. About 50 per cent. are nuclear. Transient diplopia, unequal pupils, hippus and strabismus are less common. (*g*) *Apoplectiform seizures* (in 1 per cent.) are marked by partial coma, increased pulse, high fever, aphasia and hemiplegia, which is usually fugitive and flaccid. In most cases negative pathological findings exist, but Leube and Leyden refer them to acute encephalomyelitis. Though usually hemiplegic, they may exceptionally produce paraplegia, hemianesthesia or ataxia, or monoplegia.

2. Sensory Symptoms.—Marked aberrations are less frequent (30 per cent.) because the sensory fibers are more resistant to disease. Subjectively, dull pains, formication and paroxysmal headache are frequent; lightning pains, crises, girdle sensation, trigeminal neuralgia, palpitation and dyspnea are very uncommon. Objectively, any species of sensation

may be involved. *Ataxia* referable to disease of the posterior columns or higher sensory tracts is marked in 50 per cent. and may produce a typical tumbling cerebellar gait.

3. Special Senses.—The optic nerve is affected in 45 per cent., usually as a *pallid partial atrophy* involving its temporal side, and less often as neuritis or atrophy with total blindness. The change is more conspicuous pathologically than clinically. It may be the first sign of the disease. In 60 per cent. of cases there is central scotoma, and at times narrowing of the field of vision.

4. Psychical Symptoms.—Mental impairment is common; the patient is complacent and the memory is weak. Compulsory laughing and weeping, partly without motive and partly emotional, probably should be referred to lesions in the medulla, pons or crus.

5. Bulbar Symptoms.—These may, in rare cases, resemble the symptoms of bulbar palsy, as dysphagia, anarthria and tongue atrophy.

6. Reflexes.—The patellars are exaggerated, there are ankle-clonus and Babinski's sign, showing disease of the pyramidal tracts. The abdominal reflexes are absent in 70 per cent. of cases.

7. Sphincters.—Lasting or severe symptoms in the bladder or rectum are uncommon; the bladder is often hypertonic and irritation is frequent.

8. Trophic Symptoms.—These are rare; they consist of skin eruptions, edema, sweating, chronic arthritis and erythromelalgia.

Course and Prognosis.—The *onset* is usually gradual; in most cases weakness in the legs appears first and for a long time is hard to interpret; it occurs without pronounced sensory symptoms, bladder disorder or atrophy. Sometimes the onset is acute, with loss of consciousness, transient hemiplegia, vertigo, headache, eye symptoms, vomiting and optic neuritis. Leg paralysis is followed by scanning speech, nystagmus and tremor. The symptoms remit and recur. Bramwell found that death occurred in less than five years in 29 per cent., in less than ten years in 66 per cent., and in less than fifteen years in 89 per cent. The terminal stage presents dementia, decubitus, sphincter paralysis, cystitis and septicopyemia.

Diagnosis.—The typical case is unmistakable, because of the cardinal symptoms: intention tremor; spastic paresis; nystagmus; scanning speech; ataxia; increased reflexes; optic atrophy; the slow, uneven course with remissions and recurrences; the variability of the symptoms; apoplectic insults; and moderate involvement of the mentality, sensation, bladder and rectum.

Differentiation.—Transient hemiplegia may suggest hemorrhage, syphilitic thrombosis, embolism, paretic dementia or tumor (*q. v.*). In *paralysis agitans* the fine tremor of the hands and fingers (not of the large joints, and much less of the head) which is not increased by movement, its persistence during rest, the mask-like facies, festinating speech, peculiar attitude, gait, propulsion, retropulsion, muscular rigidity without increased reflexes and the advanced age are clear and characteristic points.

Multiple Sclerosis.	*vs.*	Syphilis.
Occurs in the medullary substance.		Meningomyelitis and root symptoms.
A focal disease.		Diffuse.
Nystagmus, scanning speech, tremor.		More frequent insults, hemiplegia, etc.; speech may be slow in pons lesions but is not scanning.
Partial optic atrophy occurs.		Amaurosis, choked disk or neuritis frequent.
Mydriasis 1 per cent., myosis 4 per cent.		Mydriasis is very common.
Argyll-Robertson pupil very rare.		Argyll-Robertson pupil frequent.

Hysteria more than any other disease simulates early multiple sclerosis; the two affections often occur together and in both the symptoms are changeable. In hysteria sensory symptoms are much more prominent, there are often limitation of the visual fields, monocular diplopia, contractures and convulsions, but there is no optic atrophy, nystagmus, scanning speech, or intention tremor. The movements in hysteria are often accompanied by contraction of the antagonist muscles, a most valuable diagnostic aid. In extremely rare cases, described first by Westphal as *pseudosclerosis,* the autopsy shows nothing; the tremor affects the arms chiefly; there is no ataxia or nystagmus; the facies is somewhat rigid and outbursts of anger are frequent; syphilis is a possible cause. *Birth palsies* accompanied by jerky movements, incoördination and even scanning speech are excluded by the history.

Treatment.—Drugs, hydro- and electrotherapeutic measures are futile. The patient should live in a warm climate during the winter months.

SYRINGOMYELIA.

The term refers to cavities in the cord, and was first employed by Ollivier (1824). The affection was first described by Morgagni. Syringomyelia must be distinguished from hydromyelus, which is a dilatation of the central canal due to developmental anomalies; it is usually an accidental finding at necropsy, or if symptoms are present they are those of syringomyelia.

Etiology.—Sixty-three per cent. of cases occur between the tenth and thirtieth years and 70 per cent. in males.

Pathology.—1. Gross Pathology.—The cord is often lax, sometimes fluctuating or flat. The *cavity* is usually localized but may extend from the medulla to the filum terminale; it is most often found in the lower cervical or upper dorsal region; in size it ranges from a narrow slit to a cavity as wide as the cord itself; it may communicate with the central canal, may be multilocular and diverticula may be found. Its wall is smooth, sometimes pigmented from rupture of the new-formed blood-vessels. The cord usually suffers sclerosis in the form of tumor-like, *asymmetrical neurogliar proliferations,* which later break down and form secondary cavities. The posterior horns and columns are often affected, the anterior horns and columns less frequently and the lateral columns very rarely.

2. Minute Pathology.—The essential finding is *the neurogliar hyperplasia,* probably due to some congenital anomaly, which also explains its frequent association with hydromyelus; the overgrowth is rich in

fibers, whereas the ordinary glioma (gliomatosis) especially abounds in cells; syringomyelia and gliomatosis may coexist. This proliferation contains large, sharply nucleated cells, and a few epithelial cells, is firm as a whole, but somewhat softer toward the posterior horns, is often hyaline nearest the cavity from compression and abounds in bloodvessels. The cavity is usually lined with cylindrical epithelium like that of the central canal; its contents resemble cerebrospinal fluid, but is occasionally blood tinged or less often gelatinous, mucoid or milky. The nerve fibers are not much involved. Secondary degeneration may occur in the posterior and less frequently in the lateral columns.

Symptoms.—There are *three cardinal symptoms.*

1. Anesthesia.—*Partial or dissociated anesthesia* occurs in the typical case, in which the sensation of touch, pressure and locality is preserved while heat, cold and pain are not recognized. *Thermo-anesthesia* usually concerns perception of both heat and cold, sometimes of only one of them, or one in one place and the other in another; *analgesia* is frequently preceded by hyperalgesia; it usually corresponds with the thermo-anesthesia. Thermo-anesthesia and analgesia begin in the hands and fingers and are later found on the upper trunk; sometimes the entire trunk, legs, mucous membranes and face may be affected, depending on the location of the cavity. The patient may suffer burns or injuries in these parts without pain. Dissociated anesthesia is most probably caused by disturbed conduction in the gray substance due to neurogliar proliferation and cavity formation. Anesthesia may involve all varieties of sensation. Subjective sensory disturbances include paresthesia and burning, tabes-like pain in the neck, arms or trunk and sometimes in the joints, spine or legs.

2. Muscular Atrophy.—Muscular atrophy is due to involvement of the anterior horn and affects the arms mostly, because the disease is largely in the lower cervical and upper dorsal cord. It is slow in onset, more marked than the attendant flaccid paresis, degenerative and often observed with fibrillary muscular contractions and increased myotatic irritability. It usually involves the small muscles of the hand, as the interossei, produces flattening of the thenar and hypothenar eminences and the *claw-like hand,* as seen in the Duchenne-Aran type. The radial supply is first affected, then the ulnar and median. Sometimes the scapulohumeral type is observed, especially with bulbar symptoms. The muscles of the trunk, and much less often of the legs, may be affected. From cervical compression of the pyramidal tracts, paraplegia results, with increased reflexes and spasticity, in 12 per cent. of cases. Other motor symptoms occasionally occur, as tremor, contractures, spasms, opisthotonos or choreiform movements. The gait is normal, spastic or paretic.

3. Trophic Symptoms.—(*a*) Phlegmons and *felons* develop, particularly on the fingers. They are painless, chronic, often recurrent and lead to necrosis of the bone, ankylosis, deformity or mutilation. (*b*) The *joints* suffer in over 10 per cent., especially in males (75 per cent.), probably due to loss of articular sensation (whereby the joints are used inappropriately) or to lack of the nutritional reflex. These arthropathies are of

the hypertrophic form, with thickening and bony formation, like arthritis deformans, or of the atrophic form with relaxation of ligaments or flail joint. There is little exudation, no fever, no pain. They are similar to the tabetic joints but are more chronic and more often involve the upper extremities (shoulder 32 per cent., elbow 28, hand 16, hip 8, ankle 8, knee 6 per cent.). (*c*) *Painless bone necroses* and spontaneous fractures may occur late in the disease. Moderate scoliosis, usually confined to the dorsal spine, sometimes with kyphosis, is due to trophic vertebral changes or atrophy of the back muscles; perforating ulcer, thick, friable nails and clubbed fingers are less common. Morvan (1883) described a *paresie analgesique avec panaris*, endemic in Brittany, which is clearly syringomyelia. "Morvan's disease" is marked by trophic disorders, excessive or deficient secretion of sweat, edema on the dorsum of the hands, erythema, urticaria, local asphyxia and herpes zoster.

4. Other Symptoms.—(*a*) The skin *reflexes* are usually normal; the abdominal, cremasteric and plantar reflexes are sometimes increased; the tendon jerks are often decreased in the upper and increased in the lower extremities. (*b*) The *sphincters* are involved late in the disease. Retention of urine and constipation are more frequent than incontinence. (*c*) *Bulbar or pontine symptoms* are not infrequent, are usually unilateral and appear in the later stages. They include facial hemiatrophy or paresis, paresis of the tongue with atrophy, dysphagia, paresis of the palate (nasal speech), paralysis of the vocal cords, spinal accessory nerve (trapezius paralysis) and trigeminus (sensory symptoms), eye paralyses (abducens), unequal pupils, nystagmus, tinnitus, salivation, apoplectiform attacks, polyuria, glycosuria and vagus symptoms (disturbed pulse or respiration, vomiting, and singultus).

Diagnosis.—A positive diagnosis can be made from the three cardinal symptoms: (1) the dissociated anesthesia (thermo-anesthesia and analgesia with practically normal tactile sensation) is the earliest and most constant sign. Grasset collected cases of twenty or more nervous diseases (functional, traumatic, neoplastic, specific) in which this sensory dissociation has been found; but it remains highly distinctive in conjunction with (2) *muscular atrophy* of the *upper extremities* with paresis and (3) *trophic disorders, e. g., felons, arthropathies* and scoliosis; to which may be added the spastic paresis of the legs and bladder symptoms.

Differentiation.—*Progressive spinal muscular atrophy* and *amyotrophic lateral sclerosis* present no anesthesia, no trophic disturbance and bulbar symptoms are bilateral. *Juvenile muscular dystrophy* may be simulated in the scapulohumeral type of syringomyelia. In *peripheral neuritis* all varieties of sensation are disturbed; the paralysis is peripheral, not spinal or segmental, and there is the reaction of degeneration; neuritis of the upper brachial plexus produces paralysis of the shoulder, arm and supinator longus, a combination not found in syringomyelia. *Focal disease* of the anterior and posterior gray substance, as multiple sclerosis, hemorrhage or myelitis may simulate syringomyelia, but their course is less chronic and there is less trophic disorder. The root irritation of *tumor* of the cord is lacking. Located in the dorsolumbar region, syringomyelia may closely simulate *tabes*, from the lancinating pains,

crises and ataxia, but the Argyll-Robertson pupil is lacking and the sensory impairment is typical. In *leprosy,* anesthesia is peripheral, not spinal, in type; the Henssen bacillus is often found.

Course and Prognosis.—The course is progressive for years or decades. When recognized, the affection is hopelessly advanced. Death occurs from cystitis, septicemia and bulbar complications.

Treatment.—Treatment is unavailing. The danger of self-injury, as a result of the peculiar anesthesia, should be borne in mind.

SYSTEM DISEASES.

This small group of very important diseases involves *structures or systems having a common function,* thus differing from haphazard disease, as myelitis or multiple sclerosis. A *primary slow degeneration of the nerve elements* is followed by *secondary proliferation of connective tissue.* In the obscure pathogenesis of these degenerations *hereditary weakness* causing nutritional failure at a certain age (familial form) and the *selective action of toxemia* on certain nervous structures have been thought causative.

I. System Disease of the Sensory Tract.

The sensory type is locomotor ataxia in which the peripheral sensory neurone is affected, in the posterior roots and columns, while the motor types are spastic paraplegia, involving the upper motor neurone (pyramidal tracts) and muscular atrophy due to lesions in the lower neurone (anterior horns and roots).

Tabes Dorsalis, Locomotor Ataxia.—Definition.—A disease characterized (*a*) etiologically by syphilis; (*b*) pathologically by lesions in the posterior spinal roots, posterior columns and nerves; and (*c*) clinically by ataxia, Argyll-Robertson pupil, pains, defective sensation, loss of the tendon reflexes and trophic disturbances.

It is the best known and most frequent chronic disease of the cord. Todd, in 1847, gave the first accurate account, eliminated paralysis, and found the posterior columns diseased at autopsy. Stanley first (1840) referred the affection to the posterior columns and peripheral sensory nerves. Türck first examined the tissues microscopically and Romberg (1851), and Duchenne (1858), popularized the disease; it is still known in France as Duchenne's disease.

Etiology.—*Syphilis is the sole cause.* The following etiological data are those of syphilis; *age:* 50 per cent. in the thirtieth to fortieth year; 25 per cent. in the fortieth to fiftieth year; less than 25 per cent. in the twentieth to thirtieth year (Gowers); of 47 juvenile cases collected by Hirtz, the parents in 13 instances had syphilis, tabes or dementia. *Sex:* as in syphilis and paretic dementia, 10 cases occur in males to 1 in females. Like syphilis, tabes is sometimes familial. It prevails largely in cities and among the higher classes ("syphilization and civilization"). It is uncommon in the negro.

Pathology.—The *gross changes* are visible to the unaided eye; the posterior columns are smaller, outwardly flattened, gray from loss of the medullary sheaths and translucent from secondary increase of connective tissue. The pia may be secondarily opaque and thickened. The degenerated areas are sunken on section. The posterior horns and roots are atrophied and grayish-red. As to *minute changes*, there is a very *slow degeneration of the peripheral sensory neurone*, its fibers and processes of the spinal ganglia cells. These processes run in two directions: to the periphery and to the posterior roots. (*a*) The *peripheral spinal sensory nerves* are often degenerated, less in large trunks (sciatic or crural nerves) than in the peripheral filaments of the skin, joints and muscles which are important in the sensory, trophic and atactic symptoms of tabes; their white substance wastes and then their axis-cylinders. The trophic centres for these fibers are largely in the root ganglion. (*b*) The *fibers running from the ganglia to the posterior roots* are more early, regularly and obviously changed. The posterior root fibers run (i) to the posterior columns, whence there occurs, low down in the cord, degeneration in Burdach's and then higher up in Goll's columns, into which the root fibers are pushed as higher root fibers enter the cord. The location of the degenerated fibers therefore differs at successive levels (*ascending degeneration*). In the *lumbar cord* the posterior columns are most intensively and extensively diseased, especially in their middle and posterior parts, nearest the posterior roots and surface of the cord. Early and marked degeneration is found in Lissauer's zone just back of the posterior horn. The anterior part of the post. ext. column and the central part of the post. int. column are slightly, if at all, diseased as they are not derived from the post. ganglia. In the *dorsal cord* the post. columns are degenerated save in the post. ext. part and in the ant. part. In the *cervical cord* the post. int. columns are diseased (representing the root fibers of the lumbodorsal cord) and also some areas in the post. ext. column where degenerated root fibers have just entered. (ii) From the post. roots a series of fibers to Clarke's column is degenerated but the cells of Clarke's column and their ascending fibers to the cerebellum usually remain intact. (iii) The post. horns degenerate, because they contain fine root fibers. (iv) The reflex collaterals to the ant. horns also degenerate.

Degenerative changes may involve analogous neurones, as the *auditory, optic*, visceral, sympathetic or ocular nerves, or the peripheral motor nerves, pyramidal tracts, muscles, medulla and brain.

The exact *pathogenesis* is disputed: (i) Obersteiner conceives the initial *changes are in the pia* through which the dorsal nerve roots pass; (ii) Marie contends there is a *lymphangitis* of the post. columns; (iii) Edinger believes that *toxins attack* those *nervous structures* most in use and therefore *most easily fatigued* (the sensory tracts of the lumbar cord, pupil, bladder and intestine).

Symptoms.—Symptoms vary chiefly as to their intensity, duration and succession. *Three stages* are described: (1) The *initial*, neuralgic or pre-atactic stage, lasting sometimes for years (usually 6 to 8, or even 10 to 20). The earliest symptoms are *pains* which are lancinating or "rheumatic," perhaps neuralgic or like migraine or paresthesia. The

knee-jerk is *lost*, usually very early and the *pupil reacts* to accommodation but *not to light*. Much less often optic atrophy, sexual, sphincter, ocular, articular or visceral symptoms first attract attention. (2) The *atactic stage* may last many years; muscular movements become incoördinate, especially in the legs, sometimes, though later, in the arms. The tabetic gait develops, the patient sways when his eyes are closed, sensation is obtunded, especially the muscular sense, urinary and sexual disorders develop and in some cases diplopia. The gross muscular power is unimpaired. Trophic, joint, bone and skin lesions and visceral crises occur. (3) In the *terminal stage* the patient is bed-ridden, helpless from extreme ataxia, and bed-sores, lesions in the medulla and cystitis determine the fatal issue.

Special Symptoms in Detail.—1. *Motor Symptoms.*—(*a*) *Ataxia* (incoördination) is of such importance that the disease is named *locomotor ataxia*. It is present in 80 per cent. of cases, but may develop slowly or perhaps not at all; it occurred in barely 10 per cent. of Babinski's cases, who explains this low figure by his use of mercury. Ataxia is a disturbance of harmonic efficient muscular contraction and is probably a disease of the afferent muscular fibers (muscular anesthesia, Reynolds 1855), intensified by sensory impairment, disturbance in the reflex collaterals and sometimes by optic atrophy. Duchenne first made the valuable distinction between ataxia and loss of muscular power which is preserved until late in the disease. Ataxia is frequently first observed by the patient when he walks in the dark or closes his eyes, as in washing the face. When he looks upward or brings the heels and toes together he sways (*Romberg's sign*) and perhaps falls, because vision is a sensory factor in equilibration. Later, incoördination occurs with the eyes open, and the patient can walk only with a cane. It is often increased by removing the shoes, which support the feet. The ataxia is marked when the patient attempts to "about face," to walk backward, stand on tiptoe or stand with the knees half-flexed. Ataxia is elicited also when the patient, with the eyes closed, attempts to place his heel on the opposite knee or perform with one limb those movements made by the physician in another limb. The gait is atactic, the feet are lifted too high, thrown too far forward and outward (*wide-legged gait*) and brought down suddenly and forcibly—"*stamping*" or "rooster gait." A tumbling cerebellar gait is less frequent, observed later in the disease and referable to involvement of the trunk or hips. Movement in tabetics is characterized by excessive excursion, rapidity, irregularity and uncertainty of execution. The patient cannot execute finer movements, such as writing or buttoning the clothes. Ataxia is rare in the head, eyes, face, jaw and tongue. In women marked ataxia is less frequent than simple awkwardness. (*b*) *Muscular power* may be normal even with extreme ataxia. The muscles are *hypotonic*, which allows hyperextension and hyperflexion of the joints; it is due to loss of centripetal sensory conduction. There is a feeling of weakness in the legs in two-thirds of the cases. Transient muscular weakness is frequent (75 per cent.), as in the eye muscles. Paralysis, though usually transient, may be permanent from disease of the pyramidal tracts, ant. horns or peripheral nerves. (*c*) *Atrophy* is not

common, but may affect the arm (type of Duchenne-Aran), the leg (the tabetic club-foot, usually due to neuritis), or most rarely the face (hemiatrophy). (*d*) The *electrical reactions* are usually normal. Sometimes muscular contractures, due to abnormal sensation, associated movements, in which one leg moves when its fellow moves, and spontaneous movements, taking place without volition or consciousness of them and due to slight unnoticed reflexes, may occur.

2. *Sensory Symptoms.*—In the majority of cases the disease begins with irritative sensory phenomena. (*a*) Spontaneous *pains* occur in 85 per cent., are sudden, paroxysmal, *lightning-like*, lancinating and often nocturnal. They are usually severe, but may be agonizing, dull, burning or like those of muscular rheumatism, increased by dampness and cold. They last from a few hours to a day or two. The legs are the most common seat of pain, then the back, trunk, arms especially in the ulnar distribution and sometimes the head (occipital nerves) and face (trigeminus, whose ascending branch represents the sensory parts of most of the other cranial nerves); Collet found migraine-like attacks in 40 per cent.; their bilateral occurrence is very suggestive. The pains are more often deep than superficial and more often irregular than exactly conforming to a nerve's course. The pain in the trunk may assume the "girdle form" over a narrow or broad zone (31 per cent.). Sometimes the area affected shows local trophic changes, as herpes, ecchymoses or friable hair. (*b*) *Paresthesiæ* are common, as creeping sensations, "pins and needles" sensations, a sense of heat or cold. They occur in the legs in 66 per cent. of cases. If in the arms, the ulnar distribution is especially involved (17 per cent.). The ulnars at the elbows are often insensitive to pressure (*Biernacki's sign*), pain, temperature and tactile impressions. In the face a perverted feeling may be noted (*"Hutchinson's mask"*). (*c*) In the early stages hyperesthesia may be noted from nerve irritation. In 86 per cent. of developed cases *sensation is reduced;* it is most often of the spinal or segmental type, though sometimes peripheral. *Tactile sensation* is often disturbed, especially in the legs and soles. Hypesthesia may be observed on the buttocks, scrotum, perineum, penis (the cause of early decreased sexual power) or on the trunk as a zone (80 per cent.), *especially to light touch.* It is not usually found higher than the third rib, although there may be occasional occipital or trigeminal anesthesia. If the arms are affected it is mostly in the hands and palms. Sometimes a touch to one member is referred to another (allocheiria) or a single contact feels like several (polyesthesia). *Perception* of pain is lessened in 50 per cent. of cases, occurring alone or with hypesthesia; a short stab with a pin is often felt merely as a touch. *Pain conduction* is often retarded (60 per cent.); the sharp stick of a pin is felt at once as a touch, but the pain is perceived only after 7 to 15, even 35 seconds; the patient says "now" when stuck and "ouch" as he feels the pain. Analgesia may be the cause of severe injuries, as in syringomyelia. Sometimes the testicles are insensitive to pressure, and inflammations such as pleurisy may run a painless course. The *temperature sense* is rarely affected alone or completely. The *muscle sense* is disturbed; a change of angle of one or two degrees in the joints and tendons

is noted by normal individuals, while in tabetics much wider movements are not perceived, especially in the small distal joints; the toes may be moved unnoticed by the patient, perhaps in the later stages the fingers also.

3. *Reflexes.*—(*a*) The skin reflexes may be increased early in the disease. Later the plantar and gluteal reflexes are somewhat decreased. The abdominal and cremasteric may long remain normal. (*b*) Of the tendon reflexes the *knee-jerk* is *lost* early (*Westphal's sign*); this most constant finding in tabes (98 to 100 per cent.) is due to disease of the afferent nerves and reflex paths in the cord. These reflexes should be examined with the patient sitting on the edge of a table with his eyes closed and excluding cerebral inhibition of the reflexes by having him pull, in an outward direction, the locked fingers (Jendrassik's reinforcement). The patellar reflexes are rarely present save in exceptional cases of cervical tabes. The knee-jerk has been known to return after a cerebral hemiplegia. The *Achilles reflex* is lost equally frequently.

4. *The Eyes and Special Senses.*—(*a*) The *pupils contract to accommodation but not to light* in 97 per cent. (Argyll-Robertson, 1869). The patient must look to a distance in the dark room and one eye tested at a time lest accommodation be confused with contraction to light. The Argyll-Robertson pupil may depend upon a degeneration of fibers connecting the optic nerve with the oculomotor muscles (see page 805). The tabetic pupil rarely dilates on pinching the skin of the neck. In about 50 per cent. it is small (spinal myosis); the pupils are often unequal in size and irregular in contour. (*b*) The *eye muscles* are affected in 50 per cent. Möbius holds that ocular paralyses are as important as the pupillary changes, especially when painless, wherein they differ from the rheumatic and syphilitic forms; he holds that the majority of all eye paralyses in the adult are tabetic. Diplopia is common and usually unilateral. The order of frequency of involvement is the sixth, third and fourth nerves. When one muscle is paralyzed the others are usually weak. The early form of paralysis is usually transitory; the late form is often permanent from nuclear degeneration. (*c*) *Optic atrophy* (10 per cent.) is generally an early symptom. The disk is pale and shrunken, especially on its temporal side and at the edges, which are sharp. The process is always bilateral; central scotoma and actual neuritis are rare. The process usually progresses to blindness. When optic atrophy occurs early, ataxia may not appear, "tabes arrested by blindness." When it develops later it has no effect on the ataxia. (*d*) *Deafness* may develop gradually or suddenly, sometimes with vertigo or Ménière's complex. It may be permanent or transient. Auditory defects are present in 80 per cent. of cases. Atrophy of the auditory and olfactory nerves has been found.

5. *The Sphincters and Genital Sphere.*—(*a*) The *bladder* is frequently affected (60 per cent.), possibly as the first symptom of the disease. The bladder is slowly or imperfectly evacuated, because its centripetal sensory fibers are diseased. There may be overflow, incontinence and in the later stages cystitis frequently results from decomposition of the residual urine, followed in turn by pyelitis, nephritis and septicemia.

(*b*) The *sphincter ani* is weak but rarely incontinent. (*c*) The *sexual* appetite and power are reduced (66 per cent.), often early. Labor is sometimes painless.

6. *Trophic Disorders.*—Local trophic changes are more common than general malnutrition. (*a*) Trophic alterations in the *bones and joints* were first thoroughly studied by Charcot—Charcot's disease. They usually appear late and develop in 2 per cent. of cases. The onset is usually gradual, sometimes acute with marked swelling or seemingly traumatic. Two forms are observed: first the more frequent atrophic form, especially in the hip and shoulder, in which the cartilages are eroded, the articular surfaces waste and crepitate, foreign bodies develop and the joint becomes so lax that dislocation of the hip or knee (genu recurvatum) results; second, the hypertrophic form with bony formation, ossification of the ligaments, and rigidity resembling arthritis deformans but *painless*, more acute, extreme and effusive. The *x*-ray plates are characteristic. The adjacent muscles atrophy—the arthropathic muscular atrophy. The large joints are usually involved, as the knee (45 per cent.), hip (20 per cent.), shoulder (11 per cent.), elbow (5 per cent.) or ankle (4 per cent.), but smaller articulations may be affected, as the tarsus, jaw, spine, thumb, etc. The cause is the same as in syringomyelia. The *tabetic foot* is characterized by thickness and roundness of its inner border and arch, flattening of the sole, decreased motility, and pathologically by erosion and atrophy of its bones. It must be distinguished from the tabetic club-foot due to muscular atrophy of the calf muscles with contractures. The *bones* may *break easily but painlessly* (Weir Mitchell), due to rarefying osteitis, which renders the medullary substance more porous, the cortex thinner and the inorganic base less in amount. If a callus develops it is often exuberant. The tendons sometimes rupture, probably from trophic changes. (*b*) The *perforating ulcer* of the foot (*mal perforant du pied*) is more frequent. It is "punched out" in appearance, painless, secretes but little pus and is due to nerve degeneration or centric changes. Similar ulcerations resembling syphilis may occur in the nose, ear, buccal mucosa, palate or uvula. (*c*) In the *skin*, local sweating, ecchymoses, herpes, blebs, alteration of the hair or nails, gangrene and ichthyosis may occur. Decubitus is not common save in the last stages, and it may not develop even after years in bed. There may be dental caries or painless falling out of the sound teeth with analgesia of the gums.

7. *Visceral Symptoms.*—The most important are the *painful crises* first correctly interpreted by Charcot. (*a*) *Gastric crises* occur in 5 per cent. of cases. There is severe, sometimes excruciating epigastric pain, which often radiates into the back, vomiting of food, then fluid, finally bile or even blood; this is often attended by restlessness, epigastric retraction, hiccough, fever and a slow, rapid or irregular pulse; hyperacidity is less frequent than hypacidity. The alkaline urine contains indican and decreased chlorides. The crisis lasts several hours to days and may greatly reduce the general nutrition; it is sometimes the first evidence of tabes. The cause is usually considered nervous, but Pal thinks it is due to increased arterial tension. (*b*) *Laryngeal crises* are next

in frequency but are shorter in duration. They are due to spasm of the laryngeal muscles, and are often dangerous. In some instances only a hoarse cough like pertussis is noted; in others there is collapse or death (ictus laryngis). Anatomically, the ascending branch of the fifth nerve and the glossopharyngovagus nucleus or its branches are degenerated. (*c*) Other crises are far less common, as *intestinal* crises, which are characterized by colic, diarrhea and sometimes rectal tenesmus; the author saw obstruction and abdominal retraction lasting twelve days, immediately relieved by morphine gr. $\frac{1}{2}$; renal crises with scant urine, transient albuminuria or even hematuria, and less often nasal, pharyngeal, hepatic, cardiac, bronchial, vesical, urethral or vulvovaginal crises occur.

8. *Cerebral Symptoms.*—Cerebral symptoms other than the eye changes noted above, are not frequent; they are unilateral atrophy of the tongue; paralysis of the vocal cords; vagus and accessorius symptoms other than those already noted, such as rapid pulse, palatal weakness or rarely paralysis affecting the trapezius and sternomastoid, which is probably neuritic; and very rarely facial or bulbar paralysis. Lachrymation, salivation, polyuria or glycosuria is sometimes noted.

Complications.—These include true cerebrospinal syphilis; *aneurysm*, *aortic regurgitation* (5 per cent.) and especially *dementia paralytica*, a closely correlated affection; myelitis; muscular atrophy, particularly the symmetrical form due to neuritis; cerebral thrombosis due to arterial disease; and apoplectiform seizures which occur as in paretic dementia.

Course and Prognosis.—Pain is the first symptom in 68 per cent., while no other single early symptom is the first in more than 3 per cent. (Sarbó). Optic atrophy or insanity may arrest tabes. Leube instances a man who worked hard for forty years after the onset of tabes; tabetics may live for twenty years in the last stages of the disease. Crises, alcoholism, traumatism, acute onset, intercurrent affections unnoticed because of their painlessness, and affections of the bladder and kidney with sepsis are unfavorable prognostics. In 1960 well-observed cases (out of 4000) 40 per cent. followed a long course with exacerbations, 30 per cent. were steadily progressive, 19 per cent. halted for three years or more, 6 per cent. ran a severe, rapid course and 5 per cent. almost recovered (Belugou and Faure). Tabes is incurable.

Diagnosis.—The diagnosis is made by: (*a*) The *Argyll-Robertson pupil*, which is indicative of tabes, paretic dementia or rare lesions in the corp. quadrigemina. (*b*) The most common and early symptom, loss of the patellar reflex (which most rarely fails in health, marasmus, old individuals or as an hereditary lesion). (*c*) *Lightning pains*, "tabetic neuralgia," girdle sensation, *gastric and other crises*. (*d*) *Sensory changes*, subjective, objective and vesical. (*e*) *Optic atrophy*. (*f*) *Painless ocular paralysis*. (*g*) *Ataxia*, with *Romberg's sign* and the *tabetic gait*, is practically pathognomonic after exclusion of peripheral neuritis. (*h*) The *Wassermann reaction* is present in 70 to 96 per cent. of cases (see pages 219 and 229) in the blood and spinal fluid, with globulin reactions and lymphocytosis.

In *cervical tabes* (less than 1 per cent. of cases) the sensory changes, pains and ataxia are most manifest in the arms or even in the face. The

arm reflexes are sometimes abolished and the patellars rarely may be normal or increased. The characteristic pupils are present and gastric crises appear early and frequently.

Differentiation.—In the earliest stage confusion with neurasthenia, hypochondriasis and nosophobia is possible, from common symptoms, as pains, paresthesia, girdle sensation and weakness of the legs, but close observation determines the diagnosis; the tabes of Hippocrates was probably neurasthenia. One form of *multiple neuritis* (*q. v.*), viz., pseudotabes, may require differentiation. *Cerebrospinal syphilis*, especially of the roots and the membranes, may sometimes cause confusion.

For differentiation from *Friedreich's ataxia*, see page 865. In *cerebellar tumor*, usually the ataxia ceases when the eyes are closed and is not characterized by the excessive excursion or rapidity of tabes; the patellar reflexes are increased, there is no anesthesia and signs of tumor are often present. In *multiple sclerosis* there exceptionally may be crises; pains and sensory disturbance, but the Argyll-Robertson pupil, ataxia, marked sensory and bladder disturbances are most rare. There are increased reflexes, nystagmus, scanning speech, intention tremor, central scotoma and functional oculomotor involvement. *Paraplegias* are not difficult to differentiate, since true motor disturbance is rare or late in tabes; the reflexes are increased and spastic rigidity is marked, even in atactic paraplegia. *Tabetic pain* is often misinterpreted; pain as a rule suggests disease of the vertebræ and membranes rather than of the cord itself, but in vertebral disease or cord tumor the pain is fixed, and often increased on pressure and movement. Tabes may be confused (on hasty or partial examinations) with *rheumatism*, intercostal or trigeminal neuralgia, and gastric, renal or other *visceral disease*. Bilateral sciatica always suggests tabes, diabetes or nephritis. Strümpell describes, among workers in tobacco, a *nicotine tabes*, in which pain, pupillary alteration, atactic gait and absence of knee-jerks are observed, but also a characteristic tremor and increased cutaneous reflexes in the legs obtain.

Treatment.—1. Etiological Treatment. — Antisyphilitic remedies (*q. v.*) are indicated.

2. Hygienic Treatment.—Overwork, overexercise, worry, trauma, exposure and excess in tobacco, alcohol or sexual indulgence should be avoided. Tabetics should not marry. The diet should be regulated, since dyspepsia promotes pain. If possible, the patient should live in a warm climate and at a moderate altitude.

3. Drug Treatment.—Silver, strychnine and nitroglycerin give uncertain results. Gowers considers arsenic beneficial.

4. Balneotherapy.—Warm baths are injurious; lukewarm baths, *electrotherapy and massage* exert no specific influence on the disease.

5. Symptomatic Therapy.—(*a*) If *pain* is superficial, sinapisms, chloroform liniments or the galvanocautery should be tried; if moderate, acetanilide, cannabis indica or peripheral faradization is beneficial; morphine should be avoided as long as possible, because the habit is readily acquired. Gowers described cases in which pain was a salient symptom and existed without ataxia or loss of the patellar reflexes; for this "tabetic neuralgia," differing from the ordinary pains of tabes, he recommends

salicylates and aluminium chloride, gr. v–x, t. i. d. (*b*) *Crises* usually necessitate morphine hypodermics. Laryngeal crises often respond to nitroglycerin hypodermically, chloroform by inhalation or cocaine locally. Stretching the spine and cord probably relieves pain; the patient sits on the floor with the legs extended; without other movement, the body is carefully pushed forward (flexed) as far as possible. Section of the nerve roots (rhizotomy) and cocaine injections are recommended. (*c*) For *vesical* affections, catheterization for residual urine is indicated, but under most rigid antisepsis; belladonna and faradization are often helpful. Cystitis is treated by lavage. (*d*) Secondary infection should be avoided. Corns should never be cut but always rubbed off.

6. Ataxia.—This may be relieved by H. G. Frenkel's systematic exercises, "a reëducation of the central nervous system by repeated exercises;" carefully and persistently executed muscular movements, not forceful movements, are desirable. Nerve stretching and suspension are useless.

II. System Diseases of the Motor Tract.

The motor paths may be diseased (1) in the upper neurone, as in spastic spinal paralysis; (2) in both neurones, as amyotrophic lateral sclerosis; (3) in the peripheral neurone, as spinal muscular atrophy or bulbar paralysis; (4) in the peripheral motor nerve, as the neuritic muscular atrophy and (5) in the muscles themselves, as the dystrophies. These types may blend; separate descriptions are necessary to bring out the classical symptoms.

1. Spastic Spinal Paraplegia.—Lateral sclerosis described first by Erb, in 1875, concerns only the upper motor neurone.

Etiology and Pathology.—The disease represents a symptom-complex rather than a pathological entity. The causal factors are uncertain, as syphilis or an hereditary tendency. In 75 per cent. it occurs between the twentieth and fortieth years. It was thought that there was a primary sclerosis of the lateral pyramidal tracts, especially their lower parts, for which confirmation is still lacking; there are some lesions in other neurones, as in the post. columns.

Symptoms.—There are *three cardinal findings,* motor weakness, muscular rigidity and increased reflexes. (*a*) *Motor weakness* is usually the first sign; it begins slowly and is evidenced by weakness or stiffness in the flexors of the hips especially but also the knees and ankles. Every gradation from paresis to paralysis may be noted in different cases or in different stages. Strümpell attributes the loss of finer synergistic movements to involvement of the lateral pyramidal tracts; the coarser movements for groups of muscles are possible *via* the anterior pyramidal tracts. The muscles are large and well nourished. (*b*) *Muscular rigidity* results also from disease of the lat. pyram. tracts, which lessens the inhibition and is proportionate to the loss of power. The muscles resist quick, passive efforts at flexion, but yield to slow flexion and remain in the position given them, "*lead-pipe contraction.*" The knees are rigidly extended, even when the patient sits on the edge of the bed; the great toe is strongly flexed dorsally; when one leg is lifted the other comes

with it; the specially tense adductors prevent abduction of the thighs. The muscles are stiff in the morning; painful spasms may occur during the night; hypertonicity may lead to active contractures and patients complain of the "tendons getting too short." In active movements rigidity is manifest; the gait is paretic and spastic, the knees are extended and closely apposed, the steps are short, the balls of the feet cleave to the ground and the feet scuffle. (*c*) *Increase of the reflexes* is also due to lateral pyramidal sclerosis. The *knee-jerks* are most *excessive;* ankle-clonus and Babinski's reflex are present; a strong clonic contraction of the quadriceps follows depression of the patella and the gait may be hopping. The *arms* are often normal; there may be rigidity like that of a late hemiplegia; there may be clonic spasm of the fingers and increased tendon and periosteal reflexes. There sometimes are weakness in the back and painful contractions of the chest and back muscles with dull aching. The cranial nerves are rarely diseased, although the jaw-jerk may be increased. The electrical reactions, sensation, muscular nutrition and eyes are usually normal. The sphincters are usually affected late.

Clinical Forms and Diagnosis.—Diagnosis concerns the paresis, hypertonicity, increased reflexes and absence of sensory, trophic and other symptoms. Six types may be described: (1) There are rigidity and spasticity in the legs, arms and perhaps the face, sometimes with compulsory laughing or weeping, as in amyotrophic lateral sclerosis, but without muscular atrophy.

2. The *hereditary or familial form* commences between twenty and thirty years of age; the paresis develops late; this form is characterized anatomically by sclerosis of the (congenitally weak) lateral pyramidal tracts and also by sclerosis of the cerebellar and Goll's columns (more properly classified under combined system disease).

3. In *infantile* diplegia, the rigidity is less, foot-clonus less common and tonic spasms rare; athetoid movements, deficient mentality and epileptiform convulsions are common.

4. In the *syphilitic form of Erb* there are paresis and slight spasticity, plus urinary incontinence, retention and impotence.

5. There are *forms symptomatic of brain disease* (tumor, syphilis, paresis), *cord disease* (tumor, compression, syringomyelia, multiple sclerosis) and *hysteria* which has not the peculiar extensor spasm; loss of power is rarely complete and ankle-clonus is rare, although a spurious half-voluntary contraction of the calf muscles may be noted.

6. *Periodic familial paralysis* affects the legs chiefly, is flaccid and results in decrease of the patellar and absence of the plantar reflexes, decreased faradic irritability, sleepiness, but without sensory symptoms; it lasts one to three days. The prognosis is good.

Prognosis.—"It is perhaps the least dangerous to life of any chronic spinal lesion." Recovery or arrest is rare.

Treatment.—Overexertion, fatigue, electrotherapy and strychnine are to be avoided. Massage and warm baths relieve the spasticity.

2. Amyotrophic Lateral Sclerosis.—This is often called the *maladie de Charcot* (Charcot and Joffroy, 1865).

Etiology.—It occurs mostly between the twenty-fifth and forty-fifth years, more frequently in males; its cause is not known.

Pathology.—The name amyotrophic lateral sclerosis designates the leading changes, which are, *amyotrophy*, from atrophy of the anterior horns and *sclerosis of the lateral columns.* (*a*) The *lateral pyramidal tracts,* and to a lesser extent the anterior, show symmetrical primary degeneration, followed by interstitial sclerosis which is most marked in the lower part of the upper neurone, but in some cases extends upward to the medulla, pons, crus or even the central convolutions, the ganglionic cells of which may be atrophied. In some cases unimportant changes may be found in the cerebellar tracts or posterior columns. (*b*) The *ganglionic cells of the anterior horn* are degenerated, so that the anterior motor part of the cord is distinctly smaller and flatter. Likewise the nerve filaments from the pyramidal fibers, anastomosing with the processes of the ant. horn, are degenerated. These changes in the trophic centre of the second motor neurone explain the atrophy of the anterior roots, motor nerves and muscles. (*c*) The *bulbar nuclei,* analogues of the ant. horns, with their nerves and muscles, show identical changes.

Symptoms.—The symptoms, like the lesions, are strictly motor.

Stage 1.—The disease begins with weakness and atrophy of the *muscles* of one, then the other hand, most marked in the thenar, hypothenar and interosseal muscles, and followed by wasting and weakness in the *forearm extensors, deltoid* and *triceps;* other muscles of the arm and forearm escape or are involved later. Sensory symptoms are absent. Weakness is often manifest before the atrophy. Other symptoms of disease in the lower neurone appear, as *fibrillary contractions* and the *reaction of degeneration;* the nerve trunks respond normally. As evidences of involvement of the upper neurone, paralysis often precedes the wasting and accentuates the weakness from ant. horn involvement; there are *increase of the reflexes* (which never occurs in muscular atrophy due to disease of the ant. horns alone) and *rigidity and contracture* in which the arm hugs the chest, the forearm is flexed and pronated, and the wrist and fingers are flexed. Sometimes the neck is rigid and trismus is observed.

Stage 2.—In less than a year the *legs are involved;* weakness, especially in the flexors of the hip, knee and dorsal flexors of the foot and spasticity prevail. The *gait is spastic* and *paretic* as in spastic spinal paralysis and walking becomes impossible because of the rigidity and the *twitching tetanic movements* of the *extensors.* The *patellar reflexes* are *exaggerated;* ankle-clonus is frequent; when the patient attempts to flex the thigh while the examiner holds the ankle, there is a visible contraction of the tibialis anticus (the tibialis phenomenon); *Babinski's reflex* is present—evidences, in short, of pyramidal disease, because symptoms referable to the lower neurone, as atrophy, are much less frequent, less marked and less early in the legs than in the arms.

Stage 3.—This stage occurs in one or two years with dysphagia, atrophy of the lips and tongue with fibrillation, as in primary *bulbar paralysis* (*q. v.*). A marked jaw-jerk is elicited, tonic tension of the facial muscles may occur and spasmodic laughing or weeping from loss of in-

hibition. The mind is clear. Death results from respiratory failure, aspiration pneumonia, etc.

Course and Prognosis.—In some cases the pyramidal or paretic, in others the atrophic, symptoms are dominant. The course covers one to four years and is fatal.

Diagnosis.—The involvement of both motor neurones, muscle atrophy with increased reflexes, rigidity and the final bulbar symptoms establish the diagnosis.

3. Progressive Spinal Muscular Atrophy.—Duchenne and Aran (1849–50) gave the first full clinical description of this disease.

Etiology.—The ultimate causes are not known. Seventy-five per cent. of cases occur in males, mostly between the twenty-fifth and forty-fifth years. Hereditary factors, often observed in the muscular dystrophies (*v. i.*) are infrequent, although 28 familial infantile cases are reported. Hard work, mental distress, syphilis, trauma, infections and poliomyelitis are possible factors.

Pathology.—In the cord, especially in the cervical region, (*a*) *the anterior horns are small and flattened;* their ganglion cells are deformed, wasted, their processes lost; interstitial changes are secondary. (*b*) The *anterior roots,* especially the cervical, are *degenerated* and often also the peripheral nerves, although changes in them are most difficult to distinguish. (*c*) The *muscles are small, stringy* and pale and their individual fibers are small; their transverse striation persists remarkably, and waxy, fatty or granular degeneration and longitudinal splitting are not common; the muscle nuclei multiply, round cells may wander in, and there is usually increase of connective tissue. Amyotrophic lateral sclerosis, spinal muscular atrophy and bulbar paralysis are one affection.

Symptoms.—(*a*) The essential symptoms are motor. The earliest is *muscular atrophy* with *weakness* in *the upper extremity* (90 per cent.), especially the (right) *thenar* and *hypothenar eminences.* The abductor indicis and opponens pollicis are first involved, then the flexor brevis and adductor, the wasting of which brings closer together the metacarpi of the thumb and index-finger (the "*ape-hand*"). It is finally impossible to appose the thumb to the fingers. The interossei on the dorsum waste and leave marked furrows, producing the "claw-hand," *main en griffe,* from contraction of the antagonist ext. digitorum communis, the tendons of which stand out prominently. The wasting lumbricales hollow the palm. In this Duchenne-Aran type, individual muscles are selectively involved. The muscles become lax, thin and flat. The *atrophy long exceeds the paresis.* The *forearm* may be next involved, first the extensors below the int. condyle, later the supinators and flexors. A deep depression between the ulna and radius eventually occurs. After years, the *shoulder* muscles, the posterior and middle thirds of the deltoid, the biceps and brachialis waste and much later the triceps. Sometimes the wasting of the muscles of the shoulder and arm immediately follows the involvement of the hand, or perhaps the disease begins in the shoulder. The bones of the shoulder stand out clearly, covered only by the skin, "the living skeletons" seen in museums. The trunk muscles are involved in this order: trapezius in its middle and lower parts (rarely in its upper third, which is the

ultimum moriens of Duchenne); pectoralis, latissimus dorsi, rhomboidei (causing the shoulder to droop) and extensors of the head (allowing the head to fall forward). Lordosis is common and the spine stands out distinctly. The platysma and face escape. The respiratory muscles, intercostals or diaphragm are involved, which latter imperils life. In the leg the muscles are affected late and to a lesser degree, as the glutei, quadriceps and the peronei. Very exceptionally the disease first develops in them. In the fully developed cases there is extreme helplessness; an added misery in the form of the sister-process, bulbar paralysis, may develop.

(*b*) In the wasted muscles there are usually *fibrillary contractions;* they are involuntary twitchings, tremor- or even wave-like in character. They may appear in muscles not yet wasted. (*c*) The *electrical reaction* in the nerves is normal. In the muscles there is usually only quantitative reduction and the voltaic excitability remains long after the faradic is lost; in the last stages there is a partial reaction of degeneration. (*d*) The *reflexes* are *weakened* or abolished in proportion to the atrophy and due to disease in the motor side of the reflex arc. (*e*) *Sensation* shows no objective change; in some instances there are dull, aching pains in the muscles. (*f*) Vasomotor, secretory, sphincter and trophic symptoms are lacking.

Diagnosis.—The diagnosis is based on the slow course; the involvement of the hand and its slow "individualization"; fibrillation; partial reaction of degeneration; and decreased reflexes.

Differentiation is necessary (1) from disease in which muscular atrophy is but one among several symptoms. (*a*) *Syringomyelia* is marked by its trophic characteristics, its analgesia, thermo-anesthesia and less symmetrical wasting. (*b*) *Meningitis,* spondylitis and tumors are manifested by anesthesia, pain, tenderness and rigidity. (*c*) In *multiple neuritis* there is a clearer reaction of degeneration, more sensory signs and tenderness in the nerve trunks. (*d*) The *occupation atrophies* are distinguished by the history.

2. *Other muscular atrophies.* (*a*) *Chronic poliomyelitis* causes paralysis first and atrophy afterward; it affects groups of muscles at once, its course is rapid and fibrillary contractions are infrequent. (*b*) The *myopathic atrophies* (see p. 864). (*c*) *Amyotrophic lateral sclerosis* is an allied, perhaps identical affection; the atrophy is not "individualized," but occurs *en masse;* the distinguishing features are the increase of the reflexes, spasticity and a much more rapid course. (*d*) *Arthritic muscular atrophy,* probably due to reflex influences, or direct extension of inflammation to the muscles or nerves, involves chiefly the extensors in a moderate wasting; diffuse wasting suggests a nervous cause. There is usually increased myotatic irritability and reflex action.

Course and Prognosis.—The onset between the twenty-fifth and forty-fifth years is gradual, the course is slowly progressive, remissions infrequent, and death usually results from intercurrent disease, respiratory affections (tuberculosis) or bulbar palsy. Arrest is rare and chiefly in cases marked by symmetry and synchronous involvement of both hands.

Treatment.—Gowers gives strychnine hypodermically; he has seen 7 consecutive cases of arrest of which 6 were permanent; it has little

effect by mouth. Massage, active and passive gymnastics and electricity do not stay the disease.

4. Neural Muscular Atrophy.—Charcot-Marie type. Peroneal family type (Tooth). Neurotic type (Hoffmann).

FIG. 74. — Typical pseudohypertrophic muscular paralysis. (Dercum.)

Etiology.—It is often hereditary, even through five generations; it is usually familial. It occurs in the second half of childhood and rarely after the twentieth year. Sixty-six per cent. of cases are in males.

Pathology and Symptoms.—The pathological characteristic is *neuritis;* secondary sclerosis has been seen in the posterior and lateral pyramidal columns. Early atrophy occurs in the *small muscles of the feet,* with bilateral, symmetrical peroneal paralysis and club-feet (*pes equinus* or *varo-equinus*). The calf muscles suffer next and the thigh still later. After years, paralysis and atrophy of the hands, as in progressive spinal atrophy, develop, with the "claw-hand"; in the forearm, the extensors suffer more than the flexors. The shoulder, neck, trunk and face are seldom diseased. Fibrillation is infrequent; the reaction of degeneration is generally present, paresthesia and anesthesia (especially plantar) are common; the patellar reflexes may long persist.

5. Muscular Dystrophy.—The dystrophies or myopathies are often classed with diseases of the muscles, which indeed show the sole constant pathological lesions. They are so closely related clinically to the less frequent spinal atrophies that they are here considered. Changes in the cord, nerve trunks and nerve filaments have been found in some cases of each type. (*a*) *Heredity* is an obvious cause in 56 per cent. or a familial incidence is often observed; the muscular system seems deficient in development or vitality. (*b*) It occurs in *childhood* or pubescence. (*c*) The *muscles waste,* with or without hypertrophy or with increase of fat or interstitial growth.

Pseudohypertrophic Muscular Paralysis.—Lipomatous muscular atrophy is the least frequent, but is the oldest and best known type (Bell, 1830; Partridge, 1837; Meyron, 1852; Duchenne, 1861). It occurs in males (80 per cent.) and is less marked, less early in life and less fatal in women. It is often transmitted by healthy mothers, the males being impotent,

and may be seen in five generations. It begins early (75 per cent. under ten years).

1. *Impaired motility* is the earliest symptom. The weakest muscles are the invisible hip flexors and the knee extensors which make climbing of stairs difficult, cause the waddling gait (paresis of glutei) and interfere with rising from the floor, so that the child must rise by resting on all fours and by "climbing his own legs with his hands." Paresis of the back muscles and hip extensors causes lumbar lordosis and abdominal protrusion. The ankle extensors also weaken. The child stands with legs far apart. The depressors of the shoulders are weakened early in the disease, later the shoulder muscles, biceps and triceps, but the hand remains normal for a long time, or escapes. The weak muscles show nuclear increase, atrophy and division of their fibers, which are usually undersized, pale and narrow; fatty degeneration and loss of striation are rare. Increase of connective tissue is a later but most characteristic change.

2. Certain muscles show apparent increase in size (*pseudohypertrophy*), due largely to early adipose deposit or rarely to actual muscular hypertrophy. The calf muscles are especially enlarged, then the glutei, knee extensors, or infraspinatus which contrasts strongly with the wasted latissimus and deltoid.

3. The electrical irritability is lowered, but there is no reaction of degeneration. The deep reflexes disappear, from the muscular lesion. Mentality, sensation and the sphincters are normal. Cardiac hypertrophy was observed in 60 per cent. of Glotz's cases. The patient becomes bed-ridden, arrest is rare and death occurs after years from intercurrent disease or respiratory inflammation.

Infantile Atrophic Form with or without Facial Involvement.—Wasting occurs from the beginning. It may be combined with the first form, and the legs then show pseudohypertrophy and the arms simple atrophy. The pathology is that of the first form, but there is no hypertrophy, true or false; increased connective tissue characterizes this type of atrophy and also that of the juvenile form (*v. i.*).

The *face* may be involved. The lids droop and cannot be wholly closed, the forehead is smooth, the cheeks are flaccid and droop, the lips part and functionate poorly, the lower lip protrudes, the nasolabial fold disappears, the face becomes expressionless (*facies myopathica*). The masseters, tongue, palate, larynx, pharynx and eye-balls are implicated rarely. Simultaneously or more often later, the shoulder girdle wastes as in the first type—the *facioscapulohumeral* form of Landouzy and Déjérine; the latissimus and lower part of the pectoralis are usually first affected, but in the biceps, triceps and often in the supinator longus, the symmetrical wasting and paresis are more conspicuous. The forearm, save for the supinator longus, and the hand usually escape. The neck is seemingly lengthened (Brissaud), because of the drooping of the shoulders and clavicles. The leg muscles then waste, especially the hip flexors, knee extensors and less often the glutei; lordosis, the waddling gait and symptoms observed in the next type (*v. i.*) may develop. The facial wasting may follow that of the arms. The course is progressive, very

chronic (ten to fifty years) and death results from intercurrent infections, respiratory catarrh, tuberculosis, etc. In the second and third types the prognosis is more favorable than in the first form and the outlook is better when the face is unaffected.

The Juvenile Form of Erb.—This type begins after puberty, possibly later (twentieth to fortieth year) and is more frequent in girls than the above forms. It begins more often in the shoulders and arms than in the legs. The pectoralis, latissimus, trapezius, serratus mag., rhomboidei, sacrolumbalis, longiss. dorsi, supinator longus and later the triceps are wasted, while the sternomastoid, supra- and infraspinatus, lev. anguli scapulæ, coracobrachialis, teretes, deltoid and the hand and forearm (except the supin. long.) are almost always exempt. If we lift the patient with our hands beneath his shoulders he tends to "slip through"; this is due to "loose shoulders" (Erb). The scapulæ stand out prominently from weakness of the serrati. The glutei, quadriceps and less often tibialis ant. and peronei become atrophied. The lordosis and gait are as above described.

Diagnosis of Myopathic Atrophies.—(*a*) *Atrophy* from *cerebral lesions* is less marked and follows the marked loss of power. (*b*) *Spinal muscular atrophy:*

Spinal Atrophy.———	*vs.* ———Myopathic Dystrophy.
Begins in small hand muscles.	In trunk, shoulder or pelvic girdle, leg and upper arm.
Extends upward (face free).	Downward (face sometimes affected).
Muscular atrophy.	Frequently with hypertrophy.
Fibrillation the rule.	Very exceptional.
Tendon reflexes increased in amyotrophic lat. sclerosis.	Never increased; decreased.
Partial reaction of degeneration.	Practically never present.
Age; usually begins after thirtieth year.	In childhood, rarely after twentieth year.
Heredity—rare.	Frequent.
Bulbar symptoms not uncommon.	Never present.

(*c*) In *congenital spastic paraplegia*, increased reflexes and a regressive rather than a progressive tendency are characteristic. (*d*) *Peripheral neuritis* (*q. v.*) is toxic, acute and shows sensory symptoms and local tenderness over the nerves and muscles. (*e*) In *congenital defects* in the pectoral muscles and trapezius, progression is absent.

Treatment.—Electricity causes too much pain. Reasonable exercise is advisable, because the patients begin to fail when they cannot walk. The respiratory tract must be watched, since catarrh predisposes to tuberculosis.

Under diseases of the motor neurones, ophthalmoplegia, Landry's paralysis, bulbar palsy, myasthenia, poliomyelitis, etc., might also be described, but are treated elsewhere for differential reasons.

III. Combined System Diseases.

Kahler and Peck, in 1877, directed attention to cases with simultaneous disease in several systems of neurones. Von Leyden, Goldscheider and Nonne consider them diffuse, vascular or even myelitic affections rather

than true system diseases, of which, in their opinion, there are but two examples, tabes and progressive muscular atrophy.

1. Hereditary Ataxia, Friedreich's Ataxia (Friedreich, 1861).—**Etiology.**—This affection is hereditary in 33 per cent., and in 80 per cent. familial. Most cases occur in boys. It is generally considered a congenital abiotrophy of the pyramidal and other tracts (Freidreich, Schultze, Strümpell).

Pathology.—The cord, medulla and pons are small. The earliest and dominant change is atrophy with secondary *sclerosis of the post. roots and post. columns*, and in Goll's more than in Burdach's columns. Lissauer's "root zone" is less affected than in tabes, but more than in ataxic paraplegia. The *direct cerebellar tract*, lat. ascending tract of Gowers and Clarke's gray columns share in the atrophy. The *lat. pyramidal tract* suffers in most cases, its inner portion being usually normal. Chronic leptomeningitis, with annular sclerosis of the cord, is occasional.

Symptoms.—(*a*) The *cardinal sign is ataxia*, which is less rapid, excessive and stamping than tabetic ataxia, and more closely resembles cerebellar ataxia in its tumbling character and involvement of the trunk. Static ataxia appears later, the head oscillates or the arms show jerky movements (51 per cent.). *Romberg's sign* is usually present and the subject leans over to watch his feet; this leads to some kyphosis. Testing the sense of location elicits ataxia, as in the knee-ankle test. The arms become atactic after a few years. *Nystagmus* develops as a late symptom; it is elicited chiefly on lateral (or upward) movements and is atactic, as is also the *disturbance* of speech, which is monotonous, irregular, unclear, slightly scanning, slow and strained; syllables are often elided.

(*b*) The *knee-jerks* are early decreased or *abolished* and Babinski's sign obtains. The skin, pupillary and sphincter reflexes are usually normal. In some (possibly syphilitic) cases the pupil reflex may disappear. (*c*) *Club-foot* (pes equinovarus) is common; the foot is shortened and widened, the arch is exaggerated (pes cavus), the great toe is strongly extended dorsally. Scoliosis and kyphosis are common. Muscular paralysis or wasting is rare. Paresthesiæ, crises, lancinating pains and trophic, vasomotor and sensory disturbances are lacking. General or sexual development may be retarded.

Course and Prognosis.—About 33 per cent. develop before the sixth year, 33 between the sixth and tenth and 33 after the tenth year. The atactic gait is the first sign, then speech is involved, possibly then the kyphosis and club-foot develop, and later the arms are affected; the course is progressive, possibly with remissions, and the patient becomes bed-ridden in twenty to thirty years. Death follows from acute infection, cystitis and sepsis.

Diagnosis.—The diagnosis is based on the hereditary or familial etiology, age, ataxia, absent knee-jerks and disturbance of speech.

DIFFERENTIATION.—1. In *tabes* there are the syphilitic stigmata, immobile pupil, optic atrophy, pains, crises and vesical, trophic and marked sensory disturbances.

2. *Multiple sclerosis* is differentiated by the spasticity, increased reflexes, optic atrophy, compulsory laughing, apoplectiform insults, intentional tremor and scanning speech.

3. In *cerebellar tumors* there are optic neuritis, headache and vomiting. Marie, Londe and Nonne described an *hereditary cerebellar ataxia,* which is possibly due to congenital atrophy of the cerebellum (middle lobe), degeneration in Clarke's columns, cerebellar tracts and posterior columns, is also hereditary or familial, but develops later, between the twentieth and thirtieth years. The ataxia is cerebellar. The *reflexes are increased* (which Patrick thinks is the sole differentiating point), often with spasticity; quite frequently there are *optic atrophy,* limitation of the visual field, eye paralysis and sometimes an immobile pupil, nystagmus, choreiform movements and moderate sensory and speech disturbances. Clubfoot, scoliosis and Romberg's sign are absent. Sanger Brown reported 25 and Neff 13 cases.

4. *Cerebral diplegia* of children and ataxic paraplegia are distinguished by reflex increase and spasticity.

5. "*Amaurotic family idiocy*" (Warren Tay, 1881, and B. Sachs, 1887) is characterized by paralysis, idiocy, optic atrophy, marasmus and death before the third year of life. Most cases are Jews. At autopsy hypoplasia, microgyria, decreased pyramidal cells, tangential and other fibers are noted. Bing (1909) collected 106 cases.

Treatment.—Treatment is symptomatic.

2. Ataxic Paraplegia.—Most cases occur in males between thirty and forty; syphilis is a rare cause. *Pathologically,* there is a lateroposterior sclerosis; in the lat. column the degeneration is not wholly symmetrical or pyramidal; it may invade the mixed zone; in the post. column the process is not more intense in the lumbar cord and is less intense in the root zone than in tabes. Sometimes the process is so diffuse that it suggests myelitis or arterial obstruction.

Symptoms.—The early symptoms are those of a gradual *spastic paraplegia* (weakness, exaggerated reflexes, rigidity), followed by *ataxia* (mostly in the legs, with Romberg's sign). The arms are involved later and to a less degree. In the last stages the ataxia is obscured by the spastic paraplegia; vesical or rectal tenesmus and muscle cramps are common. Cranial nerve involvement is sometimes suggested by slightly disturbed articulation, nystagmus, tremulous facial movements and increased jaw-jerk. The mind is usually clear. Dull sacral pain is sometimes observed.

Differentiation.—(*a*) Certain *tabetic* symptoms are absent, as the high-stepping, stamping gait, immobile pupils, pains, crises, and absent knee-jerks. (*b*) *Friedreich's ataxia* is distinguished by the age, heredity, speech and absence of patellar reflexes.

Prognosis and Treatment.—See Spastic Spinal Paraplegia.

3. Other Combined System Diseases.—Combined sclerosis may also occur in pellagra, ergotism, lathyrism (*q. v.*), multiple neuritis, diabetes, pernicious anemia, icterus, leukemia, brain tumor, plumbism or diphtheria. It was found by Putnam in enfeebled, middle-aged persons, especially in women.

Pathologically, there is posterolateral sclerosis with diffuse or discrete degenerative foci. The most diffuse changes are in the cervical and the least in the lumbar cord and sometimes changes exist in the ant. horns and Clarke's columns. There are sensory *symptoms* (lancinating pains, paresthesia, anesthesia, ataxia), motor symptoms (paresis, paraplegia, chloreiform movements) or reflex symptoms (the patellars being exaggerated or lost).

DISEASES OF THE PERIPHERAL NERVES.

MONONEURITIS.

Definition.—Inflammation of a single nerve trunk.

Etiology.—(*a*) *Wounds,* fractures, dislocations, delivery, violent muscular contractions; disturbed metabolism; anesthesia paralyses, the arms being held over the head, the legs bound in holders or when Esmarch's bandage is applied too tightly; ether hypodermically, malpositions of the arm during sleep and the use of crutches are causes. (*b*) *Contiguous disease* (pleurisy, pelvic inflammation, tumors or aneurysm) may produce neuritis. (*c*) *Cold* (rheumatic paralysis) causes inflammation in the sheath with extension to the nerve. (*d*) *Toxic causes* and infections are factors (*v. i.* Multiple Neuritis).

Pathology.—In *acute cases* the nerve is red, swollen and sometimes the seat of punctate hemorrhages or extravasation of white cells in the sheath or between it and the nerve fibrils. In some cases inflammation involves chiefly the sheath (*perineuritis*) and in others the interstitial tissue (*interstitial neuritis*), in which the enclosed nerve fibrils are compressed. The process is disseminated or focal (where the nerve divides or enters the fascia or bone). It is often difficult to distinguish between inflammation and degeneration (*parenchymatous neuritis);* in both processes there are nuclear multiplication and protoplasmic swelling in the neurilemma; this compresses the myelin sheath, which becomes varicose, granular, fragmented and finally emulsified. The axis-cylinder, composed of thirty to fifty primitive fibrils, becomes granular, divided and may largely or wholly disappear. *Regeneration* is accomplished from the spinal or cerebral centres and probably also from the periphery.

Symptoms.—Constitutional and local symptoms vary with the intensity, extent and seat of the neuritis.

1. Sensory Symptoms.—The chief local symptom is *pain,* experienced locally or eccentrically over the area of distribution of the sensory nerves, increased by tension or movement and usually intense. *Tenderness* indicates involvement of the nervi nervorum. Hyperesthesia, hypesthesia and paresthesia may be noted, but complete anesthesia is rare except for relatively small areas. Sometimes the swollen or nodular nerve may be felt.

2. Motor Symptoms.—The *muscles* supplied are *tender* to touch, painful on movement, *paretic* and atrophied (separation from their trophic

centres). They are less clearly striated, granular and finally indurated. Muscular contractures may result reflexly, especially in traumatic and occupation neuritides. The *reaction of degeneration* is present. The normal nerve is stimulated by the faradic current which produces a continuous muscular contraction and by the voltaic which produces a contraction when the circuit is made or broken—the cathode closing contraction, which is first to appear and stronger than the anode closing contraction. In disease the following changes occur: (*a*) The *muscle* responds weakly and sluggishly to faradism, and tends to maintain contraction after the current is withdrawn. This *modal change* is the essential element in the reaction. (*b*) The *nerve trunk* loses progressively its responsiveness to both galvanism and faradism—a *quantitative change*. (*c*) The *muscle* becomes more excitable by galvanism and less excitable

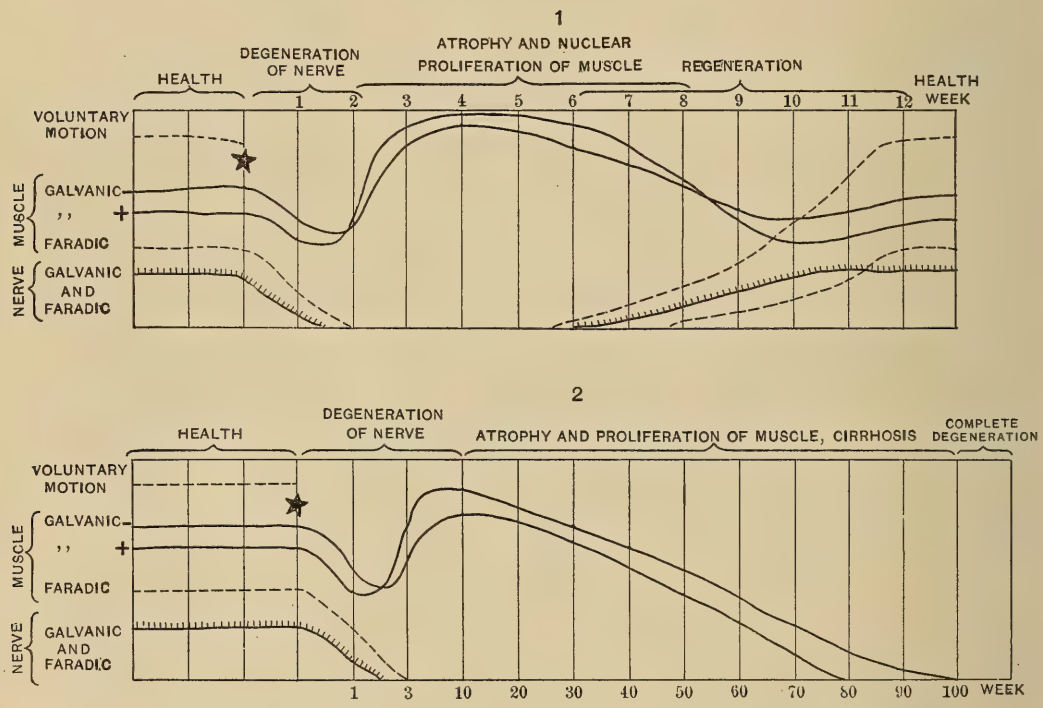


FIG. 75.—1, Paralysis with early return of motion; 2, incurable paralysis with complete degeneration.

by faradism; the latter reaction and the responses in the nerve trunk are completely lost after two or three weeks—a *qualitative change*. (*d*) A *polar change* appears in the muscle about the second week when directly stimulated by galvanism; the muscle is more readily caused to contract by the constant current, but the normal mastering strength of the negative closing contraction over the positive disappears and the positive closing contraction equals or exceeds the negative.

3. TROPHIC SYMPTOMS.—The skin is sometimes red, "glossy," edematous or more rarely thickened. Increased secretion of sweat, eruptions as herpes, erythema or pemphigus, inflammation or adhesions in the joints and perforating ulcers are less common.

Neuritis may sometimes *ascend to the cord*, as in traumatic cases or in bladder disease. In the rare *progressive hypertrophic interstitial neuritis*

of childhood (Déjérine and Sottas, 1893), sclerosis and hypertrophy of the nerves, ant. and post. nerve roots, and sclerosis of the post. columns cause muscular atrophy, pains, anesthesia, nystagmus, immobile pupils and kyphoscoliosis.

Diagnosis.—Diagnosis depends on localization of symptoms to a nerve trunk and its distribution; pain, tenderness and objective sensory alteration; muscular atrophy or paralysis; and trophic changes. The early diffuse pain may simulate disease of the *bones or joints*. Chronic sensory forms may resemble *neuralgia*, in which the pain is paroxysmal, the tender points more localized and objective sensory alteration absent. Disease of the cord or *nerve roots* usually paralyzes several muscles and affects sensation segmentally.

Prognosis.—The prognosis depends on the cause and intensity of the process. Acute cases last for weeks; chronic cases, for months. The outlook is usually good in traumatic cases.

Treatment.—The treatment is (*a*) that of the cause. (*b*) *Absolute rest* is indicated. Exertion increases as it may initiate neuritis. (*c*) *Pain.* The salicylates and iodides are of uncertain value. Local heat may cause burns and ulcerations in the anesthetic skin. Coal-tar products should be given in preference to morphine or cocaine because habits may be contracted; opiates are necessary in severe cases. The positive pole of the voltaic current, just strong enough to be felt, may relieve pain. (*d*) *Paralysis* should be treated only after the acute stage, by the weak interrupted voltaic current (not the faradic), massage and later passive motion (the veins from the nerves empty into the muscle veins).

MULTIPLE NEURITIS.

Polyneuritis, peripheral neuritis, described by James Jackson (1822) and Ladd (1854), was first accurately described pathologically by Dumeil (1864) and Leyden (1880).

Definition.—Neuritis, due to the selective action of toxic substances on the less resistant peripheral nerves, causing (*a*) multiple, symmetrical, flaccid or atrophic paralysis with (*b*) the reaction of degeneration, (*c*) sensory and (*d*) trophic disturbances.

Etiology.—The causes are (*a*) *poisons; alcohol* is the most important; lead, arsenic (in beer), mercury, phosphorus, carbon monoxide, silver, aniline and ergot may also produce the disease. (*b*) The *infections*, acute, as *diphtheria*, typhoid, sepsis and, occasionally, almost every acute infection, and chronic, as *beri-beri, leprosy*, tuberculosis or syphilis, are etiological factors. (*c*) *Intoxications*, as *diabetes, nephritis*, digestive disorders, cachexias and pregnancy, may be causal. (*d*) *Other causes* include the seemingly primary cases. Our present knowledge cannot explain the action of cold and *rheumatism;* probably these cases should be classified as infectious. It may occur with tabes or arteriosclerosis of the nerve vessels. It is a disease of adults (twentieth to fiftieth year); Thomas and Greenbaum, 1907, collected 138 non-diphtheritic cases in children.

Pathology.—In *acute cases* the nerve may be red, swollen and the seat of punctate hemorrhage, exudation of leukocytes or albuminous exudation in the connective tissue, but usually *parenchymatous degeneration* predominates. It appears first and most markedly in the smaller nerves. As in mononeuritis, the axis-cylinders waste and the medullary sheaths disintegrate. The symmetry and multiplicity of degeneration in the nerve endings are caused by the blood condition or toxins. Acute polymyositis of similar origin may accompany neuritis multiplex. In cases which are *chronic* from the beginning there is slow degeneration of the nerves. The connective tissue is increased in certain forms, as gouty or leprous neuritis. The toxic cause may also produce degeneration of the cord or brain and changes in the liver (alcoholic cirrhosis), kidneys (alcohol and lead), etc.

Symptoms.—*Acute cases* may begin with chills, fever, delirium, headache, diarrhea, icterus, splenic tumor, albuminuria or other toxemic symptoms; *chronic cases* develop slowly.

1. Sensory symptoms are usually *first to appear*, the last to disappear and throughout are the most vexing manifestations. Paresthesia precedes pain and pain precedes paralysis. The muscles, nerve trunks and skin are the seat of *spontaneous pain, tenderness to pressure* and *pain on movement.* The palms and soles are especially hyperesthetic in the tabetic, diabetic and diphtheritic forms. *Sensation* is often *blunted,* perverted or less often completely suspended, most markedly *below the elbows and knees,* although higher extension, even to the face, is possible. Some sensory disturbance is rarely absent. Delayed conduction may occur. The joints may be painful, swollen and thickened.

2. Motor Symptoms occur chiefly in the *peripheral* distribution of the *spinal nerves;* they develop first and are most marked in the *extensors* of the *leg and foot, forearm and hand,* and produce the *ankle- and wrist-drop* (ext. popliteal and musculospiral nerves). The foot is also inverted. The paralysis is flaccid. If it is only moderately severe in the legs, the "*steppage*" gait is noted and the patient lifts the legs high to clear the pendant toes ("turkey-gobbler walk"). Paralysis later involves the flexors and the muscles of the arm and thigh. The *reaction of degeneration* sometimes appears before paralysis. The muscles rarely fail to respond to the galvanic current, even in the extreme cases. Then the *muscles atrophy,* chiefly *in distal parts, i. e.,* where the sensory and motor changes predominate. Other motor manifestations are cramps, tremor, fibrillation, ataxia and contractures. The patient lies on the back with the members extended or on the side with flexor contractures, which draw the heels to the buttocks and the thighs to the abdomen. The trunk muscles may be affected as well as those of the neck, eyes, face, tongue or indeed those supplied by any cranial nerve as in the neuritic form of Landry's paralysis; rapid pulse, palpitation, dyspnea and vocal disturbance indicate involvement of the vagus; optic neuritis and amblyopia may occur. The tendon and skin *reflexes decrease or disappear.* The organic reflexes are almost always preserved. Involvement of the sphincters is very rare and referable to the mental state or neuritis of the pudendal plexus.

3. Trophic Symptoms.—The skin is cold, turgid, harsh, sometimes glossy; herpes, erythema, ulceration, bed-sores and trophic changes in the nails or hair are uncommon; edema over the wrists and ankles, especially in alcoholics, is trophic or vascular in origin.

4. General Symptoms.—Mental symptoms and malnutrition may result from the antecedent toxemia or pain; Korsakow's "neuritic psychosis" is marked by amnesia for the recent past, confabulation, delirium and hallucinations; recovery is the rule.

Course and Prognosis.—The common order of symptoms is paresthesia, pain, paralysis, amyotrophy, trophic and mental symptoms. The distal parts are involved more early and intensely than the proximal. The *prognosis* depends in part on the cause. It is good in sensory types and in forms of slow evolution, when the sensorium, arms and trunk, cranial nerves (vagus) and sphincters escape involvement, and when visceral disease, as liver cirrhosis, cord or brain complications, and edema, are absent. The prognosis must cover the possibility of recurrence and the progress of the disease, inevitably slow, requiring a year, despite treatment.

Diagnosis.—The *cardinal signs* are (*a*) the motor and sensory symptoms, which correspond in function, peripheral distribution and symmetry; (*b*) the reaction of degeneration; (*c*) tenderness of the nerves and muscles; (*d*) loss of reflexes and (*e*) the etiology.

Diagnosis of Type.—(*a*) The *alcoholic* form is the most frequent; 70 per cent. of cases occur in women; the legs are often involved; there is the "steppage" gait; atactic, sensory and mental symptoms are marked. (*b*) In *lead poisoning* the occupation, lead line, colic and constipation are characteristic; the arms alone are involved as a rule; there is little sensory participation; the involvement rarely extends; the wasting is conspicuous. (*c*) *Diphtheritic* form (*q. v.*). (*d*) In the *toxemic* form with fever and constitutional reaction, it begins in the legs or all members simultaneously, involves equally the flexors and is attended by slight sensory symptoms. (*e*) *Beri-beri, leprous neuritis,* erythromelalgia and Dercum's disease are considered elsewhere.

Differentiation.—Atactic polyneuritis (pseudotabes, Déjérine) with little motor disturbance, may resemble locomotor ataxia:

Locomotor Ataxia.		Multiple Neuritis.
History or stigmata of syphilis.		Rare; infection or intoxication.
Argyll-Robertson pupil		Loss of accommodation in diphtheria.
Optic atrophy, 10 per cent.		Rare, but toxic amaurosis is frequent.
Eye paralysis frequent.		Much more rare.
Girdle sensation and lightning pains.		Very rare.
Gait ataxic, stamping, striking heel first.		Disturbance motor. Striking toe first.
Sensory symptoms predominate.		Motor predominate.
Paralysis, reaction of degeneration and amyotrophy rare.		In nearly all cases; rapid in development and symmetrical.
Nerve trunks often insensitive.		Tender, as are muscles.
Arthropathies, osteopathies.		Unknown; glossy skin more common.
Crises.		Almost unknown; toxemic vomiting, etc.
Sphincters frequently involved.		Very rarely, and from delirium.
Clinical evolution very slow.		Rapid.
Disease incurable.		Recovery usual, if not early death.

Poliomyelitis.	*vs.*	Multiple Neuritis.
Usually in children.		In adults.
More sudden onset.		Gradual.
Embraces whole limb, at random.		Strictly peripheral and symmetrical.
Muscles functionating together. Reaction of degeneration in both, but strong galvanic current not required to produce muscular contraction.		Dissimilar muscles involved. Muscles respond only to strong galvanic current.
Pain and tenderness rare.		Common in nerves and muscles.
Sensory disturbance rare.		Usually (but not invariably) present.
Immediate tendency to regress, but usually some deformity.		Tendency to progress. Total recovery the rule.

In *acute myelitis* the onset is acute, there are girdle pains, paraplegia, with corresponding anesthesia, bed-sores, disturbance of the bladder and rectum; there is no tenderness of the nerves or muscles, little or no ataxia and recovery is unusual. *Landry's paralysis* often cannot be distinguished (one form of Landry's disease is neuritic).

Treatment.—Alcohol must always be considered and deception guarded against; it should be withdrawn at once. For pain see Neuritis. Absolute rest should be enforced. A full diet, rich in fats, should be given if the stomach is tolerant; tonics, as iron, maintain nutrition, but arsenic should be avoided. When the lower cranial nerves are affected, feeding by the nasal tube or by rectum, measures to avoid hypostasis, as in typhoid, and cardiac stimulants are indicated. To overcome contractures from ankle- or wrist-drop, splints or sand-bags are employed when they do not cause excessive pain, and frames to lift the bedclothes from the feet. Gentle massage and voltaism may be tolerated after the acute stage, may prevent contractures and aid the return venous flow. Bromides and chloral are the most certain somnifacients, but chloral requires care in chronic alcoholism.

NEUROMA.

Two varieties exist, the true and the false. The true neuroma may be medullated or non-medullated, though ganglion cells are exceptional; they are usually multiple, sometimes very numerous (3020 in one case) and have been found on every cranial, spinal and sympathetic nerve (von Recklinghausen's disease, of which Adrian assembled 447 cases). They may undergo sarcomatous degeneration. The *tubercula dolorosa* (Virchow) are small, painful, subcutaneous nodes on the trunk or extremities, the anatomy of which is obscure. The congenital *neuroma plexiforme* consists of twisted cords with nodes and occurs largely in the distribution of the fifth nerve. The *false neuroma* is usually fibrous; glioma is rare; sarcoma or carcinoma may by contiguity invade adjacent trunks; neuromata have been found in the cerebral nerves in syphilis and in the peripheral nerves in leprosy. The nerve trunks suffer more in the false than in the true form. *Neuromata in amputation stumps* are frequent.

Symptoms and Treatment.—There may be no *symptoms;* local tenderness and local or projected pain are frequent. Reflex muscular spasm is common from nerve irritation. Mentality, sensation or motility may be affected. Pigmentation is usual. *Treatment* is symptomatic for the exhausting pain or radical in localized growths.

DISEASES OF THE CRANIAL NERVES.

OLFACTORY NERVE.

The end nerves located on the two upper turbinate bodies and the upper septum, run to the olfactory bulbs, which are part of the brain, thence to the centre in the uncinate gyrus and hippocampus, with relations to the optic thalamus, opposite internal capsule and cerebral cortex. The anterior nares perceive odors, the posterior flavors.

Anosmia is loss of smell. Tests are made not with pungent substances, as ammonia, which irritate the fifth nerve, but with musk, asafetida, cologne-water and the essential oils. (*a*) Disease in the nasal mucosa is the most frequent cause—from paralysis of the fifth nerve, arrest of secretion or atrophy. (*b*) Disease may result from congenital absence of the bulb, basal fracture or meningitis, tabes, foci in the opposite internal capsule, etc. (*c*) Central disease, as softening, may develop in the hippocampus and uncinate convolution. The prognosis is unfavorable and treatment ineffectual. *Hyperosmia,* oversensitiveness of smell, occurs in insane and neurotic subjects; it may be cultivated, so that individuals are recognized by their odor. *Parosmia,* perverted sensation, is rare and arises from irritation of the nerves or centres. Hallucinations of smell may occur, as of burning flesh in the epileptic aura.

OPTIC NERVE.

1. The Retina.—*Retinitis* occurs in general diseases, as nephritis, especially late in interstitial forms. The degenerative form is most common, next the hemorrhagic form, with "flame-like" hemorrhages along the vessels, and finally the inflammatory type, with marked swelling of the retina, serum exudation, cloudiness and disk blurring; the white foci are due to exudation, fatty change or sclerosis. Similar findings may occur in syphilis, anemia, malaria, leukemia, plumbism, diabetes, etc. (see Plates IV and VI). There are apparently primary cases.

2. Optic Nerve.—*Optic neuritis* (papillitis, choked disk) has been described (see Brain Tumor and Plates IV and VI). Tumors or inflammation in or back of the orbit or errors in refraction may be causal. In the *primary form* there is no initial inflammation. The edges of the disk are distinct, its arteries nearly normal and its color grayish; it may develop hereditarily in males at puberty; it occurs in tabes, paretic dementia, multiple sclerosis, syphilis and less often from diabetes, alcoholism, plumbism, specific fevers, amaurotic family idiocy and familial cerebellar ataxia. The *form secondary to inflammation* commonly follows choked disk and has the same significance; the disk has an irregular contour, small arteries and is translucent. The symptoms are diminution in acuity of vision, field of vision, color perception and the pupillary reaction.

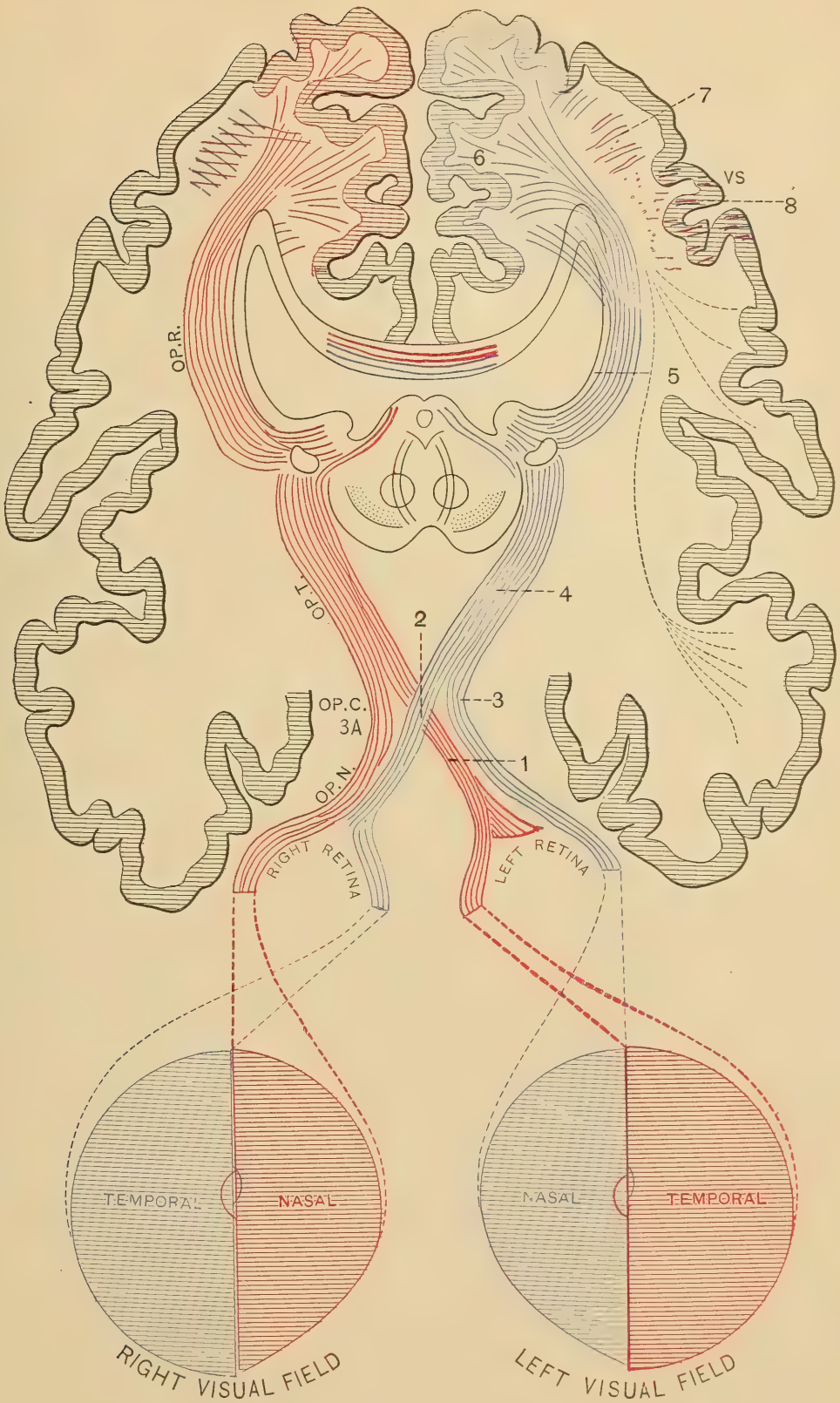
3. Chiasm.—Each half of the retina contains fibers from the corresponding occipital lobe (see Plates XXIII), that is, each right half receives fibers from the right and each left half from the left occipital lobe. The

outer temporal portion of the field of vision is more limited than the inner nasal portion, because the nose cuts off light and it receives fewer nerve fibers. The larger number of fibers from each eye, *i. e.*, those from the nasal retinal half, decussate at the chiasm and lie in its centre. Those from the temporal half are direct and lie at the side of the chiasm. While disease of one optic nerve causes blindness in the corresponding eye, lesions affecting the centre of the chiasm (decussating fibers to the nasal half of each retina) cause half-blindness in the opposite (temporal) visual fields, *bitemporal hemianopsia,* since oblique rays passing the pupil fall on the opposite half of the retina. The macula, the point of sharpest vision, receives fibers from both sides of the brain and escapes involvement. Temporal hemianopsia may result from tumors of the pituitary gland, pressure from the third ventricle, gummata and hemorrhage. The "oscillating" or varying bitemporal hemianopsia often indicates syphilis. The diagram illustrates effects of lesions of the lateral part of the chiasm (*3*), lesions affecting both sides of the chiasm (*3* and *3A*), as calcareous or aneurysmatic carotids; *binasal hemianopsia* is most exceptional; a large lesion might produce temporal hemianopsia in one eye and total blindness in the other; still more extensive disease might produce total blindness.

4. Optic Tract.—Just back of the chiasm the temporal fibers of the right and the nasal fibers of the left side form the right optic tract; *i. e.*, the right tract contains all fibers from the right half of each retina. The tract runs over the crus cerebri, where a lesion very often causes hemiplegia on the same side as the blind field. The fibers enter the geniculate bodies (in which 80 per cent. of the optic fibers end), opt. thalamus and ant. quadrigeminate body, from which gray ganglia the fibers pass by way of the posterior limb of the internal capsule and, by the optic radiation, to the visual centre in the occipital lobe, the cuneus. A lesion in the optic tract anywhere between the chiasm and cuneus produces *homonymous hemianopsia* (at *5*, in diagram); for instance, if it occurs in the right optic tract it produces loss of function in the temporal half of the right and nasal half of the left retina, so that the left half of each field of vision is lacking. Hemianopsia may be partial, or only a quadrant may remain after partial recovery. *Heteronymous hemianopsia* designates blindness of the right half of one field and the left half of the other or the converse. Disease in the gray ganglia or internal capsule may produce contralateral hemiplegia; in 50 per cent. of cases of hemianopsia this association is observed, and less frequently hemianesthesia. The optic tract is injured in tumors, multiple sclerosis, trauma, hemorrhage or softening.

5. Optic Centre (See page 753).—A lesion in the cuneus produces (*a*) *homonymous hemianopsia.* Sometimes only an upper or lower quadrant is blind. Purely cortical hemianopsia is impossible, Monakow holding that the optic radiation must be involved; (*b*) *hemichromatopsia,* or homonymous color-blindness or confusion, in which all colors seem gray, is probably due to lesion of the anterior superficial cuneal cortex. Other results are (*c*) *mind-blindness;* (*d*) *alexia;* (*e*) *optic aphasia;* and (*f*) crossed *amblyopia,* concentric blurring or limitation of the visual fields

PLATE XXIII



Visual Paths. (After Vialet.)

OP.N., optic nerve. *OP.C.*, optic chiasm; *OP.T.*, optic tract; *OP.R.*, optic radiation; *V.S.*, visual speech centre. A lesion at (1) causes total blindness in that eye; lesion at (2), bitemporal hemianopsia; at (3), unilateral nasal, and lesions at (3) and (3*A*), bilateral nasal hemianopsia; at (4), hemianopsia of both eyes and the hemianopsic pupillary reaction; at (5) or (6), hemianopsia of both eyes (pupillary reflexes being normal; at (7), amblyopia, especially of the opposite eye; at (8), word-blindness.

for form and color; this is usually referred, however, to disease of the gyrus angularis, in which there is thought to be a higher centre in which the half-fields are combined and the whole opposite field and part of the field of the same side are represented.

Diagnosis.—The eyes should be tested separately. Disease of *an optic nerve* causes loss of sight and pupillary reaction in the corresponding eye. *Central scotoma* (blindness in the centre of the visual field) indicates inflammation or hemorrhage in the central (axial) fibers of the nerve, usually back of the orbit; it is common in poisoning from tobacco and alcohol. *Peripheral limitation* of the visual fields indicates damage to fibers coursing in the periphery of the optic nerve and some damage to all the nerve fibers. *Sectorial blindness* indicates a pronounced but circumscribed affection of the nerve. *Bitemporal hemianopsia* always results from chiasm disease; unilateral temporal hemianopsia in one eye with complete blindness in the other has the same significance, but also implies that the process has extended laterally, possibly even to the optic tract. *Homonymous hemianopsia* indicates disease of the optic tract between the chiasm and cuneus. If the geniculate (or quadrigeminate) bodies are the seat of the lesion, *Wernicke's hemianopsic pupillary reaction* is obtained; a small beam of light in a dark room should be thrown on the hemianopsic half of the retina and the patient should look at a distance to eliminate accommodation; if the pupil does not react to light on the blind half (Wernicke's reaction) the disease focus lies at or to one side of the geniculate or quadrigeminate bodies. If the lesion lies between these ganglia and the cortex, the reaction of Wernicke is absent. Transient hemianopsia is common in vascular lesions; if it lasts for weeks it will probably remain. In Sequin's case it was present for twenty-three years. It may occur in migraine and very rarely in hysteria. Its association with the crus, internal capsule or cortex symptoms already considered is of localizing importance. *Amblyopia and concentric limitation* in the fields, marked in one eye and less in the other, may result from (*a*) optic atrophy, with the absent pupillary reaction, and seen with the ophthalmoscope; or (*b*) disease of one hemisphere, the ophthalmoscopic finding and pupillary reflex being normal.

Functional and Toxic Blindness.—(*a*) Hysterical blindness is transitory and rarely complete; the eye functionates normally, though the visual centre neglects the impression; it is often associated with loss or inversion of the color perception and with hemianesthesia. (*b*) *In amblyopia* from tobacco, vision is gradually impaired in both eyes, largely as central scotoma with invariable loss of perception for red and green colors; the fundus is usually normal, sometimes congested; recovery is usual, but atrophy may result. (*c*) *Toxic amaurosis*, usually transitory, may occur from uremia, poisoning by lead, alcohol or quinine, syphilis, acute anemia, etc.

OCULAR PARALYSIS; THIRD, FOURTH AND SIXTH NERVES.

General Etiology.—(*a*) *Trauma* to the eye, orbit or base may cause ocular paralysis, characterized by a slow course and often imperfect

recovery. (*b*) *Compression* by tumors in the orbit which often produce exophthalmos; tumors at the base, aneurysm and rarely hemorrhage into the nerve sheath may produce it. (*c*) *Neuritis* produced by the above factors, orbital cellulitis, meningitis, syphilis, cavernous thrombosis, alcoholism and acute infections, as diphtheria may be etiological factors; exophthalmic goitre, plumbism, and diabetes are rarer causes. (*d*) *Nuclear disease* is usually due to chronic wasting disease, as in tabes, multiple sclerosis or bulbar palsy, but sometimes to acute lesions, polioencephalitic or vascular. (*e*) *Supranuclear disease* (*v. i.*) or (*f*) in rare cases muscular dystrophy may be a cause.

Anatomy and Physiology.—The nerve supply is by the sympathetic (which through the ciliary ganglion dilates the pupil) and by the third, fourth and sixth cranial pairs, which arise from the middle ventricle, Sylvian aqueduct and fourth ventricle and are arranged as follows from before backward: (*a*) the third nerve nucleus with the ciliary and behind it the pupillary centre, then its muscles in this order: the internal rectus, levator palpeb. superior, inferior rectus and inferior oblique; (*b*) fourth nerve nucleus for the superior oblique and (*c*) nucleus of the sixth nerve for the ext. rectus. Their connection with the motor cortex is through the internal capsule and corona radiata. The third nucleus is connected with the seventh; winking and movements of the eyelid are thus associated with the pupil reaction and accommodation. The action of the muscles is indicated by their names but convergence is supplemented by the sup. and inf. recti and the two oblique muscles. The inf. oblique turns the eye upward, the sup. oblique downward. The upper lid is raised by the levator palp. sup. (acting usually with the sup. rectus) and by Müller's muscle.

Paralysis of the Third Nerve.—This nerve emerging from the crus follows the cavernous sinus, and therefore may be involved in disease of these structures. A complete paralysis results in drooping of the lid (ptosis), which the patient attempts to overcome by overaction of the frontalis; the eye can be moved only outward and a little down and inward (ext. rectus and sup. oblique). Accommodation is lost, the pupil is wide from the unopposed action of the sympathetic nerve and does not react to light. There is *double vision from divergent strabismus*, due to action of the unrestrained ext. rectus muscle. Paralysis is often partial; the levator palp. sup. may escape involvement or be affected alone or with its associate (the sup. rectus), especially in bilateral nuclear disease. *Ptosis* may be (*a*) part of an ordinary oculomotor palsy; (*b*) part of a chronic ophthalmoplegia (*v. i.*); (*c*) reflex in rare cases of trigeminal irritation, as from disease of the teeth; (*d*) myopathic, as in muscular dystrophy; (*e*) a result of sympathetic paralysis, false or pseudoptosis (with a red, edematous skin, slight recession of the eye-ball and contracted pupil); (*f*) an incurable congenital form, usually partial and bilateral; (*g*) a transient form, observed in the morning in women, usually with a good prognosis; (*h*) an hysterical form, usually bilateral, in various associations with the sup. rectus, inf. or int. rectus, the eyebrow being frequently lower.

Internal Oculomotor Palsy. — Internal oculomotor palsy includes cycloplegia and iridoplegia. (*a*) *Cycloplegia* (paralysis of the ciliary muscle) results in loss of accommodation. Near vision is poor, distant vision is good; extreme myopia or presbyopia make its detection difficult. Isolated cycloplegia in one eye may indicate disease of the lenticular ganglion; in both eyes it is nuclear, as in diphtheria or tabes. (*b*) *Iridoplegia* may assume three forms: The first is loss of associated action, *accommodation iridoplegia*, in which there is no pupillary narrowing in the act of accommodation. It is due to the same causes as cycloplegia and is usually associated with it. The second is *loss of the light reflex*, reflex iridoplegia, the Argyll-Robertson pupil, seen especially in tabes and paretic dementia (very seldom in softening, tumors, senile atrophy, hydrocephalus or congenitally). The reflex occurs through the optic nerve, optic tracts, corp. quadrigemina or corp. geniculata, anterior part of the oculomotor nucleus, probably the second branch of origin of the third nerve, its trunk, the ciliary ganglion and its nerves. The pupils are often equally affected and small (spinal myosis) but may be unequal (anisocoria, also observed in some normal persons). If the pupils are not too small, cutaneous iridoplegia is not present. The third form is *loss of the skin reflex*, cutaneous iridoplegia, in which the normal dilatation of the pupil upon pinching, sticking or faradizing the skin of the neck is lost. It indicates, in general terms, disease in the cervical sympathetic nerve, in the fibers between it and the cervical cord, diseases of the cord affecting sensation, or of the centre (probably in the corp. quadrigemina outside the light reflex centre in the trunks of the first and third nerves). The seat of lesion is probably nuclear, unless special localizing symptoms indicate otherwise. Specifically it is characteristic of tabes, paretic dementia or syphilis.

Recurrent Palsy.—Periodic palsy, to which Gubler (1860) and Möbius drew attention, is a rare form, involving most of the third, and rarely the sixth nerve. It occurs chiefly in women. At intervals of months to years, possibly throughout life, a recurrent paralysis develops for a few days or weeks and disappears. Forms which begin with headache, pain and vomiting were called *ophthalmoplegic migraine* by Charcot. Its nature is unknown (peripheral or nuclear); von Monakow holds that its cause is tumor, meningitis or tubercle; the lesion affects the eye nerves (or centres) and the descending branch of the fifth; few cases are seemingly hysterical.

Paralysis of the Fourth (Trochlear) Nerve.—Paralysis of the superior oblique is of little importance. In attempts to converge downward the normal inward rotation of the eye-ball is lacking.

Paralysis of the Sixth Nerve.—The eye cannot be moved outward, and is rotated inward by the unopposed internal rectus, marked *diplopia* resulting. This nerve is frequently involved, because of its long and exposed course. The muscles of the eye are involved in the following order of frequency: 1, ext. rectus; 2, sup. oblique; 3, inf. rectus; 4, sup. rectus; 5, int. rectus; 6, inf. oblique (von Gräfe).

Combined Eye Paralysis.—Convergence and accommodation are associated functionally and their centres lie adjacent, whence they are often

lost together. The internal recti may fail to contract together but may move separately in lateral eye movements. *Conjugate deviation* may occur (see pages 761, 769); if a lesion occur above the sixth nucleus, the eyes cannot be directed toward the side of lesion and are directed toward the paralyzed side; if the lesion is at the sixth nucleus the sixth nerve on that side is paralyzed, often also the seventh, and the opposite third nerve is paralyzed, not from lesion in the third nucleus but from the fact that each third nucleus receives afferent impulses from the contralateral sixth nucleus for associated eye movements.

Symptoms.—The *symptoms of paralysis* are five:

1. *Limitation of movement* in the paralyzed muscle, followed by secondary contraction of the unresisted opponent. The habitual position of the head corresponds in every way to the physiological action of the paralyzed muscle (Landholt).

2. *Strabismus* (squint), causing lack of parallelism in the visual axes, which are divergent when the int. rectus and convergent when the ext. rectus is affected; this constitutes the *primary deviation.*

3. *Secondary deviation* is the extra movement of the sound eye in its efforts to see, due to its increased innervation, detected by testing with the diseased eye closed or by testing with ground-glass over the sound one.

4. *Erroneous projection* in the paralyzed eye. Distance is normally judged by the position of the eye-balls and muscles and therefore when secondary deviation from excessive innervation of the sound muscles occurs, it increases the impression of distance.

5. *Diplopia* (double vision) results from failure of the visual axes to correspond. The sound eye sees the true image, while the paralyzed eye sees the false one, which is less clear, since the image falls on a less sensitive retinal area. When the images are either near together or else widely separated the false one may be "neglected." Erroneous projection and diplopia always produce vertigo at first. If the false image is on the same side of the true image as the eye by which it is seen, the diplopia is simple or homonymous; if on the other side, it is crossed or heteronymous. The eyes are tested by holding red glass before one eye and testing vision with a strip of white paper. If the red image and covered eye are on the same side the diplopia is simple; if on opposite sides it is crossed. When the eyes converge it is simple, when they diverge it is crossed; *i. e.*, "when the prolonged visual axes cross, the diplopia is not crossed." Holding the test object stationary and the head being turned in one way or another, in some directions the images converge or perhaps blend, while in the opposite direction they separate. "The affected eye is that one in the direction of whose image the diplopia increases; the paralyzed muscle is the one which would give to the eye the direction of the false image" (Landholt). Monocular diplopia is hysterical (unless there is disease of the eye media or error in curvature).

Treatment.—Treatment is often unsatisfactory from the etiological standpoint. Surgical interference or specific treatment is sometimes indicated, but syphilitic forms (*q. v.*) often recur or become permanent. In acute forms counter-irritation back of the ear by blisters, leeches or

hot fomentations is indicated. Some find mercury useful in peripheral forms. Galvanism is the best form of electrotherapy, but is seldom beneficial. Prisms and tenotomy are sometimes helpful.

Progressive Nuclear Ophthalmoplegia.—Nuclear palsy was first described clinically by von Gräfe (1856) and pathologically by Hutchinson and Gowers. The etiology is not clear; syphilis (in 17 per cent.), lead, alcohol and toxemia, as from diabetes, grippe or diphtheria, are possible causes. A few cases are hereditary or congenital. Most cases develop in males under thirty years of age. The *pathology* usually consists of degenerative nuclear atrophy resembling bulbar paralysis; sometimes compression or meningitis is found or rarely no anatomical alteration is detected, the functional form.

Symptoms.—The symptoms are those of a slowly progressive paralysis of the eye muscles; ophthalmoplegia is called external when it involves the extrinsic muscles, internal when it involves the pupil, or total when both are concerned. The *eye muscles* are *successively affected,* but in no given order. The *facies of Hutchinson* is peculiar, staring, from immobility of the eye-balls, or sleepy from partial bilateral ptosis. Diplopia is uncommon, or only an early symptom, because of the chronicity of the process. Associated muscles are often affected by groups, because of their successive nuclear involvement, as the pupil and convergence, one internal and the opposite external rectus, the two internal recti and the sup. rectus and levator. Chronic ophthalmoplegia may be associated with the analogous *bulbar paralysis* or *progressive spinal muscular atrophy;* with *optic atrophy* or with *double facial paralysis* (the rare and obscure infantile or congenital oculofacial paralysis, without pupillary involvement, of which Möbius collected 44 cases); it may be a part or a precursor of tabes, paralytic dementia or multiple sclerosis.

Diagnosis.—Nuclear involvement is probable when there is partial and bilateral ptosis, escape of the pupil, or involvement of one or more conjugate groups. The lesion may be supranuclear, when the head and limbs are involved, exceptionally when there is isolated convergence paralysis or paralysis of up-and-down movements with ptosis. Apoplectiform nuclear palsy from basilar thrombosis is usually asymmetrical, acute and associated with hemiplegia. Wernicke's superior poliencephalitis is distinguished by its acuity and toxemia. Ophthalmoplegia from syphilitic meningitis, polyneuritis, tumors (corp. quadrigem.) and orbital disease, is generally distinguished by the relatively acute course and concomitant symptoms.

Prognosis.—The disease is very chronic, possibly lasting twenty to forty years and is progressive. Arrest is possible, but can never be predicted. The disease itself causes little risk to life, but its associates are dangerous, as tabes or psychoses, which develop in 18 per cent.

Treatment.—Treatment is generally ineffectual. Iodides and mercury are useless in nuclear disease, but may be given if the localization is not definitely known.

Sympathetic Paralysis.—This disease was first described by Horner; it produces *myosis,* due to paralysis of the dilator iridis, and slight *ptosis,* due to paresis of Müller's muscle. The ocular tension is decreased,

the cheek wasted and the skin reddened and warmer than normal. *Irritation of the sympathetic* is characterized by wide pupils, widening of the interpalpebral space and exophthalmos.

Ocular Muscular Spasms.—Spasms occur in conjugate deviation, irritation of the nerve trunks when compressed by tumor, and rarely in chorea, facial tic and spasmodic torticollis. In hysteria the eyes may be rolled upward and to one side, concealing the iris, or they are often fixed in convergence; they never diverge; what appears to be ptosis is really spasm of the orbicularis. Convergent spasm may also occur in hypermetropia and divergent spasm in myopia. Isolated spasm of the levator palpebræ may occur in irritation of the cervical sympathetic or fifth nerve, in advanced years or Graves's disease. Secondary deviation (*v. s.*) is spasmodic, as is also *nystagmus*, which latter consists of rapid, rhythmic, clonic, involuntary oscillations, due to muscular spasm with alternating contraction of the opposing muscles. It is continuous, as distinguished from the "tremor of weakness" which occurs in paretic muscles on movement. It is usually horizontal, less often rotatory and rarely vertical. Its mechanism is probably centric. The *causes* are (*a*) disease of the eye, as opacities and choroiditis; (*b*) it occurs frequently in albinos, and (*c*) in miners, from working with the head down and eyes strained to one side; (*d*) it occurs as a cardinal sign in multiple sclerosis and Friedreich's ataxia, as a common sign in tumor of the cerebellum, corp. quadrigem. and opt. thalamus, occasionally in meningeal disease, sinus thrombosis, hemorrhage or softening and very rarely in hysteria.

FIFTH NERVE (TRIGEMINUS; TRIFACIAL NERVE).

The nerve originates from its motor nucleus in the pons, from motor fibers descending from the corpora quadrigemina, and from sensory fibers ascending from the medulla. It is connected with the motor cortex and cerebellum. The motor and sensory fibers leave the pons separately and join after the sensory trunk has passed through the Gasserian ganglion, which has three branches; (1) which passes to the orbit by the sphenoidal fissure, supplying the skin of the forehead, anterior scalp, upper lid, bridge and top of the nose; it is connected with the cervical sympathetic nerve; (2) which passes by the foramen ovale to the sphenomaxillary fossa to supply the upper and lower lids, cheek, forepart of the temples, side of the nose, upper teeth, gums, pharynx, tonsils, soft palate, uvula and roof of the mouth; it has connections with the sympathetic nerve and the facial nerve by the Vidian; (3) which passes by the foramen rotundum and sphenomaxillary fossa supplying the rest of the temples, anterior and upper parts of the ear, meatus, lower cheek, lower lip, lower teeth, gums, chin, tongue, oral mucosa, salivary glands, and as a motor nerve to the muscles of the lower jaw, temporals, masseters, pterygoids, mylohyoids and post. belly of the digastric. The lingual branch gives off the chorda tympani which joins the facial nerve. From the otic ganglion arises the small superficial petrosal nerve which connects with the facial nerve and ends in the tympanic branches of the glossopharyngeal nerve.

Causes of Paralysis.—(*a*) Lesions of the cortex in very rare cases. (*b*) Focal disease in the pons, as hemorrhage, tumors and less often tabes or bulbar paralysis. (*c*) Diseases of the base, as syphilis, meningitis, tumors, caries or aneurysms. (*d*) Affections involving the first branch by cavernous thrombosis, pituitary tumor, aneurysm or orbital disease, or the second and third branches by lesions in the sphenomaxillary fossa. (*e*) Trauma, puncture or bullet wounds, but (*f*) rarely fractures, or neuritis, unless it is secondary.

Symptoms.—1. Sensory Portion.—Anesthesia, generally unilateral, is found in one or all branches, and tactile sensation is more involved than the sense of pain. It is often preceded by tenderness along the nerves, irritation or neuralgia. In complete cases the skin and mucosa of the mouth, nose and conjunctiva are anesthetic. The tongue is furred on its anesthetic half, because the food is not chewed on that side. The conjunctival reflex is abolished. The secretions are increased by irritation or decreased by paralysis. Smell is blunted and taste is lost (*v. i.*). Sometimes there is redness, cyanosis or swelling of the face or the teeth may be loosened. Two trophic manifestations are important. The first is neuroparalytic ophthalmia, which may lead to ulceration of the eye or loss of the bulb; it is due not simply to conjunctival anesthesia, which predisposes to inflammation because of foreign particles remaining unnoticed in the eye, but is rather due to irritation of the fifth nerve or Gasserian ganglion. It has not occurred in many cases in which the ganglion has been surgically removed. The second is herpes zoster, which is most common in disease of the first branch, results from irritation of the Gasserian ganglion or the nerves just anterior to it, is often preceded by pain and sometimes leads to iritis or ophthalmitis.

2. Motor Portion.—Placing the fingers on the temporal and masseter muscles, when the patient attempts to close the teeth, reveals their failure to contract. Paralysis of the ext. pterygoid is shown by failure to move the jaw toward the sound side and by deviation toward the paralyzed side, when the jaw is depressed. The wasted muscles are too deep to elicit distinctly the reaction of degeneration.

3. Gustatory Portion.—The sense of taste covers only bitter, sweet, sour, salty and metallic substances, while flavors are recognized by the olfactory nerve. The tip and edges of the tongue detect saltiness or sourness; its base, bitterness and sweetness. It is often stated that the fifth nerve supplies the anterior two-thirds of the tongue and the glossopharyngeal nerve the posterior third and the palate; but glossopharyngeal disease alone does not destroy the sense of taste. In a study of cases of Gasserian extirpation, Cushing decides that taste is only temporarily lost and that it does not travel by the fifth nerve. Disease of the lingual nerve (from the fifth), after the chorda tympani joins it, produces loss of taste (*ageusia*); disease of the lingual above the juncture or disease of the central part of the fifth does not result in loss of taste. It is thought that the gustatory fibers of the fifth run with the chorda to the seventh nerve which they leave by the petrosal nerve to again reach the fifth through Meckel's ganglion. Anesthesia of the tongue may result from facial neuritis and middle-ear disease.

Diagnosis.—Organic irritation characterized by pain alone may be difficult to differentiate from trifacial neuralgia, although there is less radiation and more hyperesthesia in organic irritation. When all branches are anesthetic the disease probably lies at the base of the brain or in the Gasserian ganglion. In disease of the mid-pons the first division alone may be affected; in disease at its side all divisions are involved, often with implication of the third, sixth or less often seventh and eighth nerves.

Treatment.—Counter-irritation may induce ulceration. Galvanization is said to be beneficial. Gelsemium, cocaine and morphine must be used at times because of severe pain, but the acquisition of drug habits must always be borne in mind. Boric acid and castor oil should be dropped in the eye several times daily. Antisyphilitic remedies are tried in doubtful cases.

Masticatory Spasm.—Romberg's spasm is not common. In the *tonic type* (trismus) the spasm is usually bilateral and the tense muscles are hard and may be tender. The cause may be tetanus, hysteria, epilepsy, tetany, tumor in the pons, cortical hemorrhage, or it may be reflex from trigeminal irritation, as from temporomaxillary disease or dental caries. The rare *clonic type* may be serial or intermittent; chattering of the teeth is an example. It may occur in chorea, epilepsy or hysteria. Grinding of the teeth occurs in paretic dementia, meningitis or typhoidal states. *Treatment* consists of tonics, galvanization, blisters and morphine and cocaine locally.

Progressive Facial Hemiatrophy.—Of this affection, first described by Parry (1825), about 200 cases are reported. Its pathogenesis is obscure, but it is described here because the only autopsy on record showed neuritis of the ascending root of the fifth nerve. Möbius holds that acute infections, as measles or scarlatina, are causal, some virus entering through the skin or tonsils and resulting in atrophy. The essential change is a *facial hemiatrophy*, which develops most often in persons between ten and fifteen years of age, in girls (66 per cent.) and in 60 per cent. affects the left half of the face. In some half-dozen cases the atrophy was bilateral. The cardinal sign is *atrophy of the skin*, which is blanched and shrunken; its *fat disappears* and later the *muscles waste* and the rough skin lies directly on the bone. The hair, eyebrows and beard on the affected side lose pigment and may fall out, sweat secretion is lessened and the mouth is drawn toward the side of lesion. The *bones waste*, especially in young subjects, most notably the maxillæ, but even the smaller bones and nasal cartilages. On the wasted side there may be no vasomotor reaction; sensation is usually normal, although pain or paresthesia may be noted. The tongue, tonsils and palate are rarely involved. The course is *very chronic* and practically always progressive, possibly with remissions. The outlook is hopeless. There is no treatment other than thyroid extract; the injection of paraffin to lessen the facial deformity is dangerous.

Facial Hemihypertrophy.—Facial hemihypertrophy, of which only 13 cases are on record, affects largely the cheek; Werner collected 9 congenital cases. Irritative neuritis is its suspected cause.

SEVENTH OR FACIAL NERVE.

The face is represented in the lower Rolandic cortex, whence the fibers run through the corona, internal capsule, crus and pons (*q. v.*), where they decussate and enter the nucleus of the opposite side. The nucleus lies in the floor of the fourth ventricle inside of the ascending branch of the fifth nerve and receives fibers from the third nucleus above for the orbicularis of the eye (whereby ocular movements, winking and accommodation are coördinated) and fibers from the hypoglossus below for the orbicularis oris (whereby correlation of lingual and labial movements is effected). It curves around the sixth nucleus, emerging near the juncture of the pons and medulla, and enters the auditory meatus with the eighth nerve, bends sharply, and presents a ganglionic swelling which receives the large superficial petrosal from the Vidian nerve, containing fibers of taste from the fifth nerve. These latter again leave the facial nerve by the chorda tampani. In the Fallopian canal the facial nerve gives off branches as follows: (*a*) a motor branch to the tympanic plexus; (*b*) a motor twig to the stapedius muscle; (*c*) the chorda tympani nerve with secretory branches to the salivary glands. Passing through the stylomastoid foramen, it supplies (i) a few sensory branches to the external ear; (ii) motor fibers to the external auricular muscles; (iii) motor fibers to the posterior part of the occipitofrontalis; and (iv) in the parotid gland breaks up into branches which supply all the muscles of the face, the platysma, stylohyoid and post. belly of the digastric.

Peripheral Facial Paralysis.—Prosoplegia, Bell's paralysis (Bell, 1830), is the most frequent peripheral palsy.

Etiology.—1. *Neuritis* is the usual cause. Exposure to cold ("rheumatism") produces 73 per cent. of cases, especially in men between the twentieth and fortieth years. Angina, gout, diabetes, diphtheria, typhoid, erysipelas, mumps, sepsis, etc., are occasional causes.

2. Disease of the petrous portion of the *temporal bone* (7 per cent.), especially caries and otitis media in childhood, is dangerous in proportion to the extent of the bone disease; 88 per cent. of cases of labyrinth disease cause facial paralysis.

3. *Trauma* causes 6 per cent. of cases, as basal fracture by tearing, hemorrhage, inflammation or undue formation of callus; much more rarely meningeal or cerebellar hemorrhage at birth; the use of obstetrical forceps and trauma to the jaw or surgical operations on the parotid gland. Facial paralysis may occur in "head tetanus."

4. In intracranial lesions *at the base*, syphilis (3 per cent.) should always be considered; syphilis rarely causes peripheral facial paralysis. Aneurysm, tumor, meningitis and hemorrhage usually involve the closely contiguous eighth nerve.

5. *In the pons* the root fibers or nucleus may be affected, usually with the sixth nerve of the same, or the arm and leg of the opposite side. Degenerative affections, as tabes, bulbar paralysis, multiple sclerosis, progressive muscular atrophy or acute poliencephalitis rarely invade the seventh nucleus.

6. Rarer factors are hemorrhage into the Fallopian canal, disease of the parotid or lymphatic glands and leukemic infiltration.

Pathology.—Few autopsies have been made. The nerve has been found severed, its sheath and axis-cylinders destroyed, the nuclei in Schwann's sheath increased, the connective tissue increased and the muscles the seat of fatty change. The essential and usual change is *perineuritis*, exudation in the sheath compressing the nerve fibers, which undergo parenchymatous degeneration. In some cases degeneration only is seen; it is probable that slight swelling interferes with conduction. Facial neuritis differs from multiple neuritis in that alcoholism is a subordinate factor and the chief change is not in its peripheral twigs but in the trunk, usually within the Fallopian canal.

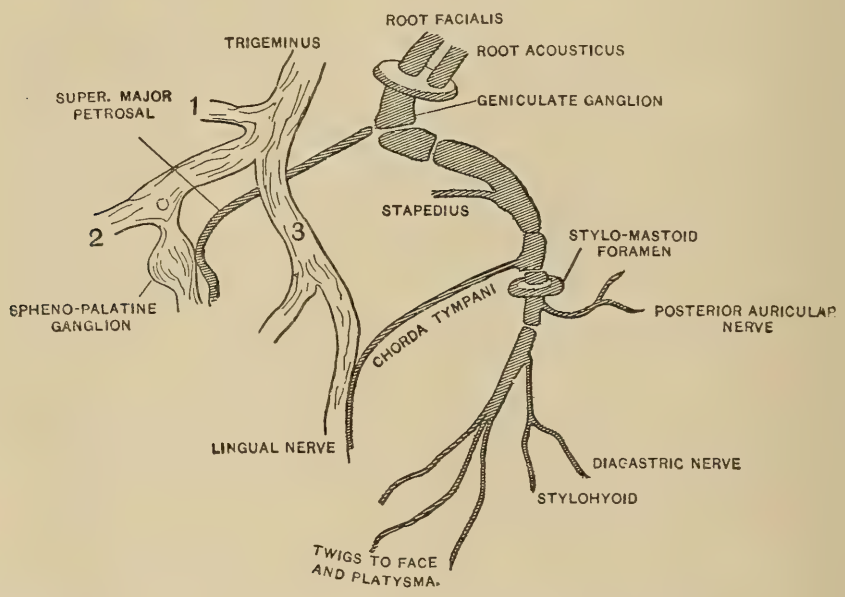


Fig. 76.—Relations of the fifth and seventh nerves and branches of the seventh.

Symptoms.—The affection sometimes begins with headache, pain, twitchings in the face, even fever or albuminuria, or without prodromes, when one *half of the face* is suddenly paralyzed and *completely immobile and toneless* within two days. The forehead is smooth, the lids cannot close (lagophthalmos), the tears run over the cheek and when a strong effort is made to close the eyes the eye on the affected side is turned upward by the inf. oblique muscle (Bell's sign). The orbicularis at times escapes involvement, since it receives some fibers from the third nerve. Smell is impaired because of absence of tears, the nasal aperture is smaller and cannot be dilated, sniffing on the affected side is impossible and the nasolabial furrow is lost. The mouth is lower from paralysis of the levator anguli oris, it cannot be closed and the saliva runs out, although its total secretion is often lessened. Labial enunciation is imperfect, puckering the lips, as for whistling, is impossible and expectoration is impaired. The loss of wrinkles and expression on the affected side is less marked in young subjects, especially about the eye, since years lessen the elasticity of the skin and the subcutaneous fat. Chewing is impaired by weak-

ness of the buccinator and food accumulates between the teeth and the cheek. The unused half of the tongue becomes furred. The tongue does not deviate on protrusion but may seem to, on account of asymmetry of the mouth. The tongue is sometimes slightly depressed from weakness of the stylohyoid and digastric muscles. The platysma paralysis is elicited by asking the patient to depress the lower lip. The palate is sometimes described as sagging, the speech as nasal, the uvula as deviating toward the sound side, but the uvula and palate are often asymmetrical in health and there is a tendency to regard the spinal accessory as the motor nerve for these structures.

Taste is lost in disease of the nerve between the geniculate ganglion and the offset of the chorda; it is lost in half the cases of "exposure" neuritis; it is not impaired in disease of the pons or in disease outside the skull. The chorda supplies the anterior two-thirds of the tongue and the glossopharyngeal the posterior third. Perverted taste (parageusia) results from irritation of Jacobson's nerve or the tympanic plexus. Deafness and tinnitus result mostly from coincident disease of the ear. When the stapedius is paralyzed the unopposed tensor tympani (fifth nerve) overacts and results in aural oversensitiveness, especially to low notes (hyperakusis or oxyokoia).

All facial reflexes are abolished, with wasting in thin subjects and the *reaction of degeneration* in a week or two in severe cases. In cases of moderate severity the excitability may be first increased and then gradually decreased in seven to ten days. Pain, even anesthesia in the ear and tenderness over the nerve trunk are sometimes observed, caused by involvement of the fifth nerve or possibly because the facial carries some sensory filaments. Sympathetic edema and sweating have been observed. Herpes indicates trifacial involvement. The saliva is decreased (the fibers for its secretion run with the chorda) and secretion of tears is lessened (the fibers from the facial nerve run to the fifth by the large petrosal nerve).

Diagnosis.—The diagnosis is made at a glance in fresh cases; in older cases secondary spasm on the paralyzed side may cause confusion. The etiological and topographical diagnosis is important.

1. *Supranuclear paralysis,* paralysis in the upper neurone, from the cortex to the nucleus, is differentiated as follows:

Supranuclear Palsy.	Peripheral Palsy.
Etiology; vascular disease, tumor, etc.	Neuritis, ear disease, etc.
Distribution of paralysis; partial, lower facial involved or only slight weakness of orbicularis palpebrarum.	*Complete;* orbicularis and occipitofrontalis involved. When eyes cannot be closed, probably peripheral or nuclear.
Reflexes persist and increased.	Lost. Paralysis flaccid.
Taste, auditory, salivary and sensory symptoms lacking.	Often present.
Pilocarpin increases salivary flow.	Does not increase it.
Voluntary motion lost, while emotional movement very often persists.	Both lost.
Degenerative reaction and wasting absent.	Present.
Cerebral symptoms present, general as headache, or focal as cortical epilepsy, alternating paralysis (see crus and pons localization).	Absent.

In lesions of the pons all parts of the facial nerve are affected, wherein it differs from facial nerve disease in the internal capsule. In nuclear disease the orbicularis oris is less involved or escapes affection, its centre being in the hypoglossus nucleus.

2. *Localization in the peripheral type* (see Fig. 76) may be (*a*) in the *pons* (*q. v.*). General disease of the posterior pons also affects the sixth nerve about whose nucleus the seventh nerve curves. (*b*) In localization *at the base,* taste is not involved and deafness results from coincident disease of the eighth nerve. (*c*) If in the *Fallopian canal* taste is impaired, the salivary flow decreased and hearing often abnormally acute. (*d*) If *external to the skull* there is no involvement of taste, salivary flow or the stapedius.

Double facial paralysis is rare and usually indicates disease within the skull, bulbar paralysis, disease of both sides of the pons or one focus in its centre, double otitis, especially syphilis, multiple neuritis, tetanus or in a form observed in childhood with ocular paralysis (*q. v.*). Organic facial paralysis may exist on one side and an hysterical paralysis on the other.

Prognosis.—Its duration varies from two to three months for moderate, and six to eight months for severe cases. Even in cases which seemingly recover and in those lasting over a month, some vestige of paralysis usually remains. The *prognosis* depends (1) on the cause; it is favorable in syphilis, birth trauma and polyneuritis; in ear disease it is better if the drum has not ruptured; it is unfavorable in tumor or nuclear disease; (2) on the electrical reaction. If after ten days the irritability is not reduced recovery may be expected in about three weeks; if after fourteen days it is lowered but not lost, recovery occurs within two months; if after fourteen days the irritability is lost, the prognosis is doubtful and the course is likely to cover months. Recurrence is infrequent, although five attacks are recorded. *Contracture* (secondary overaction) usually occurs in four to six months and progresses for eight to twelve months, when it lessens and disappears in mild but remains in severe cases. In old subjects it balances up the facial contour; in young subjects it produces deformity. It depends on functional alteration of the nucleus.

Treatment.—(*a*) The *cause* should be reached, if possible; antisyphilitic remedies and ear-drainage have cured cases even of twenty-three years' duration. (*b*) *Antirheumatic drugs* do not shorten the course. A mercurial purge, hot fomentations, which must be carefully maintained for two or three days, keeping the patient indoors, and a blister over the mastoid and one over the side of the neck are beneficial. (*c*) *Correction of deformity:* The eye should be douched with boric acid, bandaged closed during sleep and the lids massaged, the cheek and mouth should be massaged upward and a hook in the angle of the mouth (sustained by tape over the ear or by adhesive plaster) prevents deformity to some extent. (*d*) *Electricity* should not be used for two or three weeks, and then the galvanic current should be given, for fifteen minutes each day, in strength just sufficient to produce muscular contraction; the application of the anode over the muscles is the least painful method. The faradic current causes pain. (*e*) For *contracture* little can be done

excepting massage and steaming the face. (*f*) In irreparable cases, surgical transplantation of the facial trunk into the spinal accessory or hypoglossus has been successfully performed.

Facial or Mimetic Spasm (Tic Convulsif).—Etiology.—The causes are (*a*) organic disease in the cortex, pons or nerve trunk, which irritates (perhaps later paralyzes) the centre, nucleus or paths. Irritation of the fifth nerve is considered causal. (*b*) There is an idiopathic form, which occurs in persons between the fortieth and sixtieth years, most often in women with neuropathic or emotional tendencies (see HABIT SPASM under CHOREA). (*c*) There is also a reflex form which results from worms or uterine disease.

Symptoms.—The spasm begins paroxysmally on one side and is more often clonic than tonic. The *clonic form* affects the orbicularis palpebrarum (blepharospasm) and zygomatici most frequently, although the frontalis, chin depressors or all the facial muscles may be concerned in severe cases. Pain and paresis are absent save in progressive organic disease. The spasm is lessened by rest and increased by cold or emotion. It may spread to the opposite side or exceptionally to the muscles of mastication, tongue, pharynx, larynx, neck or arm. The *tonic form* is seen in photophobic disease of the eye, paralysis agitans, tetanus, tetany or hysteria.

Prognosis.—The prognosis is unfavorable after the first few months and the disease is then likely to last for years or for life.

Treatment.—Treatment varies with the cause. Trigeminal irritation should be removed if possible. Arsenic is beneficial in some cases, but nervines and antispasmodics are useless. Some cases are cured by galvanization, if tender points are found, freezing of the cheek with ethyl chloride, stretching of the facial nerve usually with but temporary relief and alcohol injections.

THE EIGHTH OR AUDITORY NERVE.

The eighth nerve in the old nomenclature was the portio mollis of the seventh pair, the facial nerve being the portio dura; as the names imply, the auditory is softer than the facial nerve and therefore less resistant to pressure. It is purely sensory and consists of two distinct roots, the cochlear and vestibular. At the point where the roots separate is found a group of cells, analogous to the spinal ganglia, from which arises Wrisberg's nerve, an accessory structure with vasomotor and salivary secreting fibers.

The *cochlear nerve* is the true auditory nerve and its nuclei are found in the floor of the fourth ventricle. It is connected through the tegmentum of the crus and internal capsule with the contralateral and to a less degree the homolateral temporosphenoidal lobe. It is distributed to the cochlea and ganglionic cells of the organ of Corti. (*a*) *Cortical disease* (see BRAIN LOCALIZATION) may in rare cases cause word-deafness, as may (*b*) *disease of the auditory tracts* by lesions of the lateral lemniscus, the post. corp. quadrigemina, internal capsule or pons. Nuclear disease is almost unknown. Neuromata are rare, but occur on the auditory

trunk oftener than on any other cranial nerve. The nerve trunk may degenerate in tabes. (*c*) *Lesions at the base* may follow trauma, meningitis, hemorrhage or syphilis. (*d*) Most auditory nerve disease results from *disease of the internal ear,* either primary or secondary to disease of the middle ear (inflammation, syphilis, degeneration or hemorrhage in the labyrinth). There are three prominent *symptoms:* (*a*) *Nervous deafness may result.* Deaf-mutism is congenital in 80 per cent. and acquired in early life in 20 per cent. of cases. Deafness from occlusion of the meatus or from disease of the middle ear is excluded by Rinne's test; a vibrating tuning-fork or watch, if held over the meatus, is not heard in these affections, but is heard when its base is placed on the mastoid which conducts vibrations to the internal ear. In disease of the internal ear, bone-conduction is lacking. Differentiation between deafness due to disease of the internal ear and that due to bone disease is only made by means of the associated symptoms. The *treatment* of nervous deafness is etiological. Iodides are sometimes beneficial, as are full doses of pilocarpine. Electricity is useless. (*b*) *Auditory hyperesthesia* may result from central or peripheral disease. In genuine hyperesthesia (hyperacusis) sounds are heard by the patient which other individuals may not hear, as in hysteria, epilepsy, migraine, meningitis, acute mania and at times after use of caffeine, alcohol or opium. Spurious hyperacusis results from paralysis of the stapedius. In dysacusis ordinary sounds cause disproportionate sensations or discomfort; this is common in headache and cerebral disorders. (*c*) *Tinnitus aurium* designates subjective distressing and intractable sensations, as ringing, roaring or whistling in the ears. In 80 per cent. of cases it is caused by disease of the internal ear. It may also occur in any disease of the middle ear, when there is cerumen or foreign bodies in the external meatus, or occlusion of the Eustachian tube, when the individual is subject to long exposure to loud sounds, as in boiler factories, and in the neuroses (hysteria, neurasthenia, migraine, epilepsy), aneurysm, anemia, gout or cinchonism. Sounds of centric origin are usually continuous and sometimes elaborate. Wax or bilateral labyrinthine disease may also cause continuous tinnitus, while in anemia or aneurysm, these sensations are intermittent. Hearing may be hyperacute, deranged or lost. Spasm of the palate or musculature of the Eustachian tube may produce a most annoying snapping sound, at times audible to bystanders. *Treatment* depends on the cause. Bromides are most useful, combined with a few drops of the tr. belladonna or cannabis indica. Quinine and salicylates seldom give relief. Full doses of nitroglycerin relieve certain cases and counter-irritation over the mastoid sometimes mitigates tinnitus.

The vestibular nerve arises from the medulla, supplies the vestibule and semicircular canals, subserves sensation of space and equilibration and has cerebral and cerebellar connections.

Ménière's Disease, Auditory Vertigo (Vertigo ab aure læsa).—Paul Ménière, in 1861, described a syndrome consisting of vertigo, tinnitus, vomiting and sometimes deafness, developing with apoplectiform suddenness and due to acute labyrinthine disease. The lesion is usually a slightly irritative disease of the vestibular nerve, which supplies the organs of

equilibration, the semicircular canals. According to Gowers, 80 per cent. occur in persons between the thirtieth and sixtieth years, 66 per cent. in males, and 90 per cent. of all cases of vertigo not due to epilepsy or organic brain disease are caused by lesions of the labyrinth. Fränkl-Hochwart classifies Ménière's complex as follows: (*a*) *Apoplectiform cases,* which occur in healthy ears, of which few more than 30 cases are recorded, due to labyrinthine hemorrhage or to trauma; leukemia and arteriosclerosis are promoting factors and it is thought that acute angioneurotic transudation of lymph may occur in the canals. (*b*) Cases developing in *ears already acutely or chronically diseased;* the internal ear is the usual seat of disease, but the external or middle ear may be affected or the branches of the eighth nerve, perhaps by tumors, syphilis, tabes, paretic dementia, gout, cold, facial paralysis or quinine and salicylates. (*c*) *External influences,* are pressure on the drum by violent douching of the ear; (*d*) *pseudo-Ménière's disease,* which occurs in epilepsy, hysteria, neurasthenia, migraine or sea-sickness.

Symptoms.—The symptoms begin gradually or suddenly, with occasionally momentary loss of consciousness; the paroxysms may be separated by weeks or months or perhaps occur daily. (*a*) The *vertigo* is subjective or objective, perhaps combined; the patient cannot stand; he is likely to fall backward or if standing is possible is ataxic. These symptoms indicate lesion in the *vestibular nerve;* slight vertigo may persist between paroxysms. (*b*) *Nausea and vomiting* are usual, save in the lighter forms and are often attended by headache, sometimes collapse, pallor and clammy skin and rarely by death. (*c*) The *tinnitus* of a roaring throbbing character and (*d*) *nervous deafness,* which is usually not absolute, indicate disease of the *cochlear nerve.* Nystagmus, double vision and ataxia are occasional symptoms and result from the relations between the labyrinth, cerebellum and cerebrum. Acute or progressive disease is unfavorable. The vertigo may cease when complete destruction of the nerve results in deafness. Some cases recover.

Diagnosis.—Aural vertigo constitutes 90 per cent. of vertigo. Differentiation is as follows: (*a*) *Gastric disease* may cause vomiting and *vertigo* (*a stomacho læso*) but does not produce tinnitus or deafness. Gastric vertigo was once thought to cause 80 per cent. of cases of vertigo; now, but 5 per cent. are attributed to it (Gowers). Many of these cases are neurotic. (*b*) *Epilepsy,* especially *petit mal,* may produce vertigo, very rarely tinnitus or deafness and much oftener loss of consciousness. (*c*) *Cardiac disease,* as aortic regurgitation or arteriosclerosis, may cause vertigo but not the other symptoms. (*d*) *Organic brain affections,* as tumor, have other distinctive signs, as headache or focal lesions. Confusion is possible with arteriosclerotic vertigo, and especially with coincident accidental deafness. (*e*) *Ocular vertigo* ceases on closing the eyes. Gerlier (1887) described a form endemic in France, Switzerland and Japan, and characterized by vertigo, great depression, paretic weakness of the neck and limbs, ptosis, strabismus, diplopia and retinal hyperemia, hemorrhage or edema. Recovery occurs in one to four months.

Treatment.—Labyrinthine disease is little influenced by drugs, except when syphilis and gout are its causes. Quinine, salicylates and nitro-

glycerin should be given (see TINNITUS). *Bromides* are most useful in daily doses of one dram. Galvanization with the anode over the ear and cathode to the neck seemingly has helped some cases, as have blisters over the mastoid. Disease of the pharynx, Eustachian tube and accumulation of wax must be treated. Removal of the malleus and incus with mobilization of the stapes relieves a few cases.

NINTH OR GLOSSOPHARYNGEAL NERVE.

This nerve is almost inseparably connected in its origin, course and functions with the vagus and the internal part of the spinal accessory nerve. Compared with disease of the first eight cranial nerves, affections of these visceral nerves are infrequent. Of all the cranial nerves, the ninth is least understood and is never alone diseased. The causes of its disease are the same as those of the vagus (*v. i.*).

It is distributed as follows: (*a*) its sensory fibers supply the back of the tongue, pharynx, soft palate, tonsil, upper pharynx, Eustachian tube and tympanic cavity. It is probably the nerve of nausea. (*b*) Its motor fibers supply the upper pharynx and possibly the palate. (*c*) Taste fibers are usually thought to be distributed to the posterior third of the tongue and palate, but they are not found in the nerve root and are considered to pass through the fifth nerve to the brain. Increased flow of saliva and loss of taste on the root of the tongue may result from disease of the middle ear due to relations between the nerve of Jacobson (ninth) and the branches of the fifth nerve. The nerve is thought to regulate contractions of the pharynx, which become frequent (spasms) in disease or on section of the nerve.

TENTH OR VAGUS NERVE.

Arising in the medulla, it has with the internal part of the spinal accessory nerve an enormously diffuse and important visceral distribution. It is a mixed nerve, the motor fibers originating from the nuclei and the sensory fibers from the jugular and plexiform ganglia; the vagus carries the sensory and the accessorius, motor fibers.

Etiology.—Affections of the vagus result from: (*a*) Nuclear disease, as degenerative bulbar palsy or acute vascular lesions, which usually also involve other nuclei; (*b*) involvement of the root and trunk by meningitis, syphilis, caries, trauma, aneurysm of the arch or its branches, carcinoma, esophageal growths, tuberculous or other adenopathies, pleural (right-sided) or pericardial effusions or adhesions, mediastinitis, goitres, operations on the neck or rarely neuroma and neuritis from diphtheria or other infections, tabes or plumbism. Cortical, especially functional, causes may occasion symptoms.

Pharyngeal Branches.—The pharyngeal plexus consists of branches from the ninth and tenth nerves. In *paralysis*, sensation and the pharyngeal reflex are lost; bilateral disease causes dysphagia. The stomach-tube can be introduced without sensation or spasm. Pulpy foods are better swallowed than dry foods or fluids. Food is likely to enter the

larynx and when the palate is involved there is regurgitation into the nose and nasal speech results. The palate is probably supplied by the accessory nerve. *Spasm* is always functional, transient or recurrent,

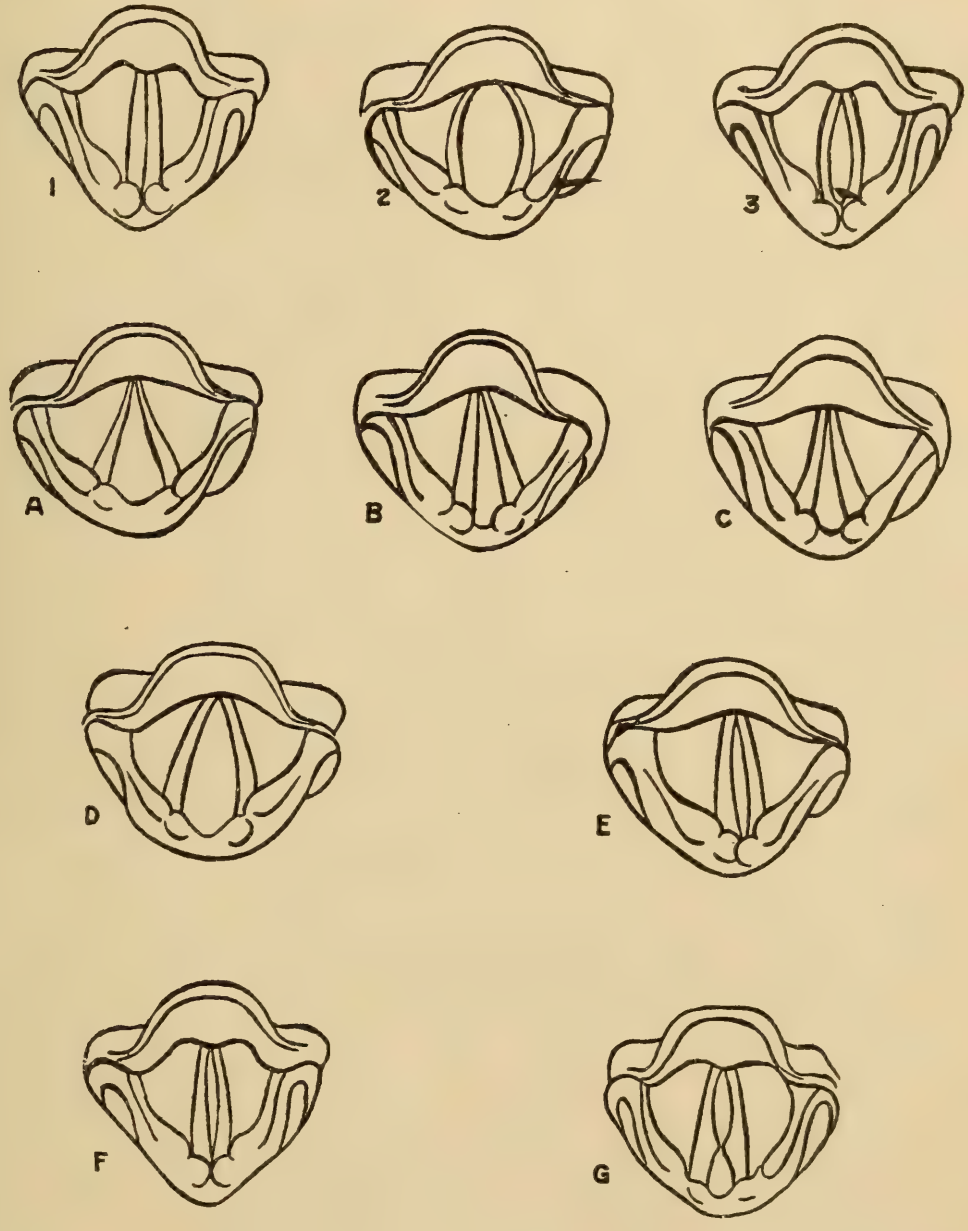


FIG. 77.—The laryngeal paralyses: *1*, normal phonation; *2*, normal deep inspiration; *3*, normal cadaveric position; *A*, bilateral adductor palsy; attempted phonation; *B*, left adductor palsy; attempted phonation; *C*, bilateral abductor paralysis; deep inspiration; *D*, left abductor paralysis; deep inspiration; affected cord in cadaveric position; *E*, left abductor paralysis; phonation; affected cord in cadaveric position; right cord crossing median line; *F*, bilateral thyro-arytenoid paralysis; *G*, bilateral thyro-arytenoid paralysis and paralysis of arytenoideus, giving an hour-glass opening. (From Church's Nervous Diseases.)

does not allow of introduction of the stomach-tube and includes the globus hystericus, esophagismus, pharyngismus, false and genuine hydrophobia and the pharyngeal crises of tabes. The esophagus is rarely affected except in diseases of the centres or trunk.

Laryngeal Branches.—The *superior laryngeal* is (*a*) the sensory nerve for the larynx above the vocal cords, (*b*) the motor nerve for but one larynx muscle, the cricothyroid, which modulates the voice by regulating tension in the cords, and (*c*) the motor nerve for the depressors of the epiglottis, the thyro-epiglottic and aryepiglottic muscles, paralysis of which causes the epiglottis to stand upright, thereby risking aspiration of food and pneumonia.

The *recurrent laryngeal* curves around the aorta on the left and the subclavian artery on the right side; (*a*) it is the sensory nerve to the larynx below the cords and the trachea and (*b*) is the motor nerve to every intrinsic larynx muscle except the cricothyroid.

Treatment.—Paralysis is usually peripheral (*v. i.* table). In nuclear types there is no therapy. Suggestion is valuable in hysterical forms. The faradic current may be used with the positive pole to the forehead and the negative to the larynx externally or internally. Other measures include laryngeal gymnastics, in which the patient repeatedly attempts to speak, intralaryngeal insufflations of alum, the administration of strychnine hypodermically and Ollivier's procedure, in which the thyroid cartilage is compressed between the index-finger and thumb during inspiration and expiration, while the subject attempts to phonate. Tracheotomy and nasal feeding are sometimes indicated.

Anesthesia and Hyperesthesia of the Larynx.—*Anesthesia* of the larynx is rare, but may occur uni- or bilaterally in disease of the sup. laryngeal nerve, the vagus roots, its nucleus or in the hemianesthesia of hysteria or organic brain disease. In nuclear disease it is partial; in affections of the nucleus, root and trunk the reflexes are lost, but they are present in supranuclear and functional affections. *Hyperesthesia* is rarer, being observed in neurotics as a tickling, tendency to cough or spasm; neuralgia and crises also occur.

Laryngeal Spasm.—The most common form of laryngeal spasm is spasm of the adductors, which are concerned in reflexes inimical to entrance of foreign bodies into the larynx; it may result from centric, local (laryngitis with croup-like cough at night) or reflex irritation from distant or neighboring foci, as elongated uvula, goitre or hypertrophy of the pharyngeal tonsil. It includes *laryngismus stridulus*, which occurs in children under three years of age and is due to rickets in 66 per cent. of cases, tetany, and reflexly to alimentary disorders; it comes on at night or on awakening in the morning, with croup-like cough, and presents all the symptoms of acute suffocation, as stridor, pallor, cyanosis, wide pupils, protruding eyes, low diaphragm, involuntary evacuations, centric vasomotor and cardiac disturbances and convulsions either generalized or of the infantile carpopedal type. The attacks last from a few seconds to one-half or one minute and are sometimes attributed to loss of temper when they follow a reproof. In the adult, laryngeal spasm occurs in tabes, hysteria, tetany, hydrophobia, epilepsy (the initial cry), asthma or migraine. The attacks are recognized by the negative laryngoscopic findings, paroxysmal occurrence and the stridor being manifest in both inspiration and expiration. Partial spasm occurs in some types of stuttering, clergymen's sore throat and explosive, unmodulated speech.

TABLE FOR DIFFERENTIATION OF LARYNGEAL PARALYSIS (Modified from Gowers and Church).

FORM OF PARALYSIS.	NERVE AND MUSCLES AFFECTED: FUNCTION.	CAUSES.	SYMPTOMS.	SIGNS BY LARYNGOSCOPY.
Total bilateral palsy.	Recurrent nerves; all muscles involved or all save cricothyroids.	Nuclear disease, disease of trunk or of recurrent nerve.	Voice lost; cough impossible; stridor only on deep inspiration.	Both cords moderately abducted, motionless in cadaveric position, neither receding on deep inspiration nor approximating in phonation. Fig. 3 (Fig. 77).
Total unilateral palsy.	Recurrent nerve; ditto.	Ditto. (In general, paralysis of one cord always suggests pressure on the recurrent.)	Voice low-pitched and hoarse; cough impossible; stridor absent or only on deep inspiration.	Paralyzed cord as above; sound cord moving freely, even beyond median line.
Bilateral abductor palsy.	Recurrent nerves; both posterior crico-arytenoids, abductors.	Central disease, toxemia, pressure or disease of trunk or recurrent nerves. (In general, marked bilateral cord paralysis always suggests centric disease.)	Voice little changed; cough possible; extreme inspiratory stridor, long inspiration, marked laryngeal excursion, cyanosis, even death; expiration free, whereby differentiate from bronchial stenosis even without laryngoscopy.	Glottis is chink-like, cords near together, not separated on inspiration but even drawn closer by unopposed adductors. Fig. C.
Unilateral abductor palsy.	Recurrent; one posterior crico-arytenoid.	Same; especially aneurysm, injury, etc., to one recurrent laryngeal nerve.	Symptoms inconclusive, little affection of voice or power to cough.	The same, but unilateral. Fig. D and E.
Bilateral adductor palsy.	Recurrent; both lateral crico-arytenoids and arytenoids; adductor muscles.	Hysteria (women under thirty years), overuse of voice, catarrh of larynx, anemia.	Voice lost, but coughing, laughing, even singing are possible, hence called phonetic paralysis by Türck; no stridor or dyspnea; often with anesthesia of larynx and pharynx (hysteria), etc., hysterical aphonia.	Cords normal in position but not brought together on phonation; normal movement on breathing. Fig. A.
Unilateral adductor palsy.	Recurrent; one lateral crico-arytenoid and arytenoid.	Toxemia, lead, diphtheria, cerebral disease, syphilis, tuberculosis.	Voice hoarse; coughing or laughing imperfect; sometimes dysphagia.	Ditto; but unilateral. Fig. B.
Paralysis of cords proper.	Recurrent; thyro-arytenoids; tensors and adductors of cords.	Very frequently involved from overuse of voice; hysteria; catarrh.	Loss falsetto notes; uncertainty of phonation; often with adductor or arytenoid paresis.	Cords atonic and concave. Fig. F. When thyro-arytenoids and arytenoids are involved, the space between cords is hour-glass in form.
Bilateral paralysis of tensors.	Superior laryngeal nerve; crico-thyroids, the tensors and modulators of cords.	Cold, straining of voice, diphtheria.	Hoarseness; high notes imperfectly taken; voice weak.	Cord higher on paralyzed side, but hard to see with mirror.

Nervous cough is present in the explosive hysterical type, in the metallic ovarian form in young girls and in the barking variety in young masturbators.

TREATMENT.—Treatment of spasm includes removal of local irritation, as affections of the ear (which has branches from the vagus) or pharynx, hygiene, tonics, as cod-liver oil in children, and such immediate measures as thrusting a finger into the throat, dashing water in the face, hydrotherapy and for the attack chloral and bromides by enema, local application of cocaine, inhalation of chloroform and in adults nitrite of amyl and morphine.

Pulmonary Branches.—The vagus and the sympathetic form the pulmonary plexus. Afferent fibers convey sensory impressions to the respiratory centre; these include accelerating fibers which predominate, and inhibitory fibers, stimulation of which (sup. laryngeal nerve) arrests breathing. Afferent impulses are also carried to the vasomotor centre. Efferent motor impulses are conveyed to the bronchi. Division of the vagus causes rapid congestion of the lungs with hemorrhage (also observed in acute lesion of the pons) and slower but deeper breathing; stimulation of the divided end of the nerve accelerates breathing even to its tetanic arrest. Bronchopneumonic foci are not trophic but aspiration pneumonia from paralysis of the palate and larynx. The pulmonary twigs are probably concerned in Cheyne-Stokes's breathing, bronchial asthma and hiccough.

Cardiac Branches.—The vagus and sympathetic nerves constitute the cardiac plexus. (*a*) Motor fibers from the spinal accessory nerve inhibit and coördinate cardiac action, which is sometimes under the control of the will; the heart may at times be slowed by pressure on the neck; marked irritation of the vagus slows or even arrests the heart. Bilateral division may cause sudden death. Paralysis of these fibers allows the sympathetic accelerator fibers to dominate, producing arrhythmia and tachycardia. Digitalis is ineffective in vagus paralysis. (*b*) The sensory fibers are concerned in the cardiac neuroses (*q. v.*). (*c*) These fibers are probably trophic, as Eichhorst's experiments show fatty myocardial degeneration on division of the vagi.

Gastric Branches.—(*a*) Motor branches supply part of the stomach; they cause nausea and are concerned in reflex, direct or centric vomiting, as in brain lesions, wherein the vomiting usually occurs without nausea. (*b*) Sensory branches; the vagus is the nerve of hunger and thirst, and its disease often causes anorexia and loss of thirst. Centric disease causes the gastric crises of tabes and cortical functional disease produces the epigastric aura of epilepsy and perhaps gastralgia in the neuroses. Vertigo probably causes vomiting through the vagus. Intestinal and splenic symptoms are lacking.

ELEVENTH OR SPINAL ACCESSORY NERVE (EXTERNAL PORTION).

The internal, minor part runs with the vagus, while the major, external segment is a spinal motor nerve, arising from the lateral portion and

ant. horns of the cord between the sixth cervical segment and the medulla. It courses upward through the foramen magnum into the cranium. It is the chief supply of the sternomastoid muscle and the upper part of the trapezius, the lower two parts of which are usually supplied by the cervical and upper dorsal nerves.

Paralysis.—Paralysis is rarely caused by cortical disease, because the muscles have bilateral representation. The usual causes are bulbar paralysis, lesions near the foramen magnum, meningitis (usually with involvement of the inner accessory branches and the twelfth nerve), syphilis, trauma, tabes, syringomyelia, progressive muscular atrophy, caries or tumor of the spine, or neuritis. The symptoms are paralysis of (*a*) the sternomastoid, in which there is impaired rotation of the face to the side opposite the lesion; in old cases contracture turns the face toward the paralyzed side. (*b*) That part of the trapezius from the occiput to the acromion is paralyzed, showing a concave instead of a straight line, best seen in deep inspiration or shrugging the shoulders. (Paralysis of the second portion causes imperfect elevation of the arm, some drooping and winging out of the scapula, the lower part of which is drawn toward the spine by the rhomboids and lev. anguli scapulæ; paralysis of the third portion results in weakness in the adduction of the scapula to the spine.) If it is bilateral, the head falls forward or backward as the sternomastoids or trapezii, respectively, are affected. In peripheral disease there are wasting and the reaction of degeneration.

Accessory Spasm, Spasmodic Torticollis or Wryneck.—Etiology.—It occurs in women in 66 per cent. of cases (Gowers). A neurotic tendency, trauma, overexertion, infections, alcoholism, plumbism, cervical caries, brain tumor and diseases of the eye and ear (torticollis ab oculo læso, ab aure læsa) are among its uncertain causes.

Symptoms.—The symptoms vary as the affection is clonic or tonic, unilateral or bilateral, and with the number of muscles involved. One sternomastoid is generally involved, especially in tonic spasm, in which the occiput is turned to the diseased side, the face to the opposite side and the ear nearer the clavicle. The upper part of the trapezius comes next in frequency of involvement and is affected equally by clonic and tonic spasm. The affected shoulder is raised and slightly rotated, the scapula drawn toward the spine and the head backward. The spine is often convex toward the diseased side. The splenius is half as frequently affected, the head being drawn backward, the face a little to the affected side and upward. Less often the scalenus, platysma, rectus and obliquus capitis are affected. The muscles in tonic spasm are hard, prominent and sometimes hypertrophied. Clonic spasm disappears during sleep, is increased by fatigue and may also invade the face, neck, eye-balls, vocal cords or the arm. There may be pain, fatigue and rarely tender spots from compression of the brachial plexus by the scaleni. Bilateral clonic or tonic spasm of the trapezius draws the head backward (retrocollic spasm) and always involves the frontalis muscle.

Course and Prognosis.—The onset is gradual, the disease is usually stationary or progressive in its course, rarely recurrent; recovery is infrequent. The outlook is generally grave in well-developed types.

Diagnosis.—(*a*) In *congenital or fixed wryneck* there is no active spasm. One sternomastoid (but rarely the trapezius) shows atrophy, induration and cicatrization, due to traction on the neck during delivery or perhaps from a central lesion; 70 per cent. occur in left occipito-anterior presentations. The disease escapes notice for years because of the shortness of the child's neck. Wilks always found facial asymmetry. Tenotomy cures it. (*b*) *Hysterical torticollis* occurs in subjects under thirty years of age; other stigmata usually develop. Bompaire and Brissaud describe a *mental torticollis,* clearly a psychical phenomenon. (*c*) *In children* clonic spasms, especially in the deep muscles, lead to nodding, salaam movements, spasmus nutans, nictitatio spastica. The affection may develop from reflex dental or digestive disorders or from organic cerebral disease. It is sometimes habit spasm, hysteria, epilepsy or idiocy.

Treatment.—Treatment is etiological. Nervines, sedatives and electricity seldom produce permanent results. Morphine is a dangerous agent, although its local hypodermic use has led to recovery. Massage and deep compression are occasionally helpful. In chronic cases resection of the accessory nerve is indicated. If many muscles are involved the third and fourth cervical nerves are also resected.

THE TWELFTH NERVE.

The hypoglossal is purely motor. *Paralysis of the tongue is* (*a*) *rarely cortical;* supranuclear glossoplegia from lesions between its centre and the frontal convolutions, almost always occurs with hemiplegia and paralysis of the lower facial fibers; there is no wasting or reaction of degeneration. The root of the tongue is higher (hyoglossus paralysis) and on protrusion the tongue deviates to the paralyzed side, being pushed over by the sound genioglossus. (*b*) *Nuclear paralysis* occurs in bulbar paralysis, sometimes in tabes or paretic dementia, and much less frequently in acute inflammation or softening in the medulla. Nuclear lesions are bilateral since the nuclei are closely contiguous, and cause the reaction of degeneration and wasting, the mucosa being wrinkled over the atrophic half. Fibrillation is frequent, as in paretic dementia, from cortical and nuclear changes. In bilateral glossoplegia the tongue cannot be protruded, there is impairment of the first act of swallowing, in chewing and articulation, all of which are but slightly marked in unilateral paralysis. (*c*) *Infranuclear paralysis* results from lesions near the surface of the medulla, which may produce unilateral paralysis of the tongue, palate and vocal cord. Contralateral hemiplegia may occur. Syphilis, trauma or tumors are causal; there are recorded only 40 cases of peripheral, isolated hypoglossal paralysis (Panski, 1903). Wasting, the degenerative reaction and fibrillation are present. *Treatment* is that of other paralyses.

Spasm.—The tonic or clonic form is rare. It occurs in chorea, facial spasm, certain types of stuttering, epilepsy (in which the tongue is bitten), hysteria (in which the tongue is rarely bitten), psychoses, meningitis, neurotic spasm in speakers (aphthongia) or musicians. The prognosis in general is favorable.

DISEASES OF THE SPINAL NERVES.

The spinal nerves differ from the cerebral in that they contain sensory, motor and vasomotor fibers. Disease or injury therefore suspends motility and sensation, causes vasomotor paralysis, trophic changes, the reaction of degeneration, atrophy and contracture.

THE PHRENIC NERVE.

Paralysis.—(*a*) Disease or trauma of the vertebræ, cord, membranes or of the third, fourth and fifth cervical nerves of origin may cause it. In disease of the cord other paralysis is always present. (*b*) Its trunk may be injured in its course or damaged by aneurysm, tumors or pleurisy. Neuritis sometimes occurs in multiple neuritis or tabes, etc.

Symptoms.—The diaphragm is paralyzed, usually on both sides. The abdomen retracts on inspiration and bulges on expiration, the converse of its normal movements. Litten's sign is absent and there is no inspiratory descent of the liver or spleen. Blowing, coughing and pressing at stool are impossible. Costal breathing is exaggerated, the accessory muscles are strongly in play, dyspnea and cyanosis are present, especially on exertion, and coincident bronchitis may cause most alarming symptoms from stagnation of mucus. The nerve may be tender in the neck.

Diagnosis and Treatment.—Costal, hysterical breathing may at first cause confusion, as may inflammation from diaphragmatic pleurisy. The *outlook* is grave. It is more favorable in syphilis, lead or diphtheritic neuritis than in the alcoholic form. *Treatment* is that of the cause but is usually futile. Faradic, phrenic stimulation is sometimes beneficial, as in apparently asphyxiated newborn infants.

Phrenic Spasm.—1. In *clonic spasm,* singultus or hiccough, the diaphragm spasmodically contracts with a sound due to sudden closure of the glottis, sometimes 50 to 100 times a minute. Probably the vagus and respiratory centres are involved, for dyspnea, dysphagia or dysarthria may occur. Clonic spasm occurs (*a*) in inflammation, either thoracic (pleurisy, pericarditis) or abdominal (peritonitis, appendicitis, obstruction); (*b*) in irritation, esophageal, mediastinal, hepatic or gastric, directly or reflexly; (*c*) in nervous affections, as centric disease or the neuroses; and (*d*) in toxemia, uremia, etc. *Treatment* is that of the cause. Gastric lavage; apomorphine; circular constriction of the lower chest with the head bent forward; attempts to sneeze, cough, breathe deeply or press down with the glottis closed; faradization, blistering the neck and epigastrium; asafetida, chloral or bromides; and narcotics and inhalations of chloroform or amyl nitrite may be beneficial.

2. *Tonic phrenic spasm* is rarer. The causes are tetany, epilepsy, tetanus and hysteria. The diagnosis is easy. The lower chest and upper abdomen are distended and often sensitive; the upper chest moves rapidly and forcibly, with the accessory respiratory muscles in full play; there is no respiratory excursion, the lungs are acutely distended, the

abdominal viscera are luxated downward, there is dyspnea and cyanosis, and death may result. *Treatment* consists of the application of heat, blisters and the faradic current to the lower chest and chloroform inhalations and morphine internally. Analogous spasm may occur in the respiratory muscles, as yawning, sneezing, coughing, laughing and weeping spasms, possibly from vagus disease or from the neuroses, psychoses, multiple sclerosis, bulbar paralysis or disease of the ear, nose or stomach.

THE POSTERIOR THORACIC NERVE.

This nerve is often affected in its long course, from trauma, muscular strains in overhead work (the nerve originates in the scalenus medius) and from carrying loads on the shoulder—whence its occurrence in men in 90 per cent. of cases; it is also involved in muscular atrophies and neuritis. The results are impaired fixation of the scapula, movement of the arm and inspiration. The scapula wings out when the arm is moved forward and stands less obliquely than normal, its outer part moving forward, while the lower angle is drawn in by the rhomboids and trapezius. The sole sensory change is pain in the neck and shoulder. The course is tedious and rest, especially of the scaleni, is the prime indication.

SUPRASCAPULAR NERVE.

It originates in the fourth, fifth and sixth cervical nerves and is affected in disease of the plexus or alone in trauma, shoulder dislocations or falls on the hands. The supraspinatus and infraspinatus are paralyzed, giving some weakness in fixation of the humerus, as uncertainty in writing, turning up and in of the lower scapular angle, some anesthesia over the scapula, frequently shoulder-girdle pain and often involving the **circumflex nerve**, which arises from the fifth, sixth and seventh cervical nerves of the posterior cord of the brachial plexus. Deltoid paralysis (dislocation, crutch paralysis, plexus disease) produces inability to deviate laterally the arm, changes in the shoulder, which becomes angular, sometimes adhesions in the joint, probably trophic from its articular branch and anesthesia over the lower part of the deltoid. It may be confused with primary joint disease, in which, however, the scapula follows the arm movements.

THE MUSCULOSPIRAL NERVE.

Arising from all the roots in the plexus except the first dorsal and curving around the humerus, it is the most exposed and most frequently injured nerve in the arm or body; injuries result from fracture, callus, blows, muscular exertion, carrying heavy weights, tying the arm to the side or behind the back, sleeping with the arm over the back of a chair, especially in alcoholics, lifting the arms during surgical anesthesia and from a tight Esmarch's constrictor; less often injury results from cold or lead palsy. *Musculospiral (radial) paralysis* involves (*a*) the extensors of the upper extremity; *i. e.*, the *triceps*, so that the elbow cannot be extended, and the supinators; the triceps sometimes escapes affection;

the supinator longus is not involved in lead palsy or traumatism below the lower third of the humerus and in centric disease it escapes because its centre is with the flexors. The *extensors of the wrist* are involved, producing marked atrophy of the back of the forearm and the characteristic wrist-drop; the *extensors of the thumb and fingers* are affected; they become semiflexed and can only be extended by the interossei (ulnar nerve) after the first phalanges are passively extended. Changes in the synovial sac or bones of the dorsal wrist produce the painless carpal tumor of Gubler, from involvement of branches to the wrist or the mere mechanical action of flexion (see LEAD INTOXICATION). (*b*) *Sensory disturbance,* slight compared with the palsy, may rarely cause anesthesia over the deltoid and back of the forearm or more often over the radial side of the dorsum of the hand, thumb, index and half of the middle finger. Most cases recover with rest and immobilization. Flexor contracture can be avoided by splints.

THE MEDIAN NERVE.

The median nerve originates from the outer and inner cords of the plexus and contains fibers from all its cervical roots. Isolated paralysis is not common. It results from trauma, fractures, stabs, carrying weights in the bend of the elbow and sleeping on the arm. (*a*) *Motor symptoms* are conspicuous. Both pronators are involved, besides all flexors of the forearm, wrist and fingers except the flexor carpi ulnaris and the ulnar part of the deep flexor of the third and fourth fingers. In the hand, the palmaris longus, the abductor, opponens and short flexor of the thumb (resulting in flattening of the thenar prominence and the "ape-hand" from the index-finger and thumb lying parallel) and the two radial lumbricales are affected. The second phalanges cannot be flexed on the first (save in the two ulnar fingers); flexion of the first phalanges by the interossei is possible; metacarpophalangeal subluxation may result from the unopposed extensor action of the interossei on the second and third phalanges. The paralyzed muscles may waste and trophic manifestations are common. (*b*) *Sensory symptoms* are often slight. Anesthesia, if present, is greatest in the palm, front of the thumb, first two fingers and half of the third.

THE ULNAR NERVE.

Its origin is from the last cervical and first dorsal nerves; its superficial course at the inner condyle, forearm and wrist, exposes it to injury, as by dislocation of the elbow, cuts and pressure on the elbow during sleep and work. Neuritis occasionally causes paralysis. It must be distinguished from spinal disease, which ascends from the dorsal to the cervical region; this form is usually bilateral and is preceded by ulnar anesthesia in tabes and paretic dementia (Biernacki). The *flexor carpi ulnaris* is paralyzed (the hand deviates to the radial side in attempts at flexion), and also the ulnar half of the *deep flexors* of the *fingers,* the muscles of the little finger, palmaris brevis, the interossei, the third and

fourth lumbricals, the adductor and inner head of the short flexor of the thumb (the thumb being rotated toward the palm). The first phalanges cannot be flexed nor the others extended, although this is less marked in the first and second fingers, in which the lumbricals are supplied by the median nerve. The "*claw-hand*" results; the hypothenar eminence, palm and interosseous spaces become thin. *Sensory loss* is limited to the back and front of the ulnar side of the hand, the dorsum of two and a half fingers and the front of one and a half fingers on the ulnar side.

Combined Paralysis of the Arm Nerves.—This common affection, described by Erb (1884), is due to affections of the vertebræ, cord or nerve roots, to tumors, trauma or cicatrices of the neck and wrenches or forcible delivery in obstetrics. Several nerves are involved, as the musculospiral and ulnar in fracture of the humerus, or in subcoracoid luxation. In Erb's case the circumflex, suprascapular and musculocutaneous nerves, fifth and sixth cervical roots, with some branches from the fourth, were imbedded in a cicatrix and paralyses of the deltoid, teres minor, spinati, biceps, brachialis and supinators, with few sensory signs resulted; excision of the scar and nerve suture resulted in recovery; in one spot between the scaleni all these nerves could be stimulated and therefore simultaneously affected. Obstetrical paralysis usually recovers, while adult forms are often protracted or permanent, depending on their etiology. Flaubert (1827) described a combined paralysis, which usually bears Klumpke's (1885) name, in which paralysis of the first dorsal and eighth cervical roots (ulnar and median paralysis) also causes narrowing of the pupil and the interpalpebral fissure.

BRACHIAL NEURITIS.

Gowers first clearly described a perineuritis of the plexus or nerve roots (radicular neuritis), occurring largely in gouty or rheumatic subjects over fifty years of age. *Cervical rib* may cause compression symptoms; Reisman collected 46 cases of which 24 were operated on with good results. But 5 per cent. cause symptoms and 85 per cent. are bilateral, as shown by radioscopy; (*a*) a tumor may be palpated; (*b*) nerve pressure or wasting may be manifest; or (*c*) the subclavian artery, compressed by the scalenus anticus, may be abnormally high or pulsate like an aneurysm; even gangrene may ensue. Neuritis is marked by great pain, the first, most severe and most enduring symptom. It is constant, with paroxysmal exacerbations, is increased by the least movement and is experienced over the plexus, nerves or referred to the wrist or scapula. Associated with it are tenderness over the trunks or plexus, hyperesthesia of the skin, some muscular wasting, mostly in radicular forms, and somewhat less often anesthesia, anesthesia dolorosa, edema, glossy skin and trophic alterations in the joints. Neuralgia, angina pectoris and aneurysm may cause diagnostic difficulties. Remak holds that reported recoveries of acute poliomyelitis in children (Kennedy's temporary paralysis) are only plexus neuritis. A long course of from a month to more than a year, relapses and even wasting, and joint adhesions must be considered in framing the prognosis, despite early and careful treat-

ment. Treatment, as in neuritis, consists of rest by immobilization, injection of cocaine in severe cases and treatment of the underlying trauma, gout or rheumatism.

THE NERVES OF THE TRUNK.

These are rarely involved except in lesions of the spine or cord. The *neuritic pain in Pott's disease* or cord tumor and the *girdle pain of tabes* are of great localizing value. *Herpes zoster* is due to inflammation in the spinal root ganglia.

THE NERVES OF THE LOWER EXTREMITIES.

These nerves, except in sciatica, are less frequently affected than those of the arm.

The Lumbar Plexus.—The first three and a half lumbar roots may suffer in abdominal tumors, adenopathies, parturition, psoas abscess, pelvic inflammations, meningitis or vertebral disease. The symptoms are *paralysis* of the *flexors and adductors* of the *hip* and *extensors of the knee;* all the hip flexors are involved in plexus disease only; in neuritic forms great pain is noted in the ilio-inguinal, iliohypogastric and genito-crural areas. *Anesthesia* exists over the lower abdomen, front and sides of thigh and inner side of leg and foot and adjacent sides of first and second toes.

Anterior Crural Nerve.—Lesions in the abdomen cause paralysis of the iliacus (not the psoas), the hip flexors and knee extensors, with wasting, loss of knee-jerk and anesthesia over the front and sides of the thigh and the inner side of the leg and foot. It may result from stab wounds of the thigh, in which case the iliacus escapes involvement, or after delivery. Neuritis of its *external cutaneous branch* (multiple neuritis, alcoholism, nicotinism or gout) may cause isolated anesthesia over the lower outer half of the thigh, paresthesia or marked hyperesthesia; the last condition, known as *meralgia,* is usually neuritis but may be due to pressure from varicose veins or corsets. It is noted, chiefly, on standing or walking. In 1900 Musser and Sailer collected 99 cases (74 per cent. in men).

Pressure on the cord at the fourth lumbar roots produces the same result as a partial lesion of the ant. crural nerve, as paralysis of knee extensors, loss of knee-jerk and anesthesia of anterior part of thigh, but foot-clonus is present. Anterior horn disease may occur exceptionally, but its wider prevalence and sensory exemption are sufficiently distinctive. Caudal lesions are usually bilateral.

Obturator Nerve.—This nerve is rarely alone involved save during parturition. Its paralysis affects the thigh adductors, so that the knees cannot be crossed, and the pectineus and ext. obturator, with impairment of outward rotation of the thigh. The hip and knee-joints sometimes show trophic disturbance. The pain of hip disease is referred to the knee by the obturator nerve.

Superior Gluteal Nerve.—This arises from the lumbosacral cord, being intermediate between the lumbar and sacral plexuses. Its rare isolated involvement results in loss of thigh abduction and circumduction (gluteus medius and minimus).

The Sacral Plexus.—Half of the fourth, the fifth lumbar and the first four sacral nerves supply motion to the extensors and rotators of the hip, flexors of the knee and all the foot muscles, and sensation to the gluteal region, the back of the thigh, outside and back of the leg and most of the foot by the gluteal nerves and the two sciatic nerves. The *lesser sciatic* is affected only in disease of the plexus; it is often involved in pelvic inflammation and tumors, parturition, traction in leg deliveries and diseases of the hip.

Great Sciatic Nerve.—This is the largest, the most exposed throughout its course and hence the most frequently diseased nerve of the lower extremity. Paralysis of the gluteus maximus interferes with rising more than with walking. A lesion near the sciatic notch paralyzes the extensors of the hip, the flexors of the leg and all muscles below the knee. If the lesion lies below the upper third of the thigh the flexors of the leg may escape involvement and walking is still possible, even though all muscles below the knee are paralyzed, if the ant. crural nerve (hip flexors and knee extensors) is intact. Sensation is lessened over the outer half of the leg, most of the dorsum of the foot and all of the sole. Wasting is marked over the back of the thigh and perforating ulcer of the foot may develop. It has two *branches:* (*a*) the *external popliteal* or *peroneal nerve,* involvement of which causes paralysis of the tibialis anticus, peronei, long toe extensors, extensor brev. digitorum; the ankle cannot be flexed dorsally, the first phalanges of the toes cannot be extended, adduction and lifting the inner edge of the foot are difficult and abduction and lifting the outer edge are impossible. Foot-drop results and contracture assumes the equinovarus type. This nerve is the analogue of the musculospiral in the arm. Wasting is often observed and the anesthesia covers the anterior outer half of the leg and the dorsum of the foot. (*b*) *Internal popliteal nerve,* involvement of which affects the popliteus, tibialis posticus, long flexors of toes and all the foot muscles except the extensors; plantar flexion, abduction of the foot, inability to stand on tiptoe, talipes calcaneus and the claw-foot from overextension of the proximal and overflexion of the second and third phalanges result. Sensory alterations are variable; anesthesia may be observed on the outer lower posterior part of the leg and on the sole of the foot. Its plantar branches are rarely affected alone; (i) paralysis of the internal plantar nerve, which is analogous to the median; anesthesia on the inner part of the sole and the plantar surface of the three inner toes and one-half of the fourth; paralysis of the short toe flexors, plantar muscles of great toe (except the abductor) and the two inner lumbricals; the big toe is overextended, "the hammer toe"; (ii) paralysis of the external plantar nerve, the analogue of the ulnar, produces anesthesia of the outer half of the sole, little toe and one-half the fourth, paralysis of the accessory flexor, small toe muscles, all the interossei, two outer lumbricals and great toe adductor; the results are serious, walking being impaired by flexion of the second and third and extension of the first phalanges.

Caudal Lesions.—(See Spinal Localization.)

Sciatic Neuritis.—Sciatica, Ischialgia, Malum Cotunnii (Cotugno, 1764) is, after facial neuritis, the most frequent isolated neuritis.

Etiology.—1. General Causes.—(*a*) Sex and age; 88 per cent. of cases occur in men; 16 per cent. between twenty and thirty, 31 per cent. between thirty and forty, 25 per cent. between forty and fifty and 18 per cent. between fifty and sixty years of age. It is very rare in children. (*b*) Acute infections, as rheumatism and grippe are causative. (*c*) Intoxications; gout, fascial rheumatism (sometimes by extension from lumbago) and diabetes are factors. (*d*) Anemias and neuroses are less significant than in neuralgia.

2. Local causes include (*a*) spinal affections, tumor and caudal neuritis; (*b*) abdominal and pelvic diseases, as aneurysm, tumors or inflammation; (*c*) cold, exposure and dampness, favored by certain occupations; (*d*) trauma to the spine, pelvis, hip or peripheral course of the sciatic trunk or branches, as from the use of forceps or pressure of the head in labor, fractures, dislocations, falls, protracted sitting or riding, use of the sewing machine, marching, peripheral pressure by popliteal aneurysm, tumors, callus or varicosities.

Pathology.—The infrequent autopsies or operations show perineuritis with edema and in some cases increase of connective tissue or varicosities of the sheath vessels, especially in the mid-thigh. Sometimes interstitial neuritis is found.

Symptoms.—The symptoms are unilateral and chiefly sensory, as *pain and tenderness* over the sciatic roots, trunk and branches. (*a*) The onset of *pain* is usually gradual but is sudden and febrile in some rheumatic cases. It is usually constant, with exacerbations which may be agonizing. The pain descends more often than it ascends; it sometimes radiates to the buttocks, crural region, even into the other leg. The local pain is perineuritis and the distant pain interstitial neuritis. Any movement which increases sciatic tension increases or renews the pain (Laségue's phenomenon), as flexing the hip, extending the knee, walking, stooping or defecation. The patient seeks positions which spare stretching of the nerve and its compression by the muscles; he may lie on the opposite side with the thigh extended, knee flexed and foot extended, or on his back with hip and knee flexed and foot extended; in standing, he rests on the sound leg and in protracted cases scoliosis develops (ischias scoliotica), in which the lumbar convexity is toward the diseased side, heterologous scoliosis, which usually disappears with the disease; a homologous scoliosis (with the lumbar concavity toward the affected side) may result in case the muscles of that side become spasmodic. (*b*) *Tenderness* may be detected over the entire course of the nerve or in certain locations only: (i) over the lumbar region; (ii) near the sacrum and post. sup. iliac spine; (iii) at the lower gluteal fold where it leaves the sciatic notch; (iv) between the tuber ischii and trochanter; (v) especially over the middle of the *posterior surface of the thigh;* and (vi) lower over the popliteal space, head of fibula, calf, malleoli and sole. The enlarged nerve may occasionally be felt. Pain is often experienced in the hip and sciatic notch when pressure is made in the popliteal space. (*c*) Other symptoms are less frequent, as vasomotor changes, herpes, muscular cramps, twitchings,

fibrillation or myokymia; there is rarely a marked degenerative reaction; muscular atrophy from disuse or neuritic atrophy, sciatic paralysis, hypesthesia, paresthesia, or in severe cases anesthesia, may develop in the posterior part of the thigh, leg or foot. The patellar reflex is usually normal, the Achilles reflex is often decreased or absent and the cremasteric usually increased.

Diagnosis.—(*a*) The etiological factor should be sought. (*b*) The pelvis must be examined in women and the rectum in both sexes, because compression produces more distant than local pain; the urine should always be examined for sugar and albumin; and free catharsis and bowel lavage given. (*c*) Bilateral sciatic pain suggests diabetes, or disease of the cord, especially tabes, or a lesion of the cauda, in both of which there is no sciatic tenderness. (*d*) Coxitis, sacro-iliac and psoas disease present appropriate local signs, without Lasègue's sign or sciatic tenderness, both of which are also absent in (*e*) hysteria. (*f*) Muscular rheumatism is chiefly confused with pain over the sciatic branches.

Prognosis.—The prognosis, generally good, depends on (*a*) whether the affection is primary, as from cold, or secondary, as from disease of the cord or pelvic neoplasm; (*b*) the amount of rest which the patient accepts; (*c*) the intensity of the neuritis, which may extend to the lumbosacral plexus; and (*d*) the chance of relapse (33 per cent.). The average course is from two to eight weeks, but cases may last a year, indeed three to thirty years.

Treatment.—(*a*) Treatment is that of the *cause;* the salicylates, in some, but unfortunately few, cases afford relief; gouty or syphilitic taints may be benefited by colchicum or antisyphilitic remedies, which are often given as a last resort in other cases. (*b*) *Rest in bed* is the most important indication. In this, extension or Weir Mitchell's long splint is often beneficial. (*c*) *Counter-irritation* by blisters over the nerve's course or by touching the tender or painful points with the hot iron, may give temporary, though rarely lasting, relief. (*d*) *Analgesics and narcotics.* Acetanilide has little effect. Gelsemium (flextr. ℥ij) at intervals of two to four hours, not infrequently gives relief, though it is necessary to watch for pulse weakness and cutaneous tingling. Codeine and atropine should be given before resorting to morphine; morphine is often inevitable, but chronic morphinism may be initiated by severe pain, the long course and tendency to relapse. Hypodermics of cocaine are very beneficial, though attended by the same danger. The author has seen good results from deep hypodermic use of Schleich's solution given in quite large amounts over sensitive points.[1] Injections of chloroform and ether should be avoided because of the possibility of

[1] Hecht found the sciatic at its point of exit from the great sacrosciatic foramen in the following manner: A line is drawn with tincture of iodine from the sacrococcygeal articulation to the postero-external border of the great trochanter; at the junction of the inner one-third with the outer two-thirds of this line is the spine of the ischium. Placing the needle a thumb's breadth to the outer side of this point and penetrating in a directly perpendicular line to a variable depth, one engages the sciatic nerve. The needle with the stylet partly withdrawn is put through the skin, and when well in the subcutaneous tissue the stylet is returned to cover the point and the needle is directed perpendicularly to the necessary depth. The moment the nerve is touched the patient feels a sharp, lancinating pain at the heel or in the calf associated with a quick, jerky movement. Occasionally the pain is felt in the buttock, thigh, or at the knee. The filled syringe is now attached and the solution (60 c.c.) injected slowly with steady, firm pressure.

sloughing and accentuating the neuritis. (*e*) *Acupuncture,* the old Chinese treatment, is recommended; it resulted in complete relief in 56 per cent. of Gibson's cases, improvement in 32 per cent., slight benefit in 10 per cent. and failure in but 2 per cent. (*f*) *Massage.* Negro obtained results from frequent and severe local rubbing and rolling, over the nerve trunk. (*g*) *Electrotherapy* may aggravate the pain and is at least of uncertain value. (*h*) *Nerve stretching* may be effected by extension by weights, although it is rarely tolerated by the patient in personal experience. The direct exposure of the nerve has justly fallen into disuse, for the same results attend simple stretching, performed under anesthesia. The hip should be strongly flexed, the knee extended, the foot dorsally flexed and the pelvis firmly immobilized. Ordinary care prevents luxation of the hip. The results were excellent in half of 20 personal cases and were seemingly curative in 7.

THE NEUROSES.

HYSTERIA.

Definition.—The neuroses are nervous affections, provisionally designated "functional," since no anatomical basis has been discovered. Möbius considers hysteria "a state in which ideas control the body and produce morbid changes in its functions." The school of Charcot established its psychogenic nature. It is a cerebral, cortical condition, in which the sensory and motor centres are affected, the perception, association and recollection of ideas are disturbed and trophic and vasomotor disorders occur. As its name implies, hysteria was long thought to be due to the uterus; Sydenham first called it a nervous affection.

Etiology.—(*a*) *Age:* It is most frequent at puberty and adolescence (fifteen to twenty-five years). It is occasionally seen in children and rarely in middle or advanced life. (*b*) *Sex:* Women are ten times as frequently affected as men, although in the lower French classes it is more frequent in males. (*c*) *Race:* The Jewish, Latin and Slavic races are most frequently affected. (*d*) *Heredity:* Charcot and the French school hold that this is the sole cause, other factors being only *agents provocateurs.* Parental intoxications, toxemias, constitutional diseases, as gout, diabetes, arthritis deformans and a neurotic taint are important in the transmission of a congenitally weak nervous system. (*e*) *Acquired hysteria* may be due (i) to various intoxications, notably lead, alcohol and drugs; (ii) to infections, as typhoid, malaria, diphtheria and syphilis; (iii) to constitutional maladies, as gout, diabetes, chlorosis, secondary anemia or cancer; (iv) to exhaustion from mental or physical overwork or excesses; (v) to emotional disturbance; (vi) to trauma, especially psychic shock; (vii) it may occur as an associate with epilepsy, syringomyelia, tabes, brain tumor and multiple sclerosis; (viii) imitation is an important factor in schools, hospitals and at religious revivals; endemics occur now as they did in the middle ages. (Refer to Zola's *Lourdes.*) Reference to sexual factors is considered under Therapy.

Symptoms.—The disease is much more commonly polysymptomatic than monosymptomatic. French writers distinguish two main groups of symptoms, the *stigmata* and *accidents*.

STIGMATA.—The stigmata are usually persistent symptoms.

1. *Sensory Stigmata.*—Sensory symptoms (in 85 per cent.) are more frequent than the motor and are often combined with them. (*a*) Hysterical *anesthesia* is very seldom absent. It must often be looked for, without too obviously suggesting it to the patient. It may be absolute, affecting appreciation of touch, pain, heat and cold. *Analgesia* is its most common form and, in decreasing order of frequency, there may be complete anesthesia, hypesthesia, loss of tactile sense, loss of all sensation save to the faradic current or loss of faradic response alone. The same deficit frequently occurs in the mucosæ and sometimes in the bones, ligaments, joints and muscles. In rare cases there is universal cutaneous and muscular anesthesia; the following are more frequent: Hysterical *hemianesthesia,* which involves precisely one-half of the skin, accessible mucosæ and special senses; *segmentary* or geometrical anesthesia, which affects one limb, variously described as "mitten or stocking" anesthesia and frequently associated with other disturbance in the part affected, as paralysis, aphonia, amblyopia and deafness; and *insular* anesthesia, which affects small areas, and is therefore frequently overlooked.

Characteristics.—The anesthesia is psychical, is associated with the functions of the part affected and does not follow the area of distribution of the peripheral nerves or spinal segments; it is not noted by the patient, at least until suggested; it may be localized because of injury or disease in the part. Babinski holds that all anesthesias are initiated by suggestions. The anesthetic areas may last during life, but they move even under examination or disappear from emotion, strongly aroused attention, drugs or on application of various substances, as metal or wood (*transferring* it to the opposite side). Most of the reflexes usually altered by organic disease remain normal, as the pupillary, lachrymal, epigastric, cremasteric, patellar and organic reflexes, while the pharyngeal in 90 per cent. of cases is lost, as well as the plantar.

Hysterical hyperesthesia and *hyperalgesia* are very common in localized areas, but never in universal or hemiplegic distribution. They occur spontaneously or from attention directed to a part or organ, as by traumatism. A joint may be affected with or without contracture (Brodie's joints) and this often leads to great and unfortunate errors. There is sometimes hyperesthesia in the sphere of any of the special senses. The feeling of a nail in the top of the head (clavus hystericus), sensitive spots in the mammæ, epigastrium, groins or spine are the most common forms and may occur in the midst of anesthetic areas. The so-called ovaralgia is cutaneous; it has no relation to the location or to disease of the ovary; it occurs also in men. Because hysterical attacks have been initiated and sometimes stopped by pressure over this and other sensitive areas they have been called *hysterogenic zones,* but attacks are only so produced in patients already hysterical, from some associated memory of the patient, or from suggestion. *Taste* and *smell* may be dulled or hypersensitive, and *hearing* may be blunted, or more rarely complete central deafness results, usually associated with anesthesia in and about

the auditory meatus. Complete *loss of vision* is both rare and transitory. Hemiopia and scotomata are almost always organic. Partial stigmata are more common. *Concentric contraction of the visual field* is usually bilateral, and generally greater on the side of the cutaneous defects. Concentric *contraction of the field for colors* (dyschromatopsia) is more characteristic. In the normal eye, blue has the largest field, then yellow, orange, red, green and violet; in hysteria the red field is often wider than the blue and is the last color lost. Achromatopsia designates the complete loss of color perception. Errors in accommodation (always excluding abnormal corneal curvatures) may cause double images in one eye (monocular diplopia), triple images (polyopia) or large or small images (macropsia, micropsia). In hysterical *amblyopia* impairment of vision in the affected eye is decreased when the other eye is opened.

2. *Motor stigmata* are much less conspicuous but foreshadow the motor accidents (*v. i.*). Voluntary movements are weaker, slower, less coördinate and with a tendency toward contractures.

3. *Mental Stigmata.*—Hysteria is always a psychic affection. The most conspicuous alienation is impairment of memory (*amnesia*), which concerns memory in the ordinarily accepted sense, as memory for persons, things, words, hysterical paroxysms experienced, etc., or concerns memory and associations for speech, writing, walking or standing. The amnesia, like paralysis and anesthesia, is functional and like them returns, save in rare cases. *Mental power* is distinctly decreased, *will-power is lost* (aboulia) or lessened and the patient becomes the ready victim of impulses, sensationalism, suggestions, fixed ideas and misconceptions both on his own part and on that of others who incorrectly consider him deceitful, vain or erotic.

Accidents.—The accidents of hysteria are the incidental *seizures or paroxysms*. They are usually transient or intermittent, but may become fixed and are then ranked as stigmata.

1. *Motor Accidents.*—(*a*) *Convulsions* occur in 25 per cent. of all and 50 per cent. of female hysteria. They are much less frequently the hysteria major (*grande attaque* of Charcot) than the partial, modified type (hysteria minor). The "*grand attack*" comprises (i) a prodromal stage, with mental depression or exaltation, palpitation, vasomotor symptoms, nausea, polyuria, an aura beginning in the epigastrium and culminating in choking (globus hystericus), vertigo and unconsciousness; (ii) a period of epileptiform convulsions (so-called hystero-epilepsy) in which, in exceptional cases, there may be biting of the tongue or even the cry of epilepsy; the face is at first pale, then red; the teeth are ground; the convulsion is most pronounced on the anesthetic side, toward which the face turns; the tonic spasm lasts less than two minutes and is often characterized by stupor, stertor and slow, wide movements of supination, circumduction and overextension, for the trunk and body are tetanically rigid; then the clonic spasm develops, with sobbing respiration, audible swallowing, abdominal rumbling and small, clonic movements. (iii) The period of "clownism" is characterized by contortions, opisthotonos (*arc de cercle*), bowing movements, violent struggling, screams, strikes and bites. (iv) There is next a period of "passional attitudes," in which the subject mimics terror, rage, joy and love, fol-

lowed by (v) a stage of delirium with hallucinations, perhaps of animals (zoöpsia). The attack covers fifteen to thirty minutes and frequent repetitions constitute the "status hystericus."

The *partial attack* contains some of the elements of the major type, and infinite variations occur; the globus type is extremely common; there are also the vertiginous, tetanic, emotional and syncopal types; trance-like sleep, with catalepsy in which the limbs retain any attitude given them, with sleep, which is apparent only, for the subject later recalls everything; there may be attacks of somnambulism, in which patients run miles or undertake distant travel without recollection of the transpired interval. (*b*) *Paralysis* is an accentuation of the stigmatic muscular weakness. In over 50 per cent. it develops after convulsive paroxysms; again it follows emotional causes, traumatism or organic brain disease. It assumes the paraplegic five times as often as the hemiplegic form; the monoplegic variety is less frequent; the triplegic, diplegic (quadriplegic) or crossed forms are most common. They are rarely complete; often gesticulatory movements may be seen; the antagonistic muscles are involved, thus, in attempted flexion of the knee the knee extensors contract; the paralyzed part may be limp or contractured; the paralyses may be ephemeral or life-long in duration; paralysis may be "ideal," *i. e.*, only for standing or walking. Vasomotor changes and edema are rare; the tendon, skin and electrical reactions are usually normal, although the tendon reflexes may be increased. *Hysterical hemiplegia* occurs three times as frequently on the left as on the right side.

Organic Hemiplegia.	Hysterical Hemiplegia.
Coma: often complete.	Rarely complete (automatic movements.)
Reflexes: tendon and bone reflexes abolished at first; later increased. Ankle-clonus.	Rarely abolished; no clonus (or only a spurious ankle-clonus due to contraction of the calf muscles, Gowers).
Cutaneous reflexes (abdominal and cremasteric) early lessened or abolished; Babinski toe sign; lachrymal reflex abolished.	Skin reflexes normal; no toe sign.
Paralysis: (1) Unilateral; (2) not systematized; face, arm and leg are weak during bilateral synergetic movements. (3) Involves subconscious as well as conscious voluntary movements, producing the platysma sign (lessening of its contraction on the hemiplegic side, *e. g.*, on opening the mouth forcibly), and the combined flexion of the thigh and trunk (when the patient attempts to sit up). (4) Paralysis slowly improves some, but is not variable.	Never abolished. (1) Not always unilateral; if face involved, usually on both sides. (2) Paralysis sometimes systematized; and hemiplegic side functionates perfectly in bilateral synergetic movements. (3) Subconscious voluntary movements are not involved; absence of the platysma sign and that of combined flexion of the thigh and trunk. (4) Variable paralysis, improvement alternating with relapses. Absolute aphasia with ability to write (Charcot).
The *tongue* deviates slightly toward the paralyzed side.	Very slightly, very greatly or even toward the sound side.
Lower muscular tone, as lowering of the eyebrow or exaggerated flexion of the forearm, in early cases.	None. Asymmetry of the face is due to spasm, and exaggerated flexion of the forearm is absent.
Contracture cannot be voluntarily reproduced; at first flaccid paralysis, then contracture.	May be reproduced; variable paralysis, now flaccid, now spastic from onset, with increased reflexes.
Hemianesthesia: rarely total; of stereognostic and muscle sense; rare; coarser; more lasting when present; often with bilateral hemianopsia.	Often total, involving special senses, pain and pressure sense; frequent; variable in duration; usually with contraction of visual field.

In hysterical *paraplegia* there are usually no trophic changes, no bed-sores, no electrical alteration, no incontinence of urine or feces and rarely retention of urine; many errors in diagnosis are made. Hysterical *monoplegia* usually occurs with anesthesia of even greater extent; it is lawless and illogical, viewed from the standpoint of organic disease. In *astasia-abasia* (Charcot and Blocq) the patient is unable to stand or walk, although the muscles concerned may be efficient in other movements. Lesser degrees are called *dystasia* or *dysbasia*. The term stasobasophobia is self-descriptive. Möbius' akinesia algera is similar. (*c*) *Hysterical contractures* precede, succeed or alternate with paralysis, or occur alone; these "rigid palsies" usually persist during sleep and yield to ether anesthesia, save in severe protracted cases with irremediable deformity. In the arms, contracture of the flexors predominates; in the legs, that of the extensors (club-foot). Spinal deformities may develop, most frequently from pseudosciatica. The muscles may so contract as to resemble muscle tumors. It is stated that the *face* escapes paralysis, but its lower portion may be weak; more often, however, it is contracted early, even when paralyzed; hemispasmus glossolabialis and trismus have been observed. Spastic or pseudoptosis may resemble the paralytic form, but spasticity, the lowered or more level eyebrow and the concentric or parallel folds in the lid are distinctive of contracture. The ocular muscles are seldom involved; voluntary movement may be impossible, but associated and reflex movements are normal; convergent squint may occur, but never the divergent type nor contracture of a single rectus or oblique muscle; pain suggests hysteria. Karplus maintains that the pupil light reflex is lost during convulsive seizures. Organic disease of the *hip* may be simulated; local hysterical edema may occur. Brodie (1827) first drew attention to this group of cases. Many avoidable diagnostic errors are constantly made. (*d*) Rhythmical spasms, chorea major, the saltatoric chorea (*v. i.*) and local manifestations, as barking cough or the various tics, may occur. Hysterical tremor occurs without voluntary movement. It may resemble the senile, Basedow, paralysis agitans or lead, but rarely the multiple sclerosis tremor.

2. *Sensory Accidents.*—These are frequent, severe and painful. Headache is often dull or may resemble trigeminal neuralgia, migraine or organic disease, as tumor. Meningitis (hysterical pseudomeningitis) may be closely counterfeited. Spinal or vertebral pain is frequent and sometimes suggests tumor or caries. One and three-tenths per cent. of cases of sciatica are said to be hysterical. Joint disease and various visceral lesions (*v. i.*) may be closely mimicked. Dieulafoy collated 59 cases of hysterical blindness, in 5 of which this accident was the only symptom.

3. *Visceral Accidents.*—(*a*) *Cardiac* accidents include tachycardia, palpitation and pseudo-angina pectoris. (*b*) *Respiratory* accidents: *Aphonia*, usually attended by normal capacity to cough and often also to sing, has been described under Paralysis of the Vocal Cords. It is usually due to an "idea," as in a motor aphasia which allows the patient to write. Stammering, cough and cries may be provoked by local naso-

pharyngeal disease. Dyspnea, resulting from paralysis of the diaphragm, and laryngeal spasm have been considered. Rapid breathing (tachypnea) also occurs; dyspnea, aphonia and paralysis of the diaphragm constitute Briquet's syndrome. Hemoptysis may occur, sometimes without phthisis, possibly as a vasomotor derangement; one fatal case is on record. (*c*) *Digestive* accidents: Hysterical anorexia results from cortical conception, stomach pain, dysphagia from spasm or simple sensory perversion; it may endure for weeks and cause extreme inanition or even death. Hysterical (pseudo-) hydrophobia is an analogous aversion to water. Vomiting is sometimes simulated by protracted globus or esophageal spasm, in which the food is simply regurgitated. Vomiting itself may be occasional or attacks may last weeks or months, often with remarkably little malnutrition; it is rarely fecal but substances injected by rectum may be vomited in a few hours; or again vomitus may be blood-tinged, possibly from vasomotor disturbance or more often from actual disease; ulcer may be simulated by pain and tenderness, which are surprisingly well confined not to one spot, but to the exact outlines of the stomach. The vomitus contains vicariously large amounts of urea when the urine is suppressed. Eructations are frequent from swallowed air; this also causes tympanites, as air reaches the gut through the incompetent pylorus. Phantom tumors or spurious pregnancy (pseudocyesis) are explained as protrusion of the distended bowel by tonic contracture of the diaphragm. Peristaltic unrest, diarrhea on eating, enteritis membranacea, spastic constipation, simulation of peritonitis, rectal stricture and intestinal obstruction also occur. (*d*) *Genito-urinary* accidents: Anuria has existed for ten days without uremia. Renal pain is not frequent, but hyperesthesia of the bladder is common. The urine is often greatly increased, with low specific gravity and solids. During attacks of the major type the solids are reduced, the urea one-third and phosphates one-half; the phosphates, which normally have a proportion of 3 of the alkaline to 1 of the earthy phosphates, show nearly an equal proportion of each. Depression of sexual desire is more common than exaltation; anesthesia is frequent; vaginismus may be observed. Hysterical ovarian and other pains are not related to pelvic disease.

Hysterical fever is a disputed topic. Severe forms may suffer a rise to 105° or 110°; this may lead to a diagnosis of meningitis, peritonitis, etc.; simulation by the patient rubbing the thermometer, or of confusion with tuberculosis or typhoid, must be kept in mind.

4. *Vasomotor and trophic accidents* are rare. Erythema is the most common. Gangrene, falling out of the hair or nails, Raynaud's local asphyxia, cutaneous hemorrhages (crucifixion stigmata, stigmata diaboli), bloody sweats, lachrymation, hemoptysis, hematemesis, sudden tender swellings in the mammæ and angioneurotic edema, which is sometimes blue in color, have been recorded.

Course and Prognosis.—The course is always chronic. If we regard the disease as congenital the prognosis is poor as to total recovery, especially with fixed psychical alteration and major manifestations. In mild forms, relapses may occur on disproportionately slight provocation. The outlook is better in infantile and adolescent forms. The stigmata

may be ephemeral or life-long. Marriage may benefit light cases, but, with maternity, it is injurious in the severe type.

Diagnosis.—The stigmata, mental, motor and sensory, are highly characteristic. The accidents are unequivocally hysterical or prove hysterical after consideration of their associations. The danger lies in overlooking concomitant disease (*a*) nervous, as multiple sclerosis, brain tumor, paretic dementia; (*b*) postinfectious hysteria, as hysteria plus neuritis; (*c*) lung, heart and other visceral diseases, all of which, as Sydenham remarked, may resemble hysteria. Stimulation does not include mimicry of symptoms by hysterics; simulation of hysterical stigmata and accidents is practically impossible.

Treatment.—1. Prophylaxis.—When the earliest manifestations occur in childhood, home treatment is impracticable because of the family tendency. The etiological factors must be studied, especially the hygiene and hours of sleep. Useful occupations, as out-of-door gardening, are preferable to more artificial plans of distraction. Cultivation of courage and self-control, is sought. Servants who teach fear of the dark and tell fairy stories should be watched.

2. General Treatment of the Cause.—As the disease depends on disturbed cortical relations between the psychical and material processes, the principal treatment must be psychical. The "fixed idea" must be grasped and eradicated, which is best accomplished in ordinary circumstances by isolation of the patient from home or psychical or emotional circumstances surrounding the inception of the disease. Then the undue susceptibility incident to the disease may be turned to therapeutic advantage by the self-confidence of the physician and by his covert suggestions. Babinski defines hysteria as that series of phenomena produced by suggestion and cured by persuasion. The Weir Mitchell rest-cure may result in great benefit, and later "vague hints regarding the curative power of nature," and fresh air and sunlight may captivate the patient. Symptoms must not be suggested to the subject, as anesthesia or pelvic disease, and examinations, especially of the pelvis, should be omitted. Hysterics should not be allowed to associate with each other for obvious reasons. This general plan is psychotherapy, which might include hypnotherapy, as advocated by the Nancy school, Bernheim and others. Hypnotism is generally dangerous in its ultimate and often in its immediate effects; the danger of hypnotism is shown by the fact that Strümpell considers it an "artificial hysteria," and Féré defines it as a "transformation of hysteria." "It is easier to make a sound person hysterical by hypnosis than to cure an hysterical one by it" (Liebermeister). Treatment is sometimes frustrated by the patient's vanity. Successful treatment requires unusual tact, profound knowledge of human nature, strong sympathy (but dominance of the patient) and enormous optimism; certain single qualities in an ignorant quack may effect cures.

According to Breuer and Freud, certain sexual activities, mostly of a perverse nature, develop before puberty; these phantasies are mostly repressed by the hysteric and after puberty these repressed perversions come in conflict with the normal sexual proclivities. Freud traces every

hysteria back to sexual trauma in childhood. The analytic or cathartic method consists of full narration or confession by the patient. The repressed complexes, parasitically attached to or dominating the mental states, are analyzed and replaced by proper mental reactions—a process of reëducation. The subconscious, repressed complexes are brought back into higher consciousness, lived over, given verbal expression and therefore eliminated, forgotten.

3. Symptomatic Treatment.—(*a*) In relieving *pain* the physician should first acknowledge its existence and intensity to soothe the patient and then apply blisters, liniments or the galvanic current with the anode on the painful area. Ovaralgia in most cases should be ignored; in exceptional cases pelvic surgery has given relief. (*b*) *Convulsions* are treated by dashing cold water into the face, by suggestive measures or, if severe, by etherization. Bromides are of little value, valerian is sometimes beneficial and turpentine is recommended by Gowers as most efficient, 10 drops of the oil being given until slight vesical symptoms appear. Pouring water into the mouth or nose, which causes coughing, or holding a towel over the nose and mouth for fifteen to twenty seconds, may also serve. (*c*) *Paralysis* and *contracture* should receive early treatment, for neglected cases may necessitate surgical intervention. Massage, faradism, blisters and fixation in new postures are indicated and especially should their significance be minimized and the patient be assured that they are not organic. In 2 cases the author has seen the paralysis disappear at once on administration of asafetida in an effervescing mixture. (*d*) *Anesthesia* should be ignored. (*e*) *Aphonia* is treated by intralaryngeal faradization. Laryngeal and other respiratory spasms usually answer to energetic traction on the tongue. (*f*) For *insomnia*, the patient should retire at an absolutely regular hour; nervines should be given rather than bromides, and hot milk should be very slowly sipped. (*g*) *Dysphagia*, vomiting and anorexia may be treated by rectal feeding or by forced feeding through the stomach-tube. Constipation is often obstinate and its mixed spastic character is shown by the sheep-like feces. Olive oil by mouth and rectum, cold abdominal compresses and endorectal galvanization are usually effective.

NEURASTHENIA.

Definition.—Neurasthenia is a diffuse neurosis, which affects the entire nervous system, but chiefly the brain; it is characterized (*a*) by morbid irritability of the nervous system with a tendency to its rapid exhaustion ("irritable weakness"), and (*b*) particularly by psychical, motor, sensory, vasomotor, secretory and vegetative disturbances in function. The nervous tissue responds unduly to stimuli, its reaction time is short and ready exhaustion results. It seems probable that the slight cellular and protoplasmic changes in nerve cells, noted by Hodge to follow fatigue, may have some bearing on its pathology. The affection described and named by Beard, in 1869, whose description, first received with skepticism and ridicule abroad, later gained universal recognition. It is possible that the disease is more frequent than formerly because of the

more strenuous life and methods of education. The difference between "nervousness" and neurasthenia is only quantitative.

Etiology.—Neurasthenia constitutes 35 per cent. of nervous diseases and 50 per cent. of neuroses. (*a*) A *neuropathic tendency* is observed in 50 per cent. of cases, especially among Hebrews whose proneness to nervous diseases is referred by Erb to inbreeding and desire of gain. Other neuroses, as hysteria, epilepsy or migraine, the psychoses and parental infirmities or intoxications may be obtained in the family history. (*b*) *Age and Sex:* 75 per cent. of cases occur in persons between the twentieth and fiftieth year. Women are more disposed but men more exposed, to the disease by their excesses in work, sexual activity and alcoholism. (*c*) *Psychical* causes include sorrow and care; hard mental or physical work rarely produces neurasthenia. Worry is a prolific factor; it is the irritable weakness of work. Hard work is injurious when combined with excesses in tobacco, alcohol, coffee, drugs or venery. Certain occupations entailing responsibility or emotional factors predispose to nervous weakness, as in railway or telegraph employees, board-of-trade men, teachers, actors, artists and musicians. Trauma operates chiefly through psychical causes, as in the traumatic neuroses (*q. v.*). (*d*) *Intoxications,* as from stimulants, drug habits, lead or arsenic poisoning, infections, especially typhoid, grippe, malaria and syphilis, and gout, anemia or diabetes may be indirect factors. (*e*) *Sexual life:* Excessive coitus and coitus interruptus, sometimes promote neurasthenia, but their importance is enormously overestimated. Masturbation produces less direct physical than psychical injury, for it leads to brooding over its possible results (*v. s.* Freud's theory). Continence is never injurious, as sexual indulgence is rather a habit than a necessity. In neurasthenic women gynecological complaints are more often subjective, secondary and neurasthenic than objective, primary and organic. The same statement holds in male neurasthenics with chronic urethritis or prostatitis. Pregnancy and lactation are sometimes apparent causes. (*f*) *Organic diseases* may possibly be factors, but it is questionable to what extent enteroptosis, chronic gastric or other somatic lesions promote neurasthenia.

Symptoms.—A clinical description holding for all cases is impossible. A patient presents himself for the treatment of some single visceral complaint or describes symptoms in his head, heart, lung, stomach, sexual or other organs. The various types will be considered in the general picture, for pure forms are rare; they are the cerebral (*cerebrasthenia*), spinal (*myelasthenia*), cerebrospinal (*the general type*), the *sympathetic* or vasomotor and the *visceral* (cardiac, sexual, gastric) types. (*a*) The *acute form* from simple nervous exhaustion in normal individuals, or in those with neurotic stigmata; (*b*) the *subacute* or chronic acquired form and (*c*) the chronic *constitutional* neurasthenia, which is usually hereditary.

1. Cerebral Symptoms.—These are most important. Headache is almost invariable, is more often dull than acute, produces a sense of pressure ("lead-cap" headache) or intracranial paresthesia and is most often occipital. Vertigo is frequent. *Insomnia* is extremely common,

but rarely absolute, which always suggests organic brain disease; sleep is slow in onset, the patient being kept awake by his fear of sleeplessness, by a rapidly moving circle of uncontrollable memory pictures or by indigestion or palpitation; sleep, when once attained, is restless or the patient awakes in the night or early morning. It is broken by dreams, startings or sometimes emissions. In mild cases distinct *alterations in temperament* and character develop, as *loss of courage* and confidence, depression, *irritability,* undue response to emotional stimulation, as on seeing sad plays or hearing hard-luck stories, slight ethical blunting, tendency toward extremes and development of quiet self-concentration or obtrusive egotism. The brain tires readily on mental or bodily exertion, especially as the irritable patient is often prodigal of time and energy. It is remarkable how in some cases the morning depression may disappear before an afternoon or evening excitement, but this is notably followed by reaction. Psychical processes are disturbed; perception is blunted and memory is weakened or often temporarily lost (dys-, para-, amnesia), as shown by the copious notes of his symptoms which the patient produces in the physician's office (*"l'homme aux petits papiers,"* as Charcot called him); association and combination are difficult, as in thought, composition, judgment or conclusions, whence the speech is often slow and disjointed, names are forgotten and the patient re-reads addresses, runs repeatedly to see if the door was closed, etc. Compulsory conceptions may lead to homicide or suicide in highly psychopathic types. Fear, being an attribute of the weak, is common enough, and may in marked types assume the form of various phobias, as the fear of open places (agarophobia), of enclosures (claustrophobia), rivers (potomophobia), high places (acrophobia), stars (asterophobia), railways (siderdromophobia), dirt (mysophobia), of high things falling (batophobia), of standing (stasophobia), or walking (basophobia), of darkness (nyctophobia), of a single thing (monophobia), of all things (pantophobia), of places (topophobia), of men (anthrophobia) or of disease (nosophobia). Fibrillation of the lids and wide, variable, sensitive and unequal (3 per cent.) pupils are frequent; permanent irregularity is almost always organic. Asthenopia is very common, especially in hypermetropia from weakness of the ciliary and int. recti muscles. The field of vision may be concentrically limited, but this is rarely marked or permanent, and Förrter's shifting type may be found, in which objects brought from without into the field are better seen than those moved from within the field outward. Hyperesthesia of the retina and ear, ringing in the ears, Ménière's complex and disturbance of taste or smell are not common.

2. Spinal Symptoms.—These include the so-called "spinal irritation." Many symptoms ranked as spinal are essentially cerebral. In women they are more common and are localized chiefly to the upper dorsal and coccygeal regions; in men they are lumbodorsal and most frequent in the sexual type. *Sensory* symptoms are constant. Hyperesthesia is common in the bodies of the muscles; paresthesia is frequent, as tinglings, girdle sensation or itching. Paralgesia in the form of headache (rhachialgia) is as frequent as headache and the pain may radiate into the various nerve plexuses; it is very often sacral and cutaneous; the

spine may be exquisitely sensitive to pressure. Neuralgia is rare. Anesthesia is no part of neurasthenia. Of the *motor* symptoms, myasthenia is the rule, spinal and cortical in origin and conspicuous in the morning. The muscles often show fibrillary contractions and a *tremor* (85 per cent.) which is as fine and frequent as that of alcoholism or Graves's disease. Muscular fidgets, starts, cramps and irritability of the nerve trunks, as in tetany, are also observed. Actual ataxia is most rare, although suggested by the writing. The akinesia algera (Möbius) or the abasia-astasia of Blocq, as in hysteria or the psychoses, is the result of a phobia or a fixed conception. One of Erb's patients remained in bed for fourteen years. The skin, tendon and periosteal reflexes are increased.

3. Vasomotor Symptoms.—These produce many central, visceral and peripheral manifestations. During brainwork the cerebral vessels normally dilate while those of the arm contract; this does not occur in neurasthenia. The manometer in normal cases reveals no essential variation during work, while in neurasthenia marked manometric variations occur. Neurasthenics often suffer from cold and wear more clothes than is necessary; the skin is cold, clammy, even cyanotic or locally asphyxiated. On the other hand, the skin may be flushed and hot. Salivation or a dry mouth, polyuria, pseudo-angina pectoris, the wide temporal vessels, epigastric pulsation, edema, urticaria and dermographism are but vasomotor manifestations.

4. Visceral Manifestations.—(*a*) Some of the *cardiovascular* signs and symptoms have been treated. The arteries relax and throb, the pulse may be slightly of the "water-hammer" type and capillary pulsation is often apparent in the lips or nails. Palpitation, precordial anxiety and dyspnea are frequent. The pulse is frequently faster and may increase 10 or 20 beats on pressure over some sensitive area (*v. i.* Traumatic Neuroses). Many neurasthenics seek consultation for cardiac disease. (*b*) *Gastric symptoms* (in 60 per cent.) are functional in character, as achlorhydria, hyperchlorhydria, hyperesthesia. Anorexia is usually mental, as are polyphagia, polydipsia and adipsia (see Neuroses of Stomach). (*c*) *Intestinal symptoms.* Diarrhea is less common than constipation, which, like previous sexual abuses, often occupies the patients' thoughts. They "would be perfectly well if they could have one natural movement," and when they do they complain of "exhaustion during the entire day afterward." A sense of pressure, peristaltic unrest, hyperesthesia, burning and flatulency torment the patient. Enteritis membranacea and enteroptosis are not infrequent. (*d*) The amount of urine is equally often increased or decreased. The specific gravity is lowered, but the urates, uric acid and urea are increased. Phosphaturia and oxaluria occur, often attended by flatulent dyspepsia, melancholia and nervous depression. Vesical tenesmus is an occasional complaint. (*e*) *Genital symptoms.* Fear of the results of previous excesses or of self-abuse may dominate the patient's mind, but the trouble is more a matter of conception than of reality, since the sexual appetite is actually decreased as a rule. In most cases there is premature ejaculation and in some patients there is psychical impotence. Emissions are frequent, but their importance is greatly overestimated.

Course and Prognosis.—*Acute cases* are rare and fatal cases extremely exceptional. The *usual chronic course* is one of slow onset; it is attended by evidences of nervous fatigue which are at first relieved by rest, but which later persist in spite of rest and incline the patient to stimulants or drug habits. Later the signs of cerebral irritability, disturbed sleep, vasomotor disorders, reduced mental and psychical capacity, pains, spinal tenderness, paresthesia or paralgesia, sympathetic dyspnea or palpitation develop, sometimes with temporary remissions. The outlook is poor in severe hereditary forms or when there are complicating psychoses or drug habits. In other types recovery is usual, although long deferred.

Diagnosis.—The diagnosis is generally made with ease, but several rules must be borne in mind: (1) No single symptom is pathognomonic; (2) neurasthenia may precede, follow or complicate organic nervous or visceral disease, as arteriosclerosis. The *subjective* signs, irritable weakness, emotivity, muscular weakness, headache, backache, insomnia, amnesia, impaired psychical coördination and the phobias, together with *objective* findings, as asthenopia, insufficiency of the internal recti, tachycardia, epigastric pulsation, vasomotor alteration, as disturbed secretion of urine or sweat, increased reflexes, fibrillation and tender muscles cannot be mistaken. *Psychasthenia* is made by Janet to include all cases, with doubts, phobias, fixed ideas, obsessions, dazed conditions or a sense of unreality. The neurasthenic prodromes of *progressive paralysis* (*q. v.*) are usually distinguished on careful search for organic disease. *Hysteria* is typical in its stigmata and accidents; anesthesia, persistent contraction of the fields of vision, dyschromatopsia, achromatopsia, monocular diplopia, convulsions, paralyses, contractures and anuria are foreign to neurasthenia. *Hypochondriasis* differs from neurasthenia in that it is a pure psychosis, its concepts are primary, ill-based, illogical or absurd and cannot be eradicated even for a time.

Treatment.—1. Prophylaxis.—Prevention is difficult, but even with hereditary stigmata much may be done. Treatment starts with the education of both child and adult. Children are mimics and the clearest of all observers. The inculcation of self-control, fearlessness, self-sacrifice, moderation in all things, truthfulness, prompt decision and constancy of purpose must be based on actual parental example, and in this way the child should also educate the parent. The child must learn to give up, to sleep alone, to stop at the right time, to finish what he has begun, to obey and to learn that he is not the centre of the family. He should be taught without being forced. Out-door living is more important than the school-room. The English method of treating children with reserve has some advantages over American demonstrativeness. Simple, varied food, without tea, coffee or alcohol, and long sleeps develop a strong nervous system. The sexual instinct requires attention.

2. Therapy.—When once manifested, the disease should be treated as above outlined and causal factors should be investigated. Work alone is seldom injurious and while the modern complexity in the struggle for existence is increasing the number of neurasthenics, work should not bear the stigma nor should disappointment, for both make character.

Work must be systematic, not slavish. Hurry, failure to learn individual limitations, uncontrolled planning beyond the immediate day's work and worry cause the damage, especially when work produces indifference to nature, family life, exercise, rest and the things higher and above one's own small life and ambition. The time for exercise should be taken from the work hours, it should not follow them, nor should it be enforced in every case, for some are hurt by it; work under stimulation is injurious. The chief element in treatment is psychical and the physician accomplishes more than do drugs. He carefully examines and reëxamines the patient and his assurrance that no organic disease exists has its weight for the time. Conversely, careless diagnoses, as of fatty heart, work damage to the patient's mental balance. The hygiene should be regulated. Strict dietaries and stimulants should be avoided, moderate out-of-door life or exercise enjoined and in pronounced cases absolute rest from work, with sojourn near the sea or at a moderate altitude free from sudden changes or high winds is recommended. Sexual matters should be left alone unless gross violations are found. The patients need sympathy—in moderation for their sake; but the patient must be taught that he alone can cure his malady by self-control and by avoiding extremes in everything.

Pronounced cases in thin women, especially those with gastric symptoms, are often helped by the Weir Mitchell *rest-cure,* which consists of absolute rest in bed, isolation, forced feeding, massage and faradization. The patient is not allowed to lift a hand and sees only the nurse, who is selected for her strength, optimism and lack of nerves. The patient is fed milk every three hours and the muscles are rubbed and faradized. Thus weight and strength are gained without muscular degeneration. Few men will tolerate this method and obese do not fare as well as lean women. No greater mistake is made than to give all patients a rest-cure; they often want work and occupation, harmonized with rest.

3. Diet.—No one dietary is essential; some physicians favor a meat diet, others give well-cooked vegetable albumins, cooked fruit, fats, milk and eggs, especially in cardiac types. In mental anorexia, feeding by mouth or rectum must be forced. Water in abundance is indicated. *Gastric hyperesthesia* is relieved by small doses of phenol. Eructations must be controlled, since the air admitted exceeds the gas expelled; asafetida is excellent because it makes as strong an impression when swallowed as when eructed. Electricity is largely suggestive. With massage and vegetable diet, endorectal faradization relieves *constipation.* In cardiac types alcohol and tobacco are interdicted and small doses of belladonna and bromide usually control *palpitation;* other vasomotor symptoms are relieved by these remedies, ergotin or nitroglycerin, according as they are angioparetic or angiospastic; or arsenic in anemia.

Insomnia is often difficult to relieve; the following measures are valuable: avoidance of evening work, stimulants or excitement; a cool bath, the wet pack or an ice-bag over the heart; small doses of bromide with valerian for the "expectant attention" which wards off sleep; 20 grains of sulphonal in hot milk, which should be slowly sipped; codeine with hyoscine; and chloral (the continued use of which induces vasomotor

disturbance). The habit of sleep must be cultivated and the patient must not get up to read. Local, urethral and uterine affections are best ignored.

THE "TRAUMATIC NEUROSES."

This is a practical grouping of traumatic neurasthenia, hysteria, chorea and hypochondriasis. Hysterical forms were first described by Walton and Putnam; Erichsen (1866) wrote on trauma to the spine, on which the illogical name of railway spine was later conferred, and Oppenheim (1877) termed the group the "traumatic neuroses." All organic affections must be excluded, whether trauma seems directly or indirectly causal, as vertebral caries, tumor, chronic ankylosis, rarefying osteitis which Kocher refers to fracture; hematorrhachis, meningeal hematoma or pachymeningitis; and cord affections, as poliomyelitis, myelitis or lateral sclerosis. Experimental trauma may produce molecular nervous changes, and trauma may hasten arteriosclerosis. Alcoholism and syphilis are predisposing factors. *Traumatic neuroses are purely functional affections caused by trauma,* which operates psychically rather than somatically.

Symptoms.—The symptoms are those of hysteria, neurasthenia and hypochondriasis, singly or in combination. The patient may feel well for a day or two and then pain develops at the seat of injury or in the spine; anxiety, fear of his financial future or for his family, a "fixed idea" of his incapacity and the development of "litigation symptoms" are typical of hypochondriacal neurasthenia. *Psychical symptoms* are usually prominent; they are accentuated by repeated, sometimes suggestive, physical examinations, visits of lawyers or adjustors, court-room suspense, reversals of judgment or appeals. Vertigo, backache, headache, sleepiness by day and nocturnal insomnia may cause great distress. *Pain,* which seems to be nervous, may prove organic on examination by the *x*-rays; it may be simulated, but is often real, as shown by persistence after award of damages. Tender areas are difficult to pass upon, because of exaggeration or simulation; Mann, Köpf and Rumpf found that pressure on areas which are actually tender increases the pulse-rate by 10 to 30 beats; this sign is subject to several conditions: (*a*) it is not always present; (*b*) its absence is no proof of simulation; (*c*) in simulation, the writhing which occurs when certain areas are touched may increase the heart's action. Paresthesia and hyperesthesia of the skin or special senses are frequent. Anesthesia indicates traumatic hysteria. Hysteric or neurasthenic contraction of the visual fields may remain the same from day to day and not vary with approach or withdrawal of the test object; this is not a sign of simulation; Förster's type cannot be assumed. The pupils may vary, especially by dull illumination; reflex immobility indicates organic disease. *Motor symptoms* may embrace all those of hysteria or neurasthenia, abasia, astasia, paralyses, contractures suggesting vertebral injury or joint disease (Brodie's joints), pseudospastic or -tabetic gaits, tremor (*forme trepidante*) or sometimes muscular atrophy about the joints. Hysterical convulsions are rare; the author twice saw choreiform affections in telephone girls who sustained slight shocks

and once a genuine chorea. Patients must often be watched; one of the author's cases, with spastic gait, shuffled out of the office but walked perfectly well around the corner. The *reflexes* are often increased, perhaps asymmetrically; ankle-clonus, which is probably spurious, is sometimes reported. When there is albuminuria and glycosuria the question arises whether they were previously present.

Diagnosis.—Several points are often difficult to establish, because objective findings are frequently few: (*a*) It is usually possible to state whether one of the neuroses is present or whether organic disease obtains. (*b*) Exaggeration and simulation are difficult to estimate, as shown by the change of professional opinion from the idea that most cases are simulated to the present statement of some writers that it is present in 4 per cent. of cases only. (*c*) If disease is present is it directly due to injury? (*d*) If so, the most difficult of all questions is presented, namely, the degree and duration of the disease and the damages to be awarded.

Prognosis.—(*a*) *As to life,* the outlook is usually good, though some cases become insane or suicidal. (*b*) *As to complete recovery,* it is less favorable. In most cases it is surprising how soon symptoms disappear after satisfactory legal settlement is made; this is said without cynicism. Many persons do not then recover completely and symptoms may endure in those who reject litigation. Hysterical are more favorable than neurasthenic or hypochondriacal symptoms.

Treatment.—Treatment is psychical. The physician dreads the effect of litigation on the patient, assures him that there is no organic lesion and advises early settlement and return to work.

EPILEPSY.

Definition.—Epilepsy, by derivation, "being seized upon," has been called the "falling sickness" and morbus sacer. It does not include Jacksonian or symptomatic epilepsies. Classified with hysteria and neurasthenia as one of the "three great diffuse neuroses," it probably includes more than one disease. It is a *disturbance of balance between the central exciting and inhibiting nervous processes* attended by (*a*) a typical *chronic, convulsive disorder,* affecting the entire central nervous system, each attack being followed by *loss of consciousness,* (*b*) *partial or atypical manifestations* of these characteristics, or (*c*) concomitant or consecutive psychical or other symptoms. It occurs in from one to six persons per thousand.

Etiology.—Of the *ultimate causes,* the *neuro-* or *psychopathic tendency* of Griesinger is found in 37 per cent. of cases; only the tendency is transmitted by inheritance, rarely the disease itself. Neurotic or psychopathic antecedents, parental alcoholism (51 per cent.), morphinism, plumbism, syphilis, tuberculosis, gout, diabetes, or ovarian and testicular diseases lessen the nervous resistance of the offspring. The disease rarely develops after the thirtieth year; 75 per cent. of cases develop before twenty; 46 per cent. between ten and twenty, mostly at puberty; 28 per cent. before ten. The sexes are equally affected.

Inciting causes include *infections*, as malaria or typhoid; syphilis may be hereditary, induce the Jacksonian type from cortical gumma, or it may be parasyphilitic; *intoxications*, especially alcoholism; *exhausting affections*, as diseases of the blood or metabolism; and *trauma* or fright. Inciting causes are found in 37 per cent. of cases.

Reflex epilepsy is said to occur from painful scars, nerve tumors, or foreign bodies in the ear, nose, nasopharynx or larynx; diseases of the digestive tract, uterus and heart; onset of the menses or their monthly appearance; pregnancy, phimosis or onanism; from gall-stones, colic or abdominal paracentesis.

Pathology.—There is no characteristic macro- or microscopic change. The usual finding after death is that of any convulsion, viz., venous engorgement and punctate ecchymoses. Induration of the cornu ammonis, induration in the medulla, neurogliar increase in the cortex, thickening, asymmetry or deformity of the skull and persistence of the thymus have not been proved causal.

Symptoms.—The symptoms are best studied from the *completely developed attack*, in which there are three component parts: (*a*) The *prodromal stage* is not always present. Its "warnings" are either distant or immediate. The distant warnings (in 10 per cent. of cases) may precede the seizure by hours or days and take the form of restlessness, dispositional changes, depression, thoracic or cephalic oppression, insomnia, vertigo, nausea, grinding of the teeth, hyperesthesia of the special senses, angioneurotic palpitation or cerebral congestion. The immediate warning, the *aura* proper, is the beginning of the convulsion rather than a warning. It is a central cortical irritation; its frequency is 40 per cent.; it is rarer in sudden, stormy, generalized seizures. The psychical aura is rare and is manifested by emotional or intellectual alteration and anxiety. The sensory aura consists of paresthesia and a sense of deadness in a limb or joint, attacks of migraine and epigastric sensations (from the pneumogastric nerve, pressure or irritation of which sometimes inhibits the convulsion). The special senses may be irritated or blunted; there may be flashes of light or colors, hallucinations, blackness before the eyes and less often anomalies of hearing, taste or smell. The motor auræ are circumscribed clonic or much less often tonic twitchings, as in Jacksonian epilepsy, from which they are distinguished by a rapid loss of consciousness; automatic movements, like stamping or buttoning the coat, are rarer and motor weakness and singultus, coughing or sneezing are least frequent. Vasomotor auræ may be localized or generalized, as rushing of blood to the head, palpitation, anesthesia, increased temperature, increased arterial tension, pallor or chilling. The patient then falls, *pale and unconscious*, or is thrown with force, often on his face, when (*b*) the *convulsive stage* begins. First a *tonic* convulsion affects all voluntary muscles, due to infracortical irritation. The initial *cry*, guttural or groaning from tonic abdominal, thoracic and laryngeal spasm, is heard in 50 per cent. of cases The head is held back; the eyes are open and staring, the jaw set, the face flushed, the neck tense, the body often opisthotonic, breathing and pulse are suspended, the muscles are hard but slightly vibrating to the palpating hand, the arms are turned tetanic-

ally inward, the hands are clenched with the thumb adducted and under the fingers, the legs are extended, the thighs adducted and the toes flexed or extended and spread apart. In some cases the head, eyes and even trunk deviate and in a few instances the tetanic spasm spreads gradually, instead of the usual instantaneous tonic spasm. The tonic phase lasts ten to thirty seconds, relaxing in inverse order from the limbs to the neck and face. The second phase of *clonic convulsion* occurs at once, appearing first in the limbs, which are not yet free of the tonic spasm. This stage is bilateral, highly irregular and violent, with very short remissions, leading perhaps to injury, fracture, luxation, breaking of teeth, laceration of skin, tongue and cheek, or rupture of muscles. It consists largely of alternating flexion and extension; the head strikes the ground, the body turns violently, the eyes jerk and protrude, the face is distorted, the tongue is bitten as it is thrust out, bloody saliva is expressed by the masseters, inarticulate sounds arise from the movements of the diaphragm and respiratory muscles and gurgling results from intestinal movements; vomiting movements, filling of the stomach with air, cyanosis, swelling of the jugular veins, ecchymoses in the conjunctiva, skin, retina or larynx, involuntary and sometimes forcible evacuation of the bladder, less often of the rectum, and occasionally ejaculation, occur. The clonic phase lasts one-half to three minutes, ending in a fine generalized tremor. (*c*) The *comatose* stage often begins with a deep sigh; respiration and circulation become normal, the limbs relax and the condition resembles a sound sleep, from which the patient awakes in from fifteen minutes to several hours, sometimes suddenly, more often gradually and in a more or less dazed condition. He recalls nothing of the attack and the loss of memory may extend back of the seizure (retrograde amnesia). He experiences pain in the muscles, joints or head, nausea, mental irritability or depression and finally a group of *exhaustion symptoms*, viz., (i) sensory exhaustion, expressed by hypesthesia, anesthesia, hypalgesia or analgesia; (ii) exhaustion of the special senses, as concentric limitation of the visual and color fields, dulling of hearing, taste or smell, and (iii) motor exhaustion due to cortical fatigue.

During the attack *vasomotor* constriction often occurs, as shown by the initial pallor of the face, cardiac overaction or perhaps irregularity. The ecchymoses and retinal congestion are due to the convulsion. The *pupil* is reactionless, narrow at the onset, and dilated widely in the second stage; afterward it may oscillate; the pupils are sometimes unequal. The tendon and skin *reflexes* are weak or lost in complete attacks and for hours afterward, whence we may judge of the severity of the seizures. The temperature may be elevated 2° or 3°. Voisin found albumin in 50 per cent., though others consider it infrequent. The author has often noted transient albuminuria and cylindruria (granular casts) after marked paroxysms; glycosuria is uncommon. The phosphates and nitrogen are increased; polyuria is common in the third stage. The sweat is increased.

Types of Epilepsy.—1. The completely developed attack (*epilepsia gravior, grand mal*) consists of (*a*) the classic type (29 per cent.), as above described, with loss of consciousness, and tonic followed by clonic con-

vulsions; or (*b*) the slightly atypical type (19 per cent.), in which, *e. g.*, the spasm may not always be generalized.

2. *The rudimentary attack* (21 per cent.) consists of loss of consciousness, plus tonic or clonic spasms. This includes the apoplectiform cases, especially the senile (arteriosclerotic) form; forms resembling myoclonus; cases which do not fall but stumble along at the onset (epilepsia procursiva) or turn (epilepsia rotatoria).

3. The third form is the *abortive attack* (epilepsia mitior, *petit mal*, 31 per cent.). Its importance is underestimated by the profession and the public; two main types, with endless variations, are distinguished: (*a*) one with loss of consciousness with few or no motor signs, and (*b*) motor and vasomotor signs with no loss of consciousness, or very slight clouding of the intellect. The loss of consciousness is often very slight or may last for a few seconds, so that a scarcely perceptible break occurs in the patient's occupation, reading, speaking, playing the piano or a game of cards; it is described by the French as "*absence.*" He may move his lips inarticulately or there is nystagmus, a chewing or swallowing movement, tremor or twitching of the face, monospasm or vertigo. A cry may be the sole symptom. Some patients seem to fall suddenly asleep. Nothing, or everything, may be remembered. Auræ may constitute the whole attack, which aborts without unconsciousness or spasms.

4. The *psychical equivalent* of Sammt, also known as transformed or larvated epilepsy or the epileptoid condition is often mistaken. Mental symptoms may precede, succeed, alternate with or replace the ordinary convulsive type, which must be first recognized to establish this variety. The mental changes are slight (*petit mal intellectuel*) or marked (*grand mal intellectuel*) and include excitement, emotional outbursts, narcolepsy, incoherent conceptions, amnestic attacks and mania. The patient may act like a hypnotized subject; he may take sudden long journeys or commit assault, arson or murder, whence the great medicolegal significance of this variety.

The Interparoxysmal State.—Signs of psychical degeneration, poorly differentiated or large, small or prominent ears, hematoma of the ear, deformed teeth, lips or palate, coloboma, strabismus, nystagmus, small eyes, irregular pigmentation of the iris, deformed genitals or fingers, disorders of speech or irregular facial innervation may indicate hereditary taint. Neurasthenic symptoms are not unusual. The expression is often dull. When idiocy occurs with epilepsy, both are coördinate results of a common cause, or early and repeated attacks have initiated mental degeneration. However, many brilliant men have been epileptics, as Julius Cæsar, Napoleon, Carl V, Peter the Great, St. Paul, Mohamed, Petrarch and Rousseau. The importance of organic disease of the heart, lungs, stomach and kidney is difficult to estimate. Epileptics often sweat profusely, are usually constipated and frequently become obese from inordinate eating and the bromide treatment.

Diagnosis.—The diagnosis concerns the convulsive seizures and the abortive types. Similar convulsions must first be excluded which occur in (*a*) *organic brain disease*, as syphilis, tumors, vascular lesions, paretic dementia, multiple sclerosis and infantile cerebral paralysis. These

affections may produce generalized or Jacksonian fits. *Jacksonian epilepsy* is more clonic (cortical irritation) than tonic (infracortical irritation), is often associated with paresis or sensory alteration, occurs less often with coma and in 50 per cent. of cases is due to small cortical tumors. Epilepsy rarely develops after thirty years of age (E. tarda) and 90 per cent. of cases which occur after thirty—excluding alcoholism and uremia—are syphilitic. After forty-five, vascular lesions are a very common cause. (*b*) Convulsions may occur in alcoholism, *uremia* (eclampsia), plumbism or drug poisonings. (*c*) *Reflex epilepsy* (*v. s.*).

Simulation of epilepsy is impossible. In the complete type, the tongue biting, cry, stertor or involuntaries may be absent. Scars are often of diagnostic value. Nocturnal epilepsy occurs in 33 per cent., often coming on when the patient drops off to sleep or on awakening in the morning; the depression, bitten tongue, ecchymoses and involuntary evacuations are unmistakable.

Differentiation from hysteria is usually easy:

Epilepsy.	Hysteria.
Distant warnings: few, mental.	Frequent, emotional.
Aura: more frequent, momentary, oftenest unilateral or epigastric.	Less frequent, longer, bilateral, foot aura, globus.
Cause: none.	Emotional.
Onset: always sudden, often with pallor, cry which is never verbal, immediate falling and complete unconsciousness, with rigidity, etc.	More gradual, rarely with pallor or cry, which may be repeated or verbal, and recur. Rarely brusque falling, slower and less complete unconsciousness, and may react to suggestion.
Tongue biting, frothing: very common.	Exceptional, or seemingly purposive.
Convulsions: generalized, tonic followed by clonic spasms, very rarely tonic alone; flexor spasm predominates.	Rigidity, to carry out semi-intentional movement; struggling; clonic, irregular spasms; extensor spasm predominant, "*arc de cercle*," clownism.
Reflexes: pupils contracted at onset, dilated later, immobile (in clonic period). Tendon and skin reflexes absent during and after attack.	Pupils usually wide and react (may be immobile, Westphal, Karplus). Sometimes convergent strabismus. Reflexes rarely absent; or increased.
Involuntary evacuations: frequent.	Absent or most exceptional.
The urine: solids always increased, both nitrogen and phosphates.	Solids decreased; altered ratio of earthy and alkaline phosphates.
Temperature: sometimes elevated, 2°–3°.	Practically never.
Coma: complete.	No real coma.
Duration of seizure: very short.	Longer, recurrent, relapsing.
Postparoxysmal: sensory and motor exhaustion, ecchymoses, absent reflexes, mental incapacity, with no memory of transpired events.	Depression, fatigue, pain in the head or stomach, nausea, some memory of attack.

Most difficult to differentiate are *petit mal* and transformed epilepsy, in which the history of major attacks is important. *Petit mal* may be confused with cardiac syncope, labyrinthine vertigo, etc.

Course and Prognosis.—When one attack has occurred a second may be expected in 33 per cent. of cases in less than one month, in 33 per cent. in from one to twelve months, in 33 per cent. in over one year; and when epilepsy is once established, daily attacks occur in 11 per cent., in 50 per cent. they occur at intervals of less than two weeks, and in 75 per cent. they occur at intervals of less than one month (Gowers).

The mode of life, stimulation, excitement, pregnancy and sexual excesses may increase their frequency and cessation during fevers is not uncommon. In some, fortunately rare, cases the attacks occur in quick succession (*status epilepticus, état de mal*), as many as 10 or over 100 occurring in one day. In one case 2500 seizures occurred during a month; the stage of coma may last from two or three days to a week, with a temperature of 105–7° and death then results. The *outlook* (*a*) *as to recovery* is poor. Five to 10 per cent. of cases may recover spontaneously. The prospects are best in cases which begin before twenty in males, in cases with long intervals between attacks and in hereditary forms. After two years' duration the outlook is unfavorable. (*b*) *As to life,* the average duration of life is shorter than normal. A few cases drown, suffocate from turning on the face or are fatally burned during the seizure. Some patients die during the insult from vascular lesions of the brain or less often from heart rupture, respiratory cramp, edema of the lungs or vomiting into the larynx. One-sixth of epileptic deaths are sudden. Epileptics frequently become tuberculous. In 10 per cent. dementia occurs. Bodily health and intellectual vigor may be remarkably preserved.

Treatment.—1. Causal.—Syphilitic epilepsy, unless due to gummata or secondary anemia, does not respond to antisyphilitic remedies. Avoidance of alcohol may be almost curative in this form. Reflex factors should be removed. In Sweden epileptics cannot marry; 50 per cent. of hereditary epileptics transmit some transformation of the taint to their offspring.

2. Hygienic.—Children should receive the general care outlined under the other neuroses. School work should be limited and country is preferable to city life. For poor subjects, institutional treatment is indicated. Many neurologists restrict the consumption of meat. Richet withdraws salt and gives a milk and cereal diet, which promote the affinity of the bromides for the brain cells.

3. Bromides.—Introduced by Laycock (1853) and developed by Voisin, the bromides act directly on the nervous tissue; they depress the cortical cells, inhibit reflex action and lessen centripetal conduction. *Untoward symptoms may result even within therapeutic limits.* Intellection may be dulled and muscular depression occur, but these may often be counteracted by coffee; the frequent stomach symptoms can be avoided by free dilution with water; diarrhea, which results especially from potassium bromide, is relieved by opium if marked, or if less, by salicylate of bismuth, gr. x, after meals; in its elimination by the skin, acne is common, which is lessened by large quantities of water, small doses of arsenic and washing the skin with green soap. Belladonna in small doses modifies the respiratory catarrh or salivation. The anaphrodisiac effects cannot be prevented. In some individuals there are *marked idiosyncrasies,* as mental excitement, hallucinations, emaciation ("bromide cachexia") with lessened physiological resistance to acute diseases. *Toxic symptoms* may be acute, but more commonly are chronic, resembling those of paretic dementia; *bromism* is most likely to develop in those with weak digestion or circulation and when the drug is given carelessly for a long time, because its elimination is slow. In the former, hydrotherapy,

massage and tonics should be used as preliminary or concomitant measures. Cardiac symptoms are due almost wholly to the potassium salt, but especial care in the use of all bromides is indicated in senile epilepsy (myocarditis, arteriosclerosis, endocarditis) or great nutritive exhaustion, in which combination with digitalis or strychnine is indicated. In bromism the reflexes are decreased; the pharyngeal reflex is weak in 17 per cent. and absent in 25 per cent. of normal individuals, whence Ziehen watches the corneal reflex, which is never normally absent. In advanced bromism there are low temperature, bronzed skin, vasomotor relaxation, somnolence, paralytic speech, muscular relaxation, ataxia, tremor, fetor ex ore, anorexia, slow respiration (the usual cause of death), weak heart, abolition of sensation and of all reflexes and decrease or suppression of urine. Therapeutically the drug unites with the nerve cell and depresses its activity and metabolism and toxicologically it causes its degeneration or atrophy. The drug must be used for months or years and the symptoms above enumerated must be watched for; epileptics have a tolerance for bromides which Voisin regards almost as a food in this affection.

Results.—Seguin's results were: cessation of attacks in 23 per cent., notable decrease in 40 per cent., no great effect in 26 per cent., no effect in 10 per cent. Gowers noted immediate relief in 43 per cent., improvement in a short time in 47 per cent. and no effect in 10 per cent. He gives a dram a day of the potassium salt, for two years, for larger doses are no more efficacious; in nocturnal epilepsy a large dose should be given at bedtime. Charcot gave a daily dose of one dram, which was gradually increased to two, and then slowly decreased to one. Erlenmeyer's formula gives excellent results (sodium and potassium bromide, each two parts and ammon. bromide one part). The best results are obtained in cases treated early in their course and early in life and in those in which there are long intervals between attacks. Children tolerate large doses. Opium, belladonna, digitalis, thyroid extract and chloral are of but secondary importance. Nitroglycerin may be useful in *petit mal.*

4. The Attack.—This is confined to cases in which auræ are present. The patient is placed on the bed or ground to avoid injury and a towel inserted between the teeth to prevent biting of the tongue. Compression or manipulation of the limb in which the aura is felt may in a few instances avert the spasm. Charcot placed an ice-bag over the head and one over the heart. Inhalations of amyl nitrite, ether or chloroform are sometimes beneficial.

5. Surgical Indications.—Biswanger's conclusions are (*a*) that only a small percentage of traumatic cases recover; operation is indicated (*b*) when convulsions emanate from a small cortical focus or (*c*) are of a type intermediate between the Jacksonian and generalized forms; (*d*) when, with generalized convulsions, there is a suggestive scar, bone defect or localized tenderness; (*e*) the site of trephining should be governed by focal symptoms, motor or sensory auræ, or "exhaustion" signs; (*f*) cysts, tumors, bone, membrane or scars may be removed, but not the cortex; (*g*) operation must be followed by the bromide treatment; (*h*) operation is contra-indicated in ordinary epilepsy.

INFANTILE CONVULSIONS.

In young children many axis-cylinders are not yet clothed with myelin sheaths and the lower dominate the upper segments, thus predisposing to disproportionate nervous reaction.

Etiology.—(*a*) *Rickets* as a cause was especially emphasized by Wm. Jenner. Many cases of carpopedal spasm, tetany and laryngismus stridulus have a rhachitic basis; spasmophilia occurs between the eighth and twenty-fourth months of life, is prone to recur and disappears after the second year. (*b*) *Reflex spasms* may be caused by peripheral irritation, as from dentition and worms, though this type has been greatly overestimated; gastro-intestinal irritation or auto-intoxication, phimosis and otitis media are possible causes. (*c*) *Gastro-intestinal intoxication* results in debility, malnutrition or a "hydrencephaloid" condition described above under various captions and usually of most serious import. (*d*) *Nervous affections,* trauma during delivery, meningitis, tumor, sinus thrombosis, severe coughing, acute encephalitis (cerebral hemiplegia) and acute poliomyelitis are etiological factors. (*e*) In *acute infections,* as scarlatina, measles or pneumonia, convulsions and vomiting often replace the chill which is more common in adults. (*f*) Genuine *epilepsy* in 12 per cent., according to Gowers, occurs in the first three years of life.

Symptoms.—The symptoms resemble those of epilepsy in most of its phases; the eclampsia of children usually begins in the hands and the seizure is partial, tonic, intermittent and carpopedal rather than generalized. Screaming and grinding of the teeth may occur. Laryngeal spasm is described under Affections of the Vagus.

Treatment.—The occurrence of the *convulsion* is the first indication for treatment and should be met symptomatically by inhalations of chloroform or nitrite of amyl, followed by gr. v–x of chloral and ʒss of bromide by rectum. The bromide is given for several days, for its effects are cumulative rather than immediate. Opiates should generally be avoided in children under five years, especially in nurslings, weak or marantic infants; the hot bath is of little service.

The *cause is then determined* and treated, as rickets, tetany, peripheral irritation; lancing of the gums is practised less and less; in gastro-intestinal intoxication the stomach should be emptied by ipecac or the stomach-tube; lavage of the bowel is indicated in acute cases and stimulants may be indicated.

CHOREA AND CHOREIFORM AFFECTIONS.

Chorea or St. Vitus's dance, known to Paracelsus, and endemic in the middle ages is now known as chorea major; it was hysteria. Chorea in its usually accepted sense was described by Sydenham and is known as Sydenham's chorea or chorea minor.

Definition.—Sydenham's chorea is (*a*) a neurosis of late childhood; (*b*) it is characterized by subacute onset, limited duration and, usually, recovery; (*c*) it is probably cortical in localization, as indicated (*d*)

by involuntary irregular spasmodic movements, and (*e*) increased incoördination on voluntary efforts; (*f*) it is always associated with some psychical weakness or irritability and (*g*) is frequently associated with rheumatism and endocarditis.

Etiology.—(*a*) *Age:* 80 per cent. of cases occur between the fifth and fifteenth years; 90 per cent. between the fifth and twentieth years; it is most frequent at the sixth and seventh years of life or at puberty. (*b*) *Sex:* Seventy per cent. of cases are in girls. (*c*) *Rheumatism* has been considered a cause since Bright's time, especially by English and French writers. Rheumatism is a factor in at least 20 to 25 per cent. (Hughes and Sée); *endocarditis, rheumatism* and *chorea* are too frequently associated to escape serious attention; they are probably correlated, *i. e.*, due to a common virus. Rheumatism occurs usually before the chorea; in but 4 per cent. of cases do the two occur simultaneously. (*d*) *Other acute infections* are causes in 43 per cent. of cases (Neumann), *e. g.*, gonorrhea, puerperal fever, sepsis, pertussis, measles, typhoid, etc. (*e*) *Pregnancy* is a predisposing cause, particularly before the twenty-fifth year. Chorea occurs in the first three months, especially in first pregnancies, and is most severe in character. It rarely occurs postpartum. Giles de la Tourette justly holds that many cases classified as chorea are really hysteria. (*f*) *Neuropathic heredity* is observed in 16 per cent. of choreic subjects, particularly among Hebrews. (*g*) Other causes are sometimes operative, as fright, trauma and reflex irritation from nasal or digestive disease. It occurs more largely in the lower classes, is sometimes due to iodoform or mercurial poisoning and is influenced by the poor hygiene, excitement, exhibition and competition of the school-room, the "school-made chorea" of Sturges.

Pathology and Pathogenesis.—The disease is a provisional neurosis, since no constant pathological findings exist. The following changes have been recorded: hyperemia, exudation about the arteries, areas of softening, punctate hemorrhages, round hyaline bodies in the perivascular lymph sheaths and swelling of the large pyramidal cells of the cortex; none of these are characteristic or constant. In fatal cases endocarditis is the most frequent finding (90 per cent.) and Osler notes that its frequency is greater even than in rheumatism. Kirke's theory of cerebral embolism has been abandoned. The bacteriological reports are conflicting. The irritation, due to exhaustion or toxemia, is probably cortical or in the basal ganglia.

Symptoms.—1. Onset.—Prodromes are infrequent, as irritability, restlessness, depression, disturbed sleep, headache, anorexia, rheumatic pains, or sensory irritation. The affection usually begins with motor disturbance in the form of *involuntary movements* and *incoördinate voluntary movements;* the latter are often misunderstood and punished, for the child cannot sit still or breaks or drops things.

2. Chorea Movements.—These are *involuntary,* although they may seem voluntary, as the thrusting out of the tongue or the reaching of the arm at an object. They are "*irregular in time, character* and *degree,*" are not usually attended by fatigue or pain, are incomplete and without motive, are increased by attention being drawn to them and by

excitement and cease during sleep. They usually begin in the arms and later extend to the face and legs. Chorea very often begins on one side, especially the right, whence it extends to the other side, but sometimes remains largely, though never entirely unilateral (*hemichorea*). The face is contorted with varying expressions, now grimaces or again motiveless smiles, the *facies choreatica* of Sachs. The eyes roll, and diplopia is not uncommon. The pupils are often wide and react slowly. The author recently saw a case in which many of the teeth were broken. Speech is involved in 25 per cent. of cases, due rather to difficult articulation by the tongue and lips than to phonation. Speech is quicker than normal and may be aggravated by irregular movements of the glottis and respiratory muscles, resulting in panting, irregular inspiration or expiration or sobbing sounds in which the diaphragm participates. Aphasia is less frequent. The *arms are most involved;* they are flexed, extended, pronated and supinated in tireless succession—Bouillaud's *folie musculaire.* The *trunk* is sometimes involved in light cases and distinctly so in severe types. The violent movements may throw the patient out of bed. The *legs* are less involved; there is a zig-zag gait and walking may be impossible. Besides hemichorea, other irregularities are encountered, as monochorea, chorea paraplegica or chorea alternans. The *muscular power* and endurance are said to be normal (Wallenberg), but Gowers and Osler speak of marked muscular weakness. Two forms are distinguished: (*a*) paresis with chorea, in which the muscular power is reduced, as in the usual case, and (*b*) limp or paralytic chorea (*chorée molle*). The paresis may be of the mono-, para- or hemiplegic type.

3. Psychic changes are constant and the irritability and ready fatigue resemble an acute neurasthenia. The subject is wilful, forgetful, emotional, irritable, unable to concentrate the attention or to remember perfectly. Marked alteration may occur, as complications, viz., delirium, mania, melancholia or hallucinations; *chorea insaniens* has a most unfavorable prognosis. Combination with hysteria is frequent, with epilepsy infrequent. Hughlings Jackson found paroxysmal headache in 90 per cent. of cases.

4. Sensation.—Sensation is normal or slightly blunted. Tenderness over the spine and peripheral nerves is not common, though S. Weir Mitchell and Triboulet described a painful chorea. Occasionally there is a tingling sensation and numbness.

5. The reflexes are normal in light cases but may be decreased or absent. The bladder and rectum are normal save in the severest forms.

6. Electrical Reactions.—The electrical reaction and mechanical irritability of the muscles are with few exceptions unaltered.

7. The Heart.—Irregularity is common and is often due to irregularity in breathing (pseudo-arrhythmia). A slight increase in rate is due to the general neurosis. Ollivier, Roger and Simon claim that chorea may affect the "muscles of organic life", but a cardiac chorea is improbable. *Functional murmurs* are frequent, especially in thin, nervous children, from anemia, toxemia and tachycardia. Great conservatism must be exercised in their prognosis and diagnosis (see page 358).

Endocarditis has been mentioned in the definition of the disease and its causation and pathology (90 per cent. of fatal cases). It was found in 66 per cent. of Osler's cases without rheumatism. It is rarely malignant, but may produce embolism. Mitral lesions, especially insufficiency, are the most common form. The importance of endocarditis without clinical symptoms is vast. Pericarditis is not frequent. Heart disease follows 50 per cent. of cases having two attacks of chorea, and 100 per cent. of those having over three.

8. The Skin.—Herpes and pigmentation result from arsenic. Urticaria, erythema nodosum, purpura and rheumatic nodes (*v.* page 284) are sometimes observed.

9. General Symptoms.—Emaciation occurs chiefly in severer types. The urea and phosphates are often increased. Albuminuria is rare, except as a result of renal embolism. The temperature is not often elevated, save from cardiac and other complications; even in chorea insaniens it rarely passes 102°.

Course and Prognosis.—The average course of mild cases is from six to ten weeks, and six weeks to six months is the average duration of all varieties. In very rare instances the disease may last years. Three main types are distinguished: (*a*) The *mild type,* in which the patient is nervous, irritable, has but slight choreic movements and is often dyspeptic and sometimes anemic; (*b*) the *severe form* in which the spasmodic movements are severe and general; (*c*) the *maniacal type* (*chorea insaniens*), which is most often observed in adult or pregnant women, and sometimes at puberty. The movements are incessant, extreme and often associated with fever, constant insomnia and great psychical disturbance. As a rule, the shorter the duration the more intense are the symptoms. Sydenham noted the tendency to *recurrence* (in 25 to 33 per cent.). One year is the average interval and when there is no recurrence within one and one-half years, relapse is improbable (Gowers). *Recovery* is the rule; the mortality averages between 2 and 3 per cent. Twenty to 25 per cent. of pregnant cases die. Fatality is greatest at the age of puberty and in adults. As Charcot remarked, death occurs less often from complications, as endocarditis, than from toxemia, fever, emaciation, delirium and coma. Fever or obstinate insomnia is ominous. Though the immediate outlook is not influenced by endocarditis, it is a most important feature in the ultimate prognosis of a complete recovery. *Intercurrent fevers* may dissipate the spasms. In rare instances distinct psychical changes (Jolly) or permanent chorea remain (*v. s.*).

Diagnosis.—Recognition of the disease is usually most easy when one bears in mind the conception (definition) of the disease.

Differentiation.—Simple tremor or the intention tremor of multiple sclerosis is easily separable. The hemichorea of hemiplegia (see page 722) or the bilateral chorea of *infantile cerebral paralysis* (*q. v.*) is distinguished by the history and course. Friedreich's ataxia is differentiated by its slow, irregular, incoördinate movements, nystagmus, its frequent family incidence, the scoliosis, talipes, and slow, scanning speech. *Hysterical chorea* (so-called) is rhythmic, more regular and frequently produces sudden ("electrical") movements, as bowing or salaaming, nodding the

head, etc. *Chorea major* in the original sense is of historical interest; it was a pandemic or epidemic *hysteria* provoked by religious excitement. Its victims made pilgrimages to the shrine of St. Vitus. Similar outbreaks occurred in Kentucky among the early settlers. This form closely resembles the saltatoric spasm described by Bamberger, in 1859, in which the legs contract on attempting to stand, causing jumping or springing movements. It may be transitory or may last for years. Beard and Thornton described a similar neurosis endemic in Canada among the "jumping Frenchmen." The "holy rollers," and jumpers of Russia and Java come under this head or under impulsive tic (*v. i.*).

Huntingdon's chorea has no connection with Sydenham's type. Chronic chorea comprises two forms: (*a*) The *senile form*, in which the mind is less involved, the course more benign than in (*b*) the *hereditary form* of Huntingdon who, in 1872, brought out the cardinal features of the disease as observed on Long Island, viz., (i) the late and gradual onset after the thirtieth year, especially in males; (ii) its hereditary character, sometimes involving 25 or 50 per cent. of the family, sometimes skipping generations or lasting for generations or even for two centuries in a family (Osborn); (iii) its progressive character, and (iv) the ultimate slow dementia, often with epileptiform attacks or suicidal tendencies. The precise *pathology* of Huntingdon's type is not known. Pachymeningitis, hematoma of the dura, or less often chronic encephalomeningitis, vascular disease and atrophy of the convolutions are present in the isolated autopsies. The *etiology* is unknown. *Clinically* it is characterized by irregular movements, which usually begin in the hands, are somewhat controlled by voluntary effort and are more irregular, incoördinate and slow than in acute chorea. Slow, spasmodic facial contortions are observed, and the speech becomes first slow and hesitating, and later quite indistinct. The gait is unsteady and swaying. Writing is interfered with, the reflexes are moderately increased, sensation is normal, save that the muscular sense is somewhat blunted, and dementia develops. The disease is incurable.

Tic.—The term is now used to designate an "habitual, conscious, convulsive movement resulting in the involuntary contraction of one or more muscles, abruptly reproducing some reflex or automatic action of every-day life" (G. Guinon). There are three forms: (*a*) In *simple tic*, (i) localized spasm, *habit spasm* or habit chorea, which is said by S. Weir Mitchell to occur in girls between seven and fourteen years of age, may result from lesions of the nose or throat or possibly from eye-strain, and includes movements such as winking, drawing of the mouth, sniffing, shaking the head and shrugging the shoulders. It usually ceases, but may last into adult life; the child's attention should not be drawn to the movements, although the writer believes that judicious reminders and discipline inhibit these habits. Arsenic may be beneficial. (ii) The *electric chorea* (Henoch and Bergeron) consists of short, lightning-like contractions in special muscles, as those of the neck or shoulders, as though the muscles were electrically stimulated. (iii) *Dubini's disease* (1846) consists of contractions of the arms and legs, followed by hyperesthesia, pain in the head and back, muscular atrophy,

paresis and sometimes fever and convulsions. It is endemic in Lombardy, its cause is unknown, and it is usually fatal from apoplexy or coma. (iv) *Paramyoclonus multiplex*, or *myoclonia*, described by Friedreich, in 1881, is an affection of sudden onset, with symmetrical, involuntary, arrhythmic, quick, clonic contractions of the upper parts of the arms, legs and trunk, sometimes of the face and tongue, and occurs without loss of consciousness. The contractions number from 10 to 150 a minute and may terminate in a tetanic contraction. It affects male adults chiefly (75 per cent.) and follows mental or emotional agitation, so that some would classify it under hysteria. The myotatic irritability and reflexes are increased, the mind is usually normal, though sometimes fixed ideas are observed, hiccough is occasional from involvement of the glottis or diaphragm, voluntary movement is decreased or sometimes arrested, there are no sensory symptoms and the outlook is usually good. Treatment consists of administration of voltaic electricity, nervines and bromides. The family form (Unverricht) with epilepsy and spasms of the tongue and pharynx, is probably a subtype of hereditary chorea.

Tic	and	Chorea.
Form of movement: pseudo-intentional, with repetition.		Incoördination and great variability.
Rhythm: rhythmic, brusque, short, synchronistic.		Arrhythmic, slow, not synchronistic.
Will: suspends movements.		No effect.
Accessory signs: muscular power preserved; normal tendon reflexes.		Myasthenia; modified reflexes and sensation.
Etiology: hereditary degeneration.		Neuropathic tendency.

(*b*) *Tic with explosive utterances*, impulsive tic or Giles de la Tourette's disease, is closely affiliated with hysteria and monomania. It occurs in neurotic individuals between the sixth year and puberty. The *cardinal features* of the psychosis are: (i) Involuntary movements in the face and arms or in all the muscles in severe types, when the movements become violent or explosive. (ii) Explosive utterances, resembling a bark; the patient repeats words he hears (echolalia), mimics movements (echopraxia) or uses obscene or profane words (coprolalia). (iii) The subject has fixed ideas, as arithmomania, in which every action is preceded by counting or doing a certain thing a given number of times; a fear of contamination (*délere du toucher*); *onomatomania*, or repetition of a given word or name; *folie pourquoi*, demanding the reason for everything. The prognosis is usually unfavorable.

(*c*) In *complex coördinate tic*, as thumb-sucking, rocking in the bed and bumping the head, the outlook is favorable, except in feeble-minded children.

Treatment of Acute Chorea.—1. Prophylaxis.—Sensible methods of education should be adopted, the hours in the overheated, foul schoolroom should be short, and the useless forcing of children by marks, prizes and exhibition abandoned. Children can be taught more in an hour or two of judicious individual instruction than in the five or six confining hours which their wandering attention must endure. Prevention may concern other etiological factors (*q. v.*).

2. Rest.—The results obtained by *rest in bed and isolation* are as rapid as those of any medicinal therapy. It not only saves tissue waste, but is the best treatment of endocarditis.

3. Skin.—The dry and harsh skin must be given special attention—stimulation of the skin by proper covering, refrigerant diaphoretics and full warm baths. The skin must be kept clean and in severe cases the water-bed must be used.

Trauma.—Trauma is prevented in severe types by padding the bed by mattresses and pillows, since the violent movements may throw the patient to the floor. Sepsis is a frequent and most dangerous occurrence. The temperature must be taken by rectum.

5. Exhibition of Drugs.—(*a*) *Fowler's solution* is the most frequently used and the oldest remedy. It should be given in increasing doses (see page 677); children tolerate large doses of arsenic exceptionally well. It may be administered hypodermically or by clyster when the digestive tract is irritable. Arsenic is not without its dangers. C. Gamble, Jr., records an instance of fatal arsenical poisoning in a case to whom about $1\frac{1}{2}$ ounces of Fowler's solution was given in three weeks. Rest in bed gives equally good results. (*b*) *Salicylates* are of benefit only when rheumatism actually coexists and acetanilide is indicated only in the initial stages, *i. e.*, antirheumatic remedies are less important in the treatment than is rheumatism in the etiology. (*c*) *Depressomotors:* The *bromides* rarely cure the movements, although they often quiet mild psychic manifestations. Chloral, given in large and continued doses to produce continuous sleep, merely demonstrates the tolerance of the body for dangerous remedies and chloral cannot be given safely in amounts exceeding a dram for adults in three divided doses at intervals of an hour. Morphine is generally dangerous and inferior to chloral. Small doses often accentuate the mental agitation, but if combined with chloral, hyoscine and bromide, it may give some temporary relief. (*d*) *Strychnine* is valuable, especially when recovery is slow. (*e*) In *chorea gravidarum*, abortion or premature delivery is indicated.

OCCUPATION OR FATIGUE NEUROSES; WRITER'S CRAMP.

The continuous, hard, improper use of coördinate, functionally associated muscles may result in their cramping. *Writer's cramp* (graphospasmus, mogigraphia) was first described by Charles Bell (1830) and especially studied by Duchenne and Benedict. Neuropathic tendencies are noted in most cases; 83 per cent. occur in males, 93 per cent. occur in those between twenty and sixty years of age and most cases develop between twenty-five and thirty-five. Worry is a frequent factor; toxemia, cold, trauma, neuritis and neuralgia are possibly factors. The disease practically never occurs in those who write properly, *i. e.*, move the arm from the elbow or the shoulder, but develops in those who use the little finger or wrist as the basis of movement. Some accuse the coördinating cortical or subcortical centres; the occasional atrophy and paresis suggest localization in the cord or nerves. Meige terms it a functional anarchy.

Symptoms.—Gradually fatigue, pain and cramping in the muscles of the forefinger and thumb, may extend to the forearm or shoulder. There may be the "lock spasm" of Mitchell, in which the pen is spastically held or thrown from the fingers. On rest, the tonic spasm disappears, but reappears with attempts to write. In Benedict's classification we find the spasmodic, paralytic, tremulous and neuralgic forms, which are of less interest as types than as showing the various symptoms, which are often combined in a single case. Vasomotor symptoms may occur, as flushing or glossy skin.

Differentiation.—Differentiation, according to the type, from hemiplegic weakness, paralysis agitans, musculospiral paralysis and neuralgia, is easy. *Similar cramps* are observed in telegraphists, milkmaids, weavers, cigarette rollers, tailors, shoemakers, miners, athletes, watchmakers and violin and piano players. Neuroses of the throat in public speakers, jaw spasms in gum chewers and lumbar spasm in physicians who drive much are described.

The *outlook* is poor, because the unfortunate sufferer is often compelled to write until the affection is firmly fixed and when able, usually returns to the same work. In some cases the muscles may waste.

Treatment.—Rest is essential to recovery, because treatment for the usual nervous symptoms alone never succeeds. Plaster casts are sometimes successfully employed. Even when the subject learns to write with the other hand it often develops there. Prevention is possible by writing free-hand from the shoulder; holding the pen between the first and second fingers, use of pens with large handles, of quills and of forearm splints with the pen attached to the extremity, are also helpful. Active systematic gymnastics, passive manipulations and weak phenol solutions injected into tender neuritic spots are helpful.

MYOTONIA (THOMSEN'S DISEASE).

Myotonia is usually called myotonia congenita from its leading causal element, heredity. It was described by Leyden but more fully by Thomsen (1876), who was a victim among 20 cases in his own family in five generations. This rare affection, of which there are 102 reported cases (Koch, 1904), occurs chiefly in males and in Germany and Scandinavia. Its pathology is obscure, for the only two necropsies showed no nervous alteration. Muscle fibers, excised during life, show hypertrophy, decreased striation, nuclear multiplication and slight interstitial increase suggesting a myopathic process.

Symptoms.—A peculiar *transient tonic muscular rigidity* develops when the muscles are first used, which limits or arrests movement, but wears off as their use is persisted in. It is intense when a new kind of movement is undertaken. The longer the muscles are rested the more severe is the initial spasm on exertion. Rigidity does not follow passive muscle movement, although firm pressure or a sharp blow initiates it. It appears perhaps "in the cradle," but generally between the ages of four and ten; it increases as the muscles develop and then becomes stationary for life. In one case improvement was observed. The legs

are more affected than the arms, but in exceptional cases the face, tongue, bladder or the respiratory muscles may be affected. They are usually stronger and sometimes larger than normal. Sedgwick noted von Graefe's sign in five generations.

Though the tendon reflexes and the mechanical and electrical excitability of the nerves are practically normal, application of the galvanic current to the muscles produces sluggish, prolonged closing contractures, with either pole and become tetanic with strong currents, the *myotonic reaction of Erb.* Undulatory contractures follow strong faradic stimulation. Subjects of myotonia are frequently irritable or hypochondriacal.

Treatment.—Thomsen believed that exercise retarded the disease. Strychnine is apparently of value. Cold, dampness, excitement and fatigue are to be avoided.

The following are allied affections: the congenital *paramyotonia* of Eulenberg, consisting of tonic spasms, which occur especially in the arms, and often affect the eyes and mouth; the *ataxic paramyotonia* of Gowers, in which tonic spasms may last the entire day, with muscular weakness, ataxia, especially in the hands and disturbed sensation or anesthesia in the fingers. In *myotonia atrophica*, there are (1) weakness and wasting of the orbicular muscles of the mouth and eyes; the temporals and masseters; the vasti muscles of the thighs with the recti femoris frequently escaping completely; the muscles of the anterior tibial groups, resulting in bilateral foot-drop. (2) The myotonic phenomenon evidenced more particularly by a marked difficulty in extending the fingers from a flexed position. (3) The deep reflexes are depressed according to the degree of muscle wasting.

PARALYSIS AGITANS.

This affection is also known as shaking palsy or Parkinson's disease; it was first described by Parkinson in 1817.

Etiology.—(*a*) Most cases occur between the ages of *forty and sixty-five.* (*b*) Sixty-six per cent. of cases are *men.* (*c*) A *neuropathic tendency* is present in 15 per cent.

Pathology.—It is classed among the neuroses, because of the absence of any constant pathological finding. Arteriosclerosis is often found, since paralysis agitans occurs in the early degenerative period; Gordinier shows that (*a*) general arteriosclerosis is less marked than that of the nervous tissue; (*b*) the arteries in the cord are markedly changed and around them are (*c*) neurogliar sclerosis and (*d*) pigmentation and atrophy of the nerve fibers. Camp found swelling, increased nucleation, hyaloid changes and atrophy of the muscle fibers.

Symptoms.—The cardinal symptoms are tremor and stiffness, slowness of movement, shortening and weakening of the muscles. (*a*) *Tremor* in 66 per cent. of cases is the first symptom; it is sometimes absent (paralysis agitans sine agitatione). It is most marked in the *hands.* It affects first one hand and usually next reaches the leg of the same side, then the opposite hand, the opposite leg, and lastly the trunk, so that it is successively a monoplegic, hemiplegic or finally a diplegic

tremor. It is slow (4 to 7 oscillations per second), rhythmic, fine at the onset but somewhat coarser later, *persists during rest, ceases during sleep* and ceases momentarily on voluntary action. In rare cases it appears only on movement, as intention tremor. In the hands it may affect chiefly the flexors and extensors at the metacarpal joints or the interossei, which give the *spinning or pill-rolling movement.* The wrists may be abducted and adducted, rarely supinated and pronated, and the arms and shoulders are implicated but little. The writing is tremulous. The feet are mostly involved at the ankles and the toes but little. The thigh adductors often tremble. Trembling is infrequent in the face, neck and back, while the abdomenentirely escapes. The head and body may of themselves tremble, but much of it is imparted to them from the limbs. In the rarest cases the tongue, palate, larynx and epiglottis tremble.

FIG. 78.—Side view of a case of paralysis agitans, showing forward inclination of the trunk. Tendency to propulsion. (Dercum.)

(*b*) *Muscular rigidity* and *shortening,* weakness and *slowness of movement* usually follow the tremor; slight symptoms are detected early and anticipate tremor or exist without it. These changes result in the following: (i) The *mask-like Parkinson facies,* which is devoid of lines or expression; the eyelids wink seldom. The forehead shows transverse folds because of the bowed head and body. The eyes move tardily in reading, and the entire body must be turned to enable the patient to look laterally. There is also slow, monotonous, *monosyllabic speech,* with hesitation at first, but hurried at the finish. (ii) The *attitude* is characteristic, because of the flexor positions assumed; the head and the chin are held forward, the neck is extended and rigid, the body is inclined forward, the elbows are fixed and brought somewhat forward and away from the body; the forearms are brought toward the body, the trembling thumb and index-finger are apposed, or the fingers deviate toward the ulnar side as in arthritis deformans, and in standing the knees are somewhat bent and at times closely apposed. The body expression, we may say is like that of the face. (iii) The *gait* is equally characteristic. Like the speech, it is initially hesitant, later hurried. The patient, when once started, may not be able to stop himself. Trousseau spoke of the patients as "chasing their centre of gravity." This *festination* or *propulsion* is

purely mechanical from the forward bodily inclination, although interpreted by Charcot as a forced movement. To counteract the throwing forward of the centre of gravity the arms may be carried behind the back. In some cases, if the patient is pushed backward or sidewise, he may not be able to stop for some steps; this is *retro- and lateropulsion.* (iv) The *palsy* is often slight and rarely extreme, although simulated by the helplessness caused by rigidity. The patient is not inclined to exertion, "moves like a piece of machinery," as Parkinson describes it, and may not be able to turn over in bed. Patients have drowned from falling on the face in a small puddle of water. Muscular reaction requires 40 per cent. more time than normal.

The mind is clear, but *stiff* like the face and limbs, and a certain restlessness, irritability or apathy may be detected; insomnia is common. Sensation is essentially normal, although pains, pruritus, paresthesia and hypesthesia are seen in 35 per cent. of cases. Vasomotor disturbance most often assumes the form of a feeling of heat; indeed the peripheral temperature may be increased several degrees. Severe sweats are common. The pupillary, cutaneous, tendon and organic reflexes are normal.

Course and Prognosis.—The onset is usually gradual and the course covers several decades. The outlook is most favorable in atypical cases. Remissions may occur, but the disease progresses steadily. Death occurs from intercurrent pneumonia, arteriosclerosis, cerebral softening, myocardial insufficiency, nephritis and kindred lesions.

Diagnosis.—Confusion is possible in the early hemiplegic forms, but the absence of a paralytic stroke and the later course are distinctive. Charcot and Ordenstein (1868) clearly differentiated the affection from *multiple sclerosis* (*q. v.*). The fine, *senile tremor*, the *hereditary tremor* of Dana, tremors due to overexertion or excitement, to toxic causes (alcohol, nicotine, lead, mercury, coffee, opium or chloral), weakness of convalescence from acute infections, paretic dementia or epilepsy, Graves's disease, hysteria and neurasthenia are excluded by the absence of the Parkinson rigidity, facies and festination.

Treatment.—Treatment of the probably fundamental arteriosclerosis may possibly retard the disease, but prophylaxis is possible in the early stages only. All mental and physical fatigue should be avoided. The administration of full doses of iodide, chloral, opiates, bromides, hyoscine, or duboisin may mitigate the tremor, but lower vitality and disturb function. Parathyroid gland is recommended.

PERIODIC FAMILY PARALYSIS.

This affection was first described by Cavaré (1853) and more fully by Westphal (1885); Holzapple collected 70 cases (1905); Goldflam collected 11 cases in one family and E. W. Taylor 12 in another. Most cases occur in adolescence, chiefly under twenty-five; it recurs every few weeks or months, possibly every day, or only at intervals of years. Its *symptoms* are almost wholly motor, producing a *flaccid paralysis* in the legs and arms, less often in the trunk, and exceptionally in the face, neck, tongue and throat. It occurs without apparent cause, usually

during sleep, from which the patient awakes with a sense of weakness or dulness which develops into paralysis within twenty-four hours. Mental, sensory, cranial nerve and special sense symptoms are rare. The pulse is often slow and cardiac dilatation has been observed. Sudden death sometimes occurs in an attack. The deep reflexes are decreased or abolished, often also those of the skin, and the faradic excitability of the nerves and muscles is lessened or absent. The patient is well in a few hours or days, until another attack occurs. The disease usually subsides spontaneously after the fiftieth year of life. Goldflam's theory of auto-intoxication is supported (*a*) by J. K. Mitchell, Flexner and Ebersoll, who found increased secretion of kreatin before and during the attack; (*b*) by Crafts and Irwin, who isolated toxins from the feces; (*c*) possibly by the fact that potas. citrate sometimes lessens or aborts the seizure. Bernhardt and others consider the disease affiliated with myotonia and progressive muscular atrophy. Holzapple administers potas. bromide in doses of thirty grains.

MIGRAINE.

Hemicrania, megrim, is an hereditary neurosis, with paroxysmal auræ, headache and vomiting.

Etiology.—(*a*) *Heredity* is a conspicuous cause (90 per cent. Möbius), directly as migraine or indirectly from neurotic, gouty or epileptic antecedents. (*b*) Thirty-three per cent. of cases begin between the fifth and tenth, 40 per cent. between the tenth and twentieth years, and the remainder usually before the thirtieth year. (*c*) *Exciting causes* are constipation, dyspepsia, overexertion, alcohol or sexual abuse, emotional factors, pelvic disorders and lactation.

Symptoms.—As preliminary symptoms the patient may feel depressed, or, in some cases, especially well and energetic. They occur in cases in which the sensory symptoms are least marked. Sometimes the pupils are dilated. Most attacks develop during the night.

Auræ.—In over 50 per cent. of cases, auræ are (*a*) sensory, as visual auræ, a cloudiness before the eyes, sparks, lightning, zig-zag forms, etc.; they are most often bilateral and affect the homologous half-fields; taste and smell may be similarly affected; paresthesia may occur, usually as hemiparesthesia; (*b*) motor auræ are much less common, as aphasia, dysphasia or motor weakness on the contralateral side; in the very rare ophthalmoplegic migraine, there are fugitive ptosis, outward strabismus, double vision and a dilated immobile pupil; mental auræ are uncommon, as anxiety, confusion and double consciousness. The auræ last for ten to fifteen minutes.

The Attack.—(*a*) *Headache,* the cardinal symptom, at first is intense and circumscribed, over the eye, temple, occiput or forehead; then it spreads over one side (*hemicrania*), sometimes affects the opposite side or rarely reaches the neck or arm. The pain is deep, being most often boring, and even as intense as in brain tumor or meningitis. It is increased by movement, stooping or lying down. The special senses are hyperesthetic. The mind is usually clear and often acutely active,

sometimes there is double consciousness, and in rare cases stupor. (*b*) *Nausea* follows, often with vomiting of undigested food and finally of bile—"bilious headache." Feeding and medication are impossible in severe cases. Vomiting may be attended by hyperacidity or moderate collapse, as in sea-sickness. (*c*) *Vasomotor symptoms* may be present, of the *spastic type* with pallor, coldness, wide pupils and retraction of the eye-balls. Thoma found the temporal artery sclerotic on the side of lesion, and de Giovanni noted a marked arcus senilis on the same side. The pulse may be slow and tense and the breathing superficial. In the *paretic type*, there are redness of the skin, small pupils, sweating and increased flow of saliva or tears. Very rarely is there erythromelalgia, edema or ecchymosis. It is best to regard these sympathetic symptoms rather as a result than as a cause of the attack.

The attack ends with the vomiting, after which the patient frequently sleeps, or with almost critical sweating, lachrymation, polyuria or epistaxis. The attack lasts from ten to twelve hours; the term *status hemicranicus* is used for cases of long duration or those in which the attacks overlap. *Most attacks are atypical*, as headache alone, vomiting alone, or headache with vomiting; in a few cases the attack aborts with the aura. The auræ, pain, gastric and cardiac inhibition, vasomotor symptoms and vomiting prove that migraine is a *functional cerebral affection.*

Course and Prognosis.—When the disease is established in youth its attacks become more frequent in adolescence and usually last until the forty-fifth or fiftieth year. The periodicity, noted by Tissot, Trousseau and Liveing, has not been explained. Overwork, worry, menstruation or change of routine may precipitate an attack at a regular or irregular interval. Attacks may cease during an intercurrent pregnancy, tabes or gout. The school of Charcot emphasized the fortunately rare danger of transformation into epilepsy, labyrinthine vertigo or some psychosis. Complete cases are more obstinate and dangerous than light forms.

Diagnosis.—With a history of heredity and development in childhood, the full attack is most characteristic. "Sick headache" is practically always migraine. Atypical forms may be confused with various *headaches*, classified as follows:

1. *From nervous affections*, as (*a*) cerebral neurasthenia, in which there is a sense of constant pressure rather than actual headache, without nausea, but with paresthesia, and in location frontal or general, sometimes occipital or temporal; (*b*) hysteria, which is less common (clavus headache, "neuralgic headache"), and sometimes is precisely like migraine; (*c*) epilepsy, which some consider as identical with migraine; the epilepsy aura is shorter and loss of consciousness is the most constant symptom; (*d*) meningitis, which in all types is early and severe; (*e*) hydrocephalus, which is often severe, especially in acquired forms; (*f*) brain tumor, which is constant, generalized, often very severe and sometimes typically hemicranic; (*g*) paretic dementia, in which migraine is frequent; care is necessary when migraine occurs after syphilitic infection.

2. From affections of the *special senses*, as eye-strain, hypermetropia; glaucoma, which most often produces frontal headache; nasal disease, which may cause sinus involvement with frontal headache.

3. *From toxemia.* (*a*) Those due to acute infections are usually frontal and dull, but sometimes occur in other locations and are severe, deep-seated and increased by stooping; the rheumatic or indurative headache is oftenest occipital, attended by tenderness, often by induration and pain on motion; it is severe; (*b*) intoxication by alcohol, nicotine, lead, nitroglycerin and opium, and (*c*) constitutional states, as gout or arthritis. (*d*) In nephritis they are usually frontal, remittent or continuous, but sometimes sharply hemicranic. (*e*) In gastric affections they are exceedingly common, mostly occipital, but sometimes vertical or frontal.

4. *From circulatory causes,* as from wearing a tight collar, repeated coughing, cardiac disease or arteriosclerosis (with vertigo), and usually increased by lying down.

5. *From anemia,* especially from chlorosis, as a "tugging" sensation back of the eyes or frontal or generalized headache, and often relieved by lying down.

Treatment.—*Prophylaxis* is said to be of little value, but many subjects recognize the warnings and avert the attacks by resting, on noting unusual mental or physical capacity for work or an unusual appetite, which ushers in some attacks. Migraine subjects should observe *moderation* in eating, especially of meat, in work, exercise or recreation, narcotics, stimulants, as coffee or tobacco, and in sexual matters. Immoderation is a neurosis in itself. Hurry, worry and tension must be avoided and their first manifestations must be learned and treated. The extract of *cannabis indica,* gr. $\frac{1}{4}$–$\frac{1}{2}$, given for weeks or months, after Seguin's plan, is almost the only remedy which helps the condition, and then only in some cases. Möbius finds that *salicylate of soda,* gr. xv, in black coffee, given the evening before the attack, often wards it off. Nitroglycerin in full doses sometimes aborts it, as may washing out the stomach and following the lavage with a saline.

In the *treatment of the established attack,* bromides constitute the best remedy; they are given not only during the attack in the mild type, but continuously, as in epilepsy, when the type is severe and frequently repeated (Liveing and Charcot). Phenacetin often helps mild forms and those with initial rise of blood-pressure. Morphine is to be avoided. The stomach sometimes rejects all medication, which must then be given by rectum. Application of cold to the head, hot foot-baths and sinapisms to the nape of the neck and epigastrium are subordinate helps.

NEURALGIA.

Pain is only a symptom, usually of an affection of a peripheral sensory nerve, although in rare cases affections of the cortex, pons, medulla or parts near the thalamus may cause peripheral pain. The pain of neuralgia and neuritis may be confused.

Etiology.—(*a*) Neuralgia occurs mostly in *adult life* (twenty to sixty) and rarely before puberty or after sixty, in which case it is usually severe and intractable. (*b*) Most cases occur in *women.* (*c*) *Heredity* is an apparent factor in 25 per cent. of cases, especially in neuropathic, weakly

or gouty families. (*d*) *Debility* from exhausting conditions, as lactation, anemia, overwork, sensory fatigue, as from eye-strain, sexual or other excesses, emotional conditions and trauma may cause it. (*e*) It may result from *acute infections*, notably *influenza*, typhoid at its onset, *malaria*, the causal importance of which has been much overestimated, rheumatism (from exposure to cold) particularly of the fascial type, and sometimes syphilis. (*f*) Alcoholism, *diabetes*, plumbism, gout and nephritis may produce it; most of these are also causes of neuritis, differentiation from which may be difficult. (*g*) *Nerve affections*, as sciatica and neuritis, herpes zoster, reflex irritation, as radiated from a carious tooth, compression of a nerve trunk by varices, aneurysms or neuromata or the root pains of tabes, may cause neuralgia. (*h*) Arteriosclerosis may affect the nutrient nerve vessels.

General Symptoms.—The *pain* presents the following characteristics: (*a*) It is always *paroxysmal*, with intervals of total or relative relief; intense pain is never constant. (*b*) It *recurs* irregularly at intervals varying from hours to months or with surprising periodicity on a given day or hour, perhaps at the menstrual period; periodicity is as frequent in non-malarial as in malarial cases. (*c*) It is usually *unilateral*, generally constant in its seat, and most often found over the distribution of a single nerve or several nerves; bilateral pain is usually symmetrical. The pain may shift from place to place, especially when due to systemic causes. It is probable that the spinal cord may be the medium of radiation of pain from one region to another and that pain may follow the distribution of the spinal segments rather than that of the peripheral nerves, as in herpes zoster. (*d*) Its *onset* and *cessation* are *sudden and spontaneous;* an imminent attack may be precipitated or existing pain may be aggravated by muscular movements, a breath of air or a simple touch; the character of the pain is *sharp, stabbing*, rarely pulsating; if it travels along the nerve trunk its shooting direction is usually centrifugal, rarely centripetal; if it is localized it is commonly burning or boring. (*e*) The pain is *usually deep*, but the skin is very hyperesthetic. (*f*) The "*tender points*" of Valleix (1811), later called maximal points by Head, are found in the nerve (50 per cent.) both in the attack and the free interval, especially where the nerve emerges from the bone or fasciæ, lies on hard surfaces, divides or anastomoses. The occasional tenderness over the spine at the origin of a nerve (Trousseau's *point apophysaire*) occurs in many other conditions. In neuritis the nerve trunk along its entire accessible course is tender. (*g*) *Vasomotor constriction*, followed by dilatation, is fairly common. Edema, erythema, blanching or falling out of the hair, secretory disturbances, herpes, pigmentation, numbness or tingling, trophic alteration of the skin, localized hypertrophies and reflex twitchings of the muscles are much less frequent than in neuritis. Persistent pain or its etiological factors may reduce the general nutrition.

Local Clinical Varieties.—1. TRIFACIAL NEURALGIA (prosopalgia, *tic douloureux*) is the most important, severe and frequent type, because the fifth nerve is most exposed to trauma, compression and infection, by way of the eye, nose and ear. The entire nerve is not usually affected, though pain may radiate from one division into another; indeed only the

secondary branches of a single division may be involved. The general features of neuralgia are present, as already described. (*a*) Involvement of the *first branch* is the most common form; pain occurs over the eye, forehead and frontal sinus (in which the familiar pain is experienced after eating ice-cream); it sometimes radiates into the second and third branches; there are "tender points" over the lower edge of the nasal bone, the supra-orbital notch, less often over the supra-orbital nerve, even to the vertex, sometimes over the occiput or over the cervical spines; cold, carious teeth, disease of the sinus, glaucoma, hypermetropia, etc., are causal factors. (*b*) When the *second branch* is involved there is pain over the side of the nose and cheek from the infra-orbital region to the upper lip; there are "tender points" over the lower border of the nasal bone, the malar bone, the infra-orbital foramen, the gums over the canine teeth, and sometimes in the hard palate; among other causes is the dental alveolitis in the aged, due to sclerosis. Diseased teeth may cause neuralgia without local dental symptoms and in every severe case the dentist should be consulted. (*c*) Involvement of the *third branch* is the least frequent form; inframaxillary pain, along the lower teeth, tongue, chin, external ear and maxillary region; and tenderness over the auriculotemporal nerve and inferior dental foramen may occur. In old persons the loss of the teeth brings the jaws closer and therefore stretches the inf. dental nerve; it is relieved by proper plates. In severe forms vasomotor symptoms and increased secretion from the eyes, nose and salivary glands are usually present. Hyperalgesia is such that the face cannot endure simple contact, and drinking, mastication and wiping the nose cause great suffering. Herpes and trophic changes are probably neuritic rather than neuralgic symptoms. In rare instances facial spasm, transient reflex ptosis, internal strabismus or myosis coexists. *Differentiation:* In headache and migraine there are no tender points; intracranial disease, as tumor or syphilis, is usually excluded by systematic examination. The *prognosis* is doubtful in severe forms, which are often life-long in duration and agonizing in degree.

2. Cervico-occipital Neuralgia.—There is pain in the first four cervical nerves, especially the inferior occipital and auricularis magnus; this pain sometimes radiates into the fifth nerve; there is tenderness half-way between the mastoid and spine, over the parietal eminence and over the cervical plexus between the sternomastoid muscle and trapezius; this form is often bilateral and is less intermittent than other neuralgias, which facts are explained by its frequent connection with cervical caries and syphilis, and with tumors, meningitis, root disease and affections of the ear.

3. Phrenic Neuralgia.—Phrenic neuralgia is rare; it sometimes occurs in pericarditis and diaphragmatic pleurisy; it is usually left-sided, with pain and tenderness over the insertion of the diaphragm and nerve trunk in the neck; movements involving the diaphragm are painful. Differentiation is usually uncertain.

4. Cervicobrachial Neuralgia.—Any nerve may be involved, especially the ulnar and circumflex. Pain may radiate over the entire plexus and chest; the tender points are most often in the axilla, shoulder

and ulnar distribution; it is often confused with neuritis from trauma, compression, rheumatoid arthritis or other organic disease.

5. Intercostal neuralgia ranks third in frequency after trifacial and sciatic neuralgias (neuritis). Pain is felt along the intercostal nerve and tenderness over the *points* of emergence of *its three branches,* over the angle of the ribs, in midaxilla and near the mammary line. It is more easily distinguished in the upper than lower interspaces. Differentiation may be difficult in muscular rheumatism in which movement is more painful. Peritoneal disease, ulcer, spinal caries, aneurysm and tabes must be distinguished. It is most common in gastrohepatic affections and neurotic women.

Herpes zoster is an acute inflammation of the posterior root ganglia (Bärensprung and Charcot) which are the seat of a cellulohemorrhagic exudation; its fibers to the posterior column degenerate. It is mentioned in this connection because of the acute pain preceding and associated with the rash, which appears after three or four days; the pain is often protracted and intractable; the pain on the trunk follows the distribution of the spinal segments rather than that of the intercostal nerves. J. Ramsay Hunt collected 15 cases of herpes of Gasserian origin, associated with ocular palsies, and 60 palsies associated with herpetic inflammation of the geniculate ganglion, and also describes herpes zoster of the face and neck with auditory symptoms and facial palsy.

6. Lumbar Neuralgia.—This infrequent type occurs in the ilio-hypogastric, ilio-inguinal and genitocrural, sometimes in the ext. cutaneous, obturator and crural nerves. The pain is felt over the iliac crest, inguinal canal, labium or testis (irritable testicle).

7. Sacral Neuralgia.—Coccygodynia is neuralgic and occurs mostly in women. The pain is increased by sitting, coitus and defecation; it is obstinate and resists even operative measures.

8. Pododynia.—Neuralgia of the heel is termed pododynia; plantar neuralgia, which is mostly neuritic, occurs in acute infections and caisson disease. Morton's metatarsalgia, regarded as bony compression of the fourth metatarsophalangeal nerve, usually occurs in one foot and mostly in women; operation is usually indicated.

9. Visceral Neuralgias.—(See Diseases of the Heart, Stomach and Kidneys, Hysteria and Neurasthenia). Under this caption may be given *Head's areas of pain* referred to visceral disease: the organ is first given, and following it is the area to which its pain is reflected: *Heart:* 1st, 2d, 3d dorsal segments. *Lungs:* 1st, 2d, 3d, 4th, 5th dorsal segments. *Stomach:* 6th, 7th, 8th, 9th dorsal; cardiac end from 6th and 7th. Pyloric end from 9th. *Intestines:* (*a*) Down to upper part of rectum, 9th, 10th, 11th and 12th dorsal. (*b*) Rectum, 2d, 3d, and 4th sacral. *Liver and gall-bladder:* 7th, 8th, 9th, 10th dorsal; perhaps 6th dorsal. *Kidney and ureter:* 10th, 11th and 12th dorsal. The nearer the lesion lies to the kidney the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter the more does the 1st lumbar tend to appear. *Bladder:* (*a*) Mucous membrane and neck of bladder, 1st, 2d, 3d, 4th sacral. (*b*) Overdistention and ineffectual contraction, 11th and 12th dorsal and 1st lumbar. *Prostate:* 10th, 11th, 12th dorsal,

1st, 2d, 3d sacral and 5th lumbar. *Epididymis:* 11th and 12th dorsal and 1st lumbar. *Testis and ovary:* 10th dorsal. *Appendages:* 11th and 12th dorsal and 1st lumbar. *Uterus:* (*a*) In contraction. 10th, 11th, 12th dorsal and 1st lumbar. (*b*) Os uteri. 1st, 2d, 3d and 4th sacral.

Treatment.—Logically, the treatment of the cause (*q. v.*) should precede that of the symptoms, but practically, pain is the prime indication for therapeutic measures.

1. Local Measures.—Pressure on tender points may diminish or abolish pain. As in sciatica, blisters and sinapisms (both of which may leave permanent pigmentation on the face), the cautery, local heat, acupuncture or injections of water or cocaine may give relief. Freezing the skin by ethyl chloride often gives better results. Menthol, camphor-chloral (equal parts) and liniments are of some value (tr. aconiti, tr. chloroformi, tr. belladonnæ āā ʒij, linimentum saponis ʒiv). Injections of 1 per cent. solution of osmic acid into the nerve trunk are very successful; they were recommended by Billroth and Neuber (1884). Schlösser (1900) recommended alcohol injections; Patrick treated 75 cases without a failure and Kiliani 190, with 5 failures. Massage is often beneficial, for the muscles and nerves have common veins.

2. Nutrition.—A full diet, with milk and cod-liver oil, the Mitchell rest-cure, hydrotherapy and massage, are often beneficial, especially with change of climate or surroundings. Alcohol, which is always dangerous, is most often indicated in older subjects. The beneficial effects of castor oil are often great, probably because it promotes elimination. Gouty subjects may thrive on a vegetable diet.

3. General Drug Treatment.—Dana gives strychnine hypodermically in doses of $\frac{1}{30}$ gr. even up to $\frac{1}{5}$ gr. Arsenic, iron and phosphorus are excellent in anemic, malarial and degenerative types; quinine is beneficial in non-malarial as well as malarial types and in trifacial neuralgia especially, although its value is overestimated. Valerian, cannabis indica and bromides should be given for irritability; salicylates for rheumatism and colchicum for gout; iodides are sometimes of benefit to non-syphilitic subjects; full doses of nitroglycerin are frequently helpful.

4. Analgesics.—Acetanilide (gr. v) and the other coal tars should be used in fresh cases. Extr. cannabis indicæ (gr. $\frac{1}{4}$) and fluidextr. gelsemii (♏ij–iv) should be administered every three to six hours in the less severe cases. In acute febrile or rheumatic cases, fluidextr. aconiti (♏j–ij) should be given every two hours until tingling in the mouth or extremities and slowing in the pulse intervene. Liebreich advocates croton-chloral, gr. v, or with care even gr. xx, for several doses. Finally, in severe cases, morphine, as far as possible, should be avoided; sometimes its administration is inevitable; though some writers find that it tends to prevent recurrence, the pain often develops again, intensified; atropine sometimes more permanently controls the pain than does morphine.

5. Electrotherapy sometimes relieves pain, as galvanization with a weak current, just strong enough to produce a sensation of burning and applied with the sponges warm and with the anode to the painful area, especially in newly developed cases.

6. Surgical therapy includes nerve section, resection, stretching or divulsion; these procedures often fail. In incoërcible trigeminal neuralgia the Gasserian ganglion may be extirpated (Rose, 1890), but this operation entails a high mortality, and loss of the eye may result.

VASOMOTOR AND TROPHIC NEUROSES.

Vasomotor centres exist in the cerebral cortex and medulla. The fibers probably run in the lateral columns of the cord and leave by the anterior roots. *Vasomotor paralysis* occurs independently or with functional or organic disease. Paralysis of the cervical sympathetic may result from trauma, aneurysm, tuberculous apical or glandular disease or disease of the cord. It is attended by redness, rise of temperature, pulsation and tortuosity of the vessels, sweating, unrest, ringing in the ears and palpitation. In paralysis of the cervical sympathetic there are pupillary narrowing from the unopposed action of the third nerve, slight ptosis, narrowing of the interpalpebral fissure from paresis of Müller's muscle, salivation, lachrymation and in cases of long standing, retraction of the eye and flattening of the cheek. The pupil reacts somewhat to light but not to stimulation by pain or pinching of the skin of the neck (Möbius); the pupil can be widened by atropine, but to a less degree than the normal eye.

Vasomotor spasm presents converse symptoms—pallor, coldness, small vessels and frequently a sense of tingling or arterial pain. Two examples are nervous angina pectoris and Raynaud's disease; spasm (contraction, irritation) of the cervical sympathetic produces wide pupils, an increased interpalpebral fissure and exophthalmos.

Little is known of the *trophic nerves*, if indeed separate nerves subserve nutrition. If they exist they are probably closely related to the vasomotor tracts. Trophic changes may be symptomatic of disease of the nerves or cord, as in neuritis or nerve trauma, facial hemiatrophy, tabes, syringomyelia and myelitis.

Erythromelalgia.—Erythromelalgia, described by Graves (1843) and S. Weir Mitchell (1872), indicates *redness and pain in the extremities.* It occurs mostly between the years of twenty-five and forty and is slightly more frequent in males. Pathologically, Mitchell and Spiller find neuritis and sclerosed arteries. The toes (less often the fingers) and balls of the toes are the seat of severe, paroxysmal pain, swelling and redness, particularly when the limbs depend; the arteries throb and the veins are engorged; the color is sometimes blue. Pain is occasionally experienced in the head or neck and vertigo and muscular weakness may be noted. The symptoms occur mostly in warm weather and generally disappear in winter. The clinical course is long, the prognosis doubtful and the treatment consists of electrotherapy, cool baths, acetanilide and iodides.

Acroparesthesia.—Closely related to erythromelalgia, it was described first by Nothnagel, and F. Schultze (1890) named this very frequent affection; its derivation signifies *paresthesia of the extremities.* More than 90 per cent. of cases occur in women between thirty and sixty years of age. The etiology is doubtful (stasis or arteriosclerosis). The chief

feature is an unpleasant crawling, tingling sensation, sometimes amounting to actual pain in the fingers or hands, less often in the toes or feet. The discomfort is greatest at night and in the early morning; it is peripheral and symmetrical but does not correspond with any nerve area. Generally there are no objective findings other than occasional vasomotor disturbance or slight sensory blunting. The outlook is favorable, the course is chronic and the faradic brush frequently affords relief.

Spontaneous Symmetrical Gangrene.—Raynaud's disease was first described by Raynaud (1862). Sixty-six per cent. of cases occur in women, who are usually delicate, anemic and neurotic, and most cases develop in persons between twenty and forty-five years of age. In 10 per cent. it is hereditary. Other causal factors are uncertain, as exposure to cold and dampness, exhaustion and acute infections. Occasional coexistence is noted with hysteria, neurasthenia, epilepsy, tabes, syringomyelia, angioneurotic edema, neuritis, acute mania, etc. Its pathology is not established, but arterial spasm, endarteritis and neuritis are said to exist. There are *three stages:* (*a*) *Local ischemia* (or syncope) begins with pallor and coldness of one or two fingers, perhaps of the entire hand or of the toes and is attended by numbness or severe pains. The "dead fingers" or toes appear on exposure to slight cold or on emotion and reaction occurs after a variable period. The process may not pass this stage. (*b*) *Local asphyxia* (or cyanosis) develops in the parts involved, at times with some swelling or anesthesia. It is most severe in winter and chilblains are regarded as the mildest type of this stage. (*c*) *Local gangrene*, which is spontaneous, symmetrical and fortunately rare, is the last stage. It is dry, sometimes vesicular and its extent is always less than the area of ischemia and asphyxia. The tips of the fingers or toes dry up, fall off and scars mark successive attacks. Less often the tip of the nose, ear, buttock, heel, shoulder or abdomen is involved and very rarely the tongue, penis, elbow or knee. In some instances there is severe general disturbance, as fever, chill, enlarged spleen, colic, hemoglobinuria, uremia, stupor, coma, convulsions, transient hemiplegia, neuritis and spastic retinal vessels. The severe or fatal cases occur especially in children. The *course* is chronic and consists of repeated attacks, which often abate in summer.

Differentiation.—Differentiation is usually easy from hysterical gangrene (self-injury), diabetic, arteriosclerotic or embolic gangrene, syringomyelia, leprosy, ergotism and acute arteritis, as in typhoid.

Treatment.—Narcotics are given for pain; warmth and elevation promote venous return and nitroglycerin dilates the arteries. Cushing advised Esmarch's constrictor for a few minutes. In actual gangrene surgical interference should be postponed until complete demarcation develops, because the area is much more circumscribed than at first indicated.

Acute Angioneurotic Edema.—It was first fully described by Quincke (1882), although Milton (1876) described "giant urticaria." It occurs most frequently between the years of twenty and thirty-five and is slightly more frequent in women than in men. It is allied to urticaria, erythema nodosum and possibly to some forms of purpura, and consists of

a circumscribed edema, which is sudden in onset and in disappearance, is tense and pale, does not pit on pressure, sometimes causes burning and appears mostly on the eyelids, lips, cheek, tongue, hands or genitalia, sometimes on the buttocks, in the pharynx, larynx, lung or digestive tract. It sometimes attacks several areas simultaneously. Gastro-intestinal symptoms occur in 33 to 50 per cent. of cases, as epigastric pain, vomiting, colic or diarrhea (see p. 693). Hemoglobinuria, albuminuria and polyuria are less frequent. The outlook is good, except that laryngeal or pharyngeal localization may cause sudden death. *Recurrence* is not uncommon, perhaps with considerable periodicity.

Treatment.—Treatment is symptomatic; nitroglycerin is recommended. Bloodgood employed deep incisions with great benefit. Wright exhibited calcium lactate (gr. xx) with success.

Herz described a *pseudoperiostitis angioneurotica*, occurring especially on the thoracic bones; it is periodic, transitory, painful, tender and associated with neurotic cardiac disturbance.

Chronic Hereditary Trophedema.—Twenty-two cases of chronic hereditary trophedema in six generations were described by Milroy (1893) of Omaha, 8 cases in four generations by Meige (1898) and 13 cases in five generations by Hope and French (1898). A firm, pale, painless edema, chiefly below the hip or knee, develops toward puberty, without cardiac, renal or apparent local cause. It may appear in the arm and leg of the same side, including probably some of the so-called local hemihypertrophies. The first cases recorded were thought to be phlebitis. Health and life are not threatened, although treatment produces no results.

Hydrops Articulorum Intermittens.—Hydrarthrosis intermittens occurs in 71 per cent. in persons between ten and forty years of age; about 70 cases are on record. Its etiology is not known. Some cases present malarial, gonorrheal or traumatic antecedents. The knee is usually involved when several joints are affected and in 33 per cent. is alone involved. The effusion wholly disappears after two or three days, but it reappears obstinately every one to four weeks. There is no fever and usually no pain. *Treatment* is general.

Scleroderma.—Described by Lusitanus and Alibert, Thirial (1845) gave the first full account. It is a chronic thickening and hardening of the skin, followed by atrophy. It is a rare affection, but is more common than is generally recognized. The *localized form* usually develops before and the *generalized form* after the twenty-fifth year. Seventy-five per cent. of cases occur in women. The *causation* is obscure; erysipelas, trauma, cold and neuropathic disposition are sometimes considered etiological and association with other neuroses, general and vasomotor, is frequent. *Anatomically* the skin and all the contained structures are fibrosed. In the localized form it occurs in spots or streaks, which often correspond to the course of given nerve trunks or spinal segments. In the general form the body may be literally "hide-bound." Its distribution in order of frequency is the upper extremities, trunk, head and face, lower extremities, and generalized. The skin is usually first painful and sensitive, then reddened and thickened, and finally pale, glossy, dry,

atrophic and parchment-like. It is rigid and without folds, inhibiting the mimetic play of the features. The nose is peaked, the lids cannot close over the eye-ball and may become everted. The mouth is so small as to sometimes necessitate extraction of some of the teeth and rigidity in the neck may render swallowing difficult. In the fingers it occurs in 5 per cent. of cases (*sclerodactylia*, Ball). The close skin produces decided deformity and loss of function. Sensation is usually preserved, the secretion of sweat is usually reduced and pigmentation and trophic changes in the hair and nails are occasionally seen. In some cases it occurs with herpes (the herpetiform morphea of Hutchinson). Circumscribed atrophy of the bones is said to occur. Only 30 per cent. of cases improve and 15 per cent. recover.

Treatment.—Treatment is unsatisfactory. Improvement has followed administration of salol and inunctions with oil; thyroid extract, especially if used early in the disease, may promote recovery.

Ainhum.—Ainhum was first observed by Clark (1860), then by Da Silva Lima (1866). It is a chronic affection, usually limited to the great toe, which at its plantodigital fold suffers a slow, painless, non-inflammatory circular constriction and spontaneous amputation. Ainhum is chiefly found in the colored races of Brazil, Africa, India and sometimes in this country. Its nature is disputed; an ingrowth of the epidermis and scleroderma have been thought to be causes. It is not amputating leprosy, as has been claimed.

SECTION X.

DISEASES OF THE LOCOMOTOR SYSTEM.

DISEASES OF THE MUSCLES.

MYOSITIS.

Inflammation in the muscles may be single or multiple; it may be an independent or a secondary affection. It may reach the muscles by contiguity from adjacent disease, may result from trauma, trichinosis, cysticercus, echinococcus, myxo- sarco- and microsporidia, sepsis, puerperal fever, rheumatism, scarlatina, gonorrhea, syphilis or erythema multiforme. The muscles are the seat of serous, sometimes hemorrhagic or diffuse purulent exudation in septic forms and of fatty degeneration. In a form, endemic in Japan, the Staphylococcus pyogenes aureus occurs in pure culture. The muscles are usually friable and sometimes fragmented. *Polymyositis*, first described by Wagner (1887), Hepp and Unverricht, occurs most often in youth and adolescence and is attended by pain, especially near the tendons, swelling and often edema in the muscles, and decreased tendon reflexes. The skin may participate, with herpes, erythema, bullæ or purpura (*dermatomyositis*); the infection may involve the tissue in which the afferent nerves originate (muscle spindles), (*neuromyositis*, Senator). In some cases there is arthritis or phlebitis. Oppenheim describes a case of *dermatomucomyositis* with stomatitis, iritis and conjunctivitis. The inflammation occasionally reaches the tongue, pharynx, larynx or respiratory muscles and may cause difficult mastication or swallowing, ptosis or dyspnea. The systemic reaction is severe, marked by fever, chills, sweats, splenic tumor, sometimes by angina, bronchitis, albuminuria or leukocytosis. Death is the usual issue.

In muscular *fibrositis*, the morbid process is an inflammatory exudation into the fibrous tissues; the swelling and thickening may be slight and impalpable; severe and definitely tumor-forming; acute and transient; chronic, and eventuating in hard, fibrous lumps. The most constant symptoms are pain and tenderness; if vigorously rubbed, the swelling and the pain are greatly increased. If a patient presents himself with any of the ordinary manifestations of chronic rheumatism, as a lumbago, sciatica, pleurodynia, or a cephalalgia, and with any obscure myalgic or neuralgic pain in any part of the body, Telling urges that a careful investigation should be made of the fibromuscular tissues of the affected areas.

Differentiation.—Differentiation concerns *multiple neuritis* (tenderness over the nerve trunks, motor and sensory findings); trichinosis (trichinæ in the tender, swollen muscles, eosinophilia and digestive symptoms); and *periarteritis nodosa* (*q. v.*).

Treatment.—Treatment is symptomatic and is identical with that of sepsis. Sweats and salicylates are beneficial.

Myositis Ossificans.—Myositis ossificans usually develops acutely in the muscles of the neck or back before the fifteenth year and leaves residual foci of infiltration, partly in the periosteum and partly in the fasciæ and tendons, which gradually calcify. The thigh and arm are most severely and permanently affected. The process occurs in the strongest muscles and at an age when they develop most actively; it may affect many muscles and runs a chronic course, perhaps of several decades. Schutz collected 275 cases of the localized form and A. R. Elliott, 107 cases of the progressive inflammatory type, often associated with infantilism, microdactylia, etc. Massage is sometimes beneficial.

MUSCULAR RHEUMATISM (MYALGIA).

This includes many vague but important affections. It occurs particularly in men, promoted by cold, dampness and certain occupations. Muscular overexertion, as straining and lifting, is frequently a part of the history. Rheumatic and gouty subjects are specially and recurrently exposed. Disorders of metabolism or mental as well as physical overwork mark some cases. Other forms are, as Leube insists, probably infectious, for fever occurs in 33 per cent. and occasionally endocarditis; muscular rheumatism in this sense does not generally involve many muscles, but usually one muscle or a single group. The pain is not paroxysmal, as in neuralgia, and is experienced in the muscle or reaches it from its tendons or fasciæ. In some cases it is a mild neuromyositis. Its varieties are: *Myalgia capitis*, affecting the fascia of the head; *omalgia*, involving the muscles of the shoulder, or rheumatic *torticollis*, which is usually unilateral; *pleurodynia*, which affects the pectoralis, intercostals and serratus, limits respiratory movements and is likely to be confused with intercostal neuralgia, pleurisy, periostitis of the ribs or vertebral disease; *lumbago*, which involves the lumbar muscles, and is the most frequent, painful and obstinate form, causing great pain on movement or on efforts of the uninvolved muscles to maintain quiet; it is probably due in some cases to muscular rupture, as after lifting. In the abdominal muscles and glutei it may simulate pelvic disease. In *treatment*, rest is indicated, for use of the affected muscles aggravates pain and protracts the condition. Involuntary movement is prevented by careful strapping with adhesive plaster. A hypodermic of morphine into the muscle is often indicated. Deep injection of water into the muscles of the affected part may relieve pain. Ringer's method of acupuncture with long needles which are pushed three inches into the back and left there for from five to ten minutes is beneficial, but is usually vigorously opposed by the patient. Very energetic massage, with the local application of heat, probably gives the best results. The actual cautery and constant cur-

rent give good results. Belladonna plasters afford relief. Gelsemium acetanilide and salicylates may relieve certain cases.

Chronic cases, with vague, fugitive pains which come on with change of weather, are treated in the same way. Confusion is frequent with chronic alcoholism, tabes, gout and diabetes.

AMYOTONIA CONGENITA.

Of Oppenheim's disease, Griffith (1910) collected 49 cases. Some cases are apparently myopathies and others of spinal origin. This congenital condition is characterized by extreme flaccidity and weakness, smallness and softness of the muscles, oftenest of the legs and most rarely of the face. The deep reflexes are lost, the faradic excitability of the muscles is lessened and contractures are likely to develop. There is a tendency toward slow improvement.

Myotonia, paramyoclonus multiplex and myasthenia gravis are described elsewhere.

DISEASES OF THE JOINTS.

ARTHRITIS DEFORMANS AND CHRONIC RHEUMATISM.

Excluding gouty, traumatic, trophic, tuberculous, syphilitic, gonorrheal and hemophilic affections, there remains a group of articular affections of which the chief are arthritis deformans and the so-called chronic rheumatism. Several totally different affections are most unsatisfactorily gathered under these names.

Arthritis deformans is a progressive or at least chronic affection, marked by changes in the synovial sac, cartilages and bone, causing functional disturbance and usually symmetrical and polyarticular deformity.

Etiology.—(*a*) The predisposing factors are (i) *heredity,* which is causative in 13 per cent. (Garrod); (ii) *age;* 55 per cent. of cases occur between twenty and fifty years of age; 5 per cent. under twenty years and 40 per cent. over fifty years of age (Garrod); (iii) *sex;* Stewart and McCrae consider that the sexes are equally involved; (iv) fatigue, cold, dampness. poor hygiene or trauma may be predisposing factors. (*b*) *The immediate cause* is unknown; the *infective* theory is based upon finding bacteria in the joint, enlarged spleen and lymph glands and its occasional development after infections; some cases can be traced to disease of the tonsils, sinuses, gall-bladder, appendix, tubes, prostate, pyorrhea alveolaris, etc.

Pathology and Symptoms.—Whether the synovia or cartilages are first involved is undetermined; the *x*-rays do not solve the question; practitioners, who see cases early, hold that the serous sac is first affected and surgeons find the cartilages most often diseased. The synovial villi hypertrophy and the cartilage slightly fibrillates, cleaves and finally atrophies. The bone becomes thin in some places and tuberous in others

from periosteal proliferation (Haygarth's nodosities); the joint surfaces become eroded and in part grow together.

1. Diffuse Progressive Type.—(*a*) The *acute form* is less common. There is some fever, rise in the pulse-rate and general depression and the joints become red and swollen, resembling acute articular rheumatism, but differing from it in that arthritis deformans is less fugitive, involves the small joints less frequently, is attended by less sweating, and seldom by heart complications, does not react to salicylates and runs a longer course. In some cases it even resembles typhoid, because the fever and splenic enlargement antedate the arthritis. (*b*) The more frequent *chronic form* develops insidiously, with uncomfortable sensations in the joints or racking nocturnal pains about the joints and vasomotor skin symptoms. It begins most often in the small joints of the hands, with or without swelling, and remits and recurs. The hands often sweat. The fingers become fusiform from joint changes, capsular thickening and

Fig. 79.—Rheumatoid arthritis. (Musser.)

collateral edema which separate the fingers from each other. The edema often disappears, showing more clearly the osseous and capsular thickening. The cartilages become uneven and crackle on movement, loosened villi may become free bodies and the adjacent bursæ and tendons may swell. The fingers deviate and become angular from flattening of the saddle-shaped epiphyses and extensor or flexor muscular contractures. Extension to other joints occurs; the elbow becomes flexed, the shoulder often crackles and becomes fixed; the large toe is bent out and lies under the other toes, which are often extended at their first and flexed at their two distal articulations; the ankle is often abducted; the knee is swollen, shapeless and flexed from effusion or contracture; in a few cases the hip, jaw and spine are involved. The vertebræ may be affected (*v. i.*).

Motion is limited by the swelling, muscular contractures or articular fixation by interlocking extracapsular nodosities and capsular induration. The contiguous muscles atrophy from disuse, local inflammatory invasion or reflex influence on the cord. The skin may become puffy,

glossy or atrophic. The reflexes are increased. Neuritis is not uncommon. The heart was not affected in any of Garrod's 500 cases. Anemia, episcleritis and other complications are uncommon. The majority of patients do not improve or recover and recurrence of the disease is very common. In some cases the process is arrested. Aside from atheroma and nephritis few intercurrent diseases develop.

2. Monarticular Type.—Pathologically, this type resembles the form described, but differs from it in that it presents more profound changes and is limited to one or few articulations. There is the same capsular thickening and cartilaginous fibrillation; the cartilage often softens, ossifies or hypertrophies; the bone rarefies or becomes cystic and soft in some places, with flattening of the bone heads and widening of the sockets, particularly in the shoulder and hip; in other places bony overgrowths form, known as Haygarth's nodes or a new socket may develop; the bone may eburnate and the joint may be either smooth or ridged. As a rule the atrophic prevail over the hypertrophic changes. They resemble the extreme senile change (*morbus coxæ senilis*); trauma or a nervous basis is considered causal; it occurs in old persons and largely in males. Clinically, compared with the general progressive type, it is single or at least rarely symmetrical, occurs in the proximal large joints (hip and shoulder), and is less often attended by subluxation. The elbow, spine and smaller joints are less involved.

3. Vertebral Type.—Strümpell (1885) named this type *chronic ankylosing spondylitis;* vertebral arthritis deformans occurs with ankylosis of the spine, shoulders and hips. Marie (1898) made it a separate type (*spondylose rhizomyélique*), characterized by complete vertebral ankylosis, scoliosis, ankylosis of shoulders and hips, exemption of all other joints, incidence almost exclusively in males, and its appearance in the young immediately after bony development has ceased. Von Bechterew (1892) described a similar type due to heredity, trauma or meningeal inflammation and characterized by paresis of the muscles of the neck, trunk and extremities, some atrophy of the back and shoulder, involvement of the spinal nerves (hyperesthesia, pain and paresthesia) and an ascending degeneration in the cord. These are probably subtypes of arthritis deformans.

4. Juvenile Type of Still.—(*a*) The ordinary progressive form may be observed in children, as in adults, and often with almost universal ankylosis; in some cases the great toes are lengthened. (*b*) "*Still's type*" occurs largely in poor children; 70 per cent. of cases are girls; 30 per cent. occur between the second and seventh years, 32 per cent. between the seventh and thirteenth years, and 20 per cent. between the thirteenth and seventeenth years. The onset is often subacute. The multiple arthritis affects chiefly the extracapsular tissues and there is seldom crepitus. The spleen is always enlarged and the lymph glands near the affected joints are hard and large. Anemia and retarded development are common.

5. Heberden's Nodes.—Bony nodes develop on the sides of the end phalanges near the joints. They begin between thirty and forty years, but are seen chiefly at about sixty years of age. At first soft

and sometimes painful and red, they become hard, indolent and as large as a pea. Their growth is more intermittent than continuous. Disturbance in function is not uncommon, but only in extreme cases is deformity observed, as flexion of the end phalanges into the palm or deviation toward the radial side. Involvement of the large joints is rather uncommon, whence the prognosis is favorable.

Diagnosis.—(*a*) In *gout*, the history, localization, fine (not coarse) crackling in the knee-joint and radial articulation of the elbow, greater spontaneous pain, infrequent generalization, and symmetry, the uratic deposits and tophi in the ear are characteristic. (*b*) *Acute rheumatism* (*v. s.* ACUTE FORM). (*c*) *Multiple tuberculous arthritis* may cause diagnostic difficulty, particularly in children, for pulmonary tuberculosis may complicate arthritis deformans; but the tuberculous hip and knee involvement, the fusiform fingers, the hectic fever, involvement of the glands and serous membranes are generally distinctive. Poncet's ankylosing tuberculous pseudorheumatism may cause confusion (*v.* p. 182).

FIG. 80.—Heberden's nodes. (Musser.)

(*d*) *Syphilis* (*q. v.*) of the joints occurs particularly in persons between four and fifteen years of age and in about 40 per cent. of hereditary forms; the epiphyses are irregularly deformed. (*e*) *Hemophilic* arthritis is distinguished by the family history and its three stages, hemarthros, panarthritis and adhesions, erosions, deformity and ankylosis. (*f*) *Chronic articular infections*, especially subchronic gonorrheal arthritis (*q. v.*), begin acutely, usually affect the knees first, then possibly after months the ankles, hands (in women particularly), sternoclavicular and other joints; similar chronic arthritides may follow scarlatina, pneumonia, tonsillitis (*v. s.*), etc. (*g*) *Nervous arthropathies* exhibit much the same pathology; (i) the arthropathies of *tabes* (*q. v.*) occur rapidly and early, without pain, tenderness or other inflammatory evidence; are attended by much effusion, are atrophic or flail-like rather than hypertrophic, in 80 per cent. involve the lower extremities and are accompanied by other signs of tabes. (ii) In *syringomyelia* (*q. v.*) the effusions are great, painless, in 80 per cent. involve the upper extremities and are accompanied by panaritia and dissociated anesthesia. (*h*) "*Chronic rheumatism*"

rarely follows acute rheumatism; the changes involve less the bone, cartilage and serous sacs than the capsule which becomes fibrous. The cartilage may become somewhat fibrillated and fibrous, but is seldom hypertrophied or destroyed. The vascular bands become callous (Jaccoud's fibrous rheumatism) and ankylosis may result. Some maintain that ankylosis by connective tissue and the joint obliteration differentiate fibrous rheumatism from arthritis deformans, but clinical differentiation is often impossible.

X-ray examinations detect the synovial or periarticular changes (the so-called rheumatoid arthritis), the atrophy of bone or cartilage and the hypertrophic alterations (osteo-arthritis).

Treatment.—Arrest of the process results rather from spontaneous exhaustion of the disease than from therapeutic efforts. (*a*) Possible causal conditions should be corrected, as pyorrhea, tonsillar or antral disease, otitis or genito-urinary infections. Autogenous *vaccines*, in the writer's hands, have proved futile. (*b*) *"Rheumatic" influences*, as exposure to cold and dampness, overheating while at work and resting in damp garments are to be carefully avoided. Woolen underwear should be worn. General hydrotherapeutic measures are illy borne. A warm climate benefits especially those cases resembling (or including) chronic fibrous rheumatism, as Mexico, West Virginia and Arkansas. (*c*) *Diet* is not of such importance as in gout; liberal feeding always is strongly indicated. (*d*) In *acute exacerbations* rest, salicylates and coal-tar derivatives are indicated. (*e*) *Passive movements* and *massage* relieve stiffness and muscular contractures. Massage reduces the edema and adhesions. In some cases forcible pressure, as with the thumb, will cause absorption of the fresh osteoplastic nodes; it may be easily overdone, and once every day or two on the same area is sufficient. Cautious active exercise is essential. (*f*) *Extension* by pulley weights sometimes produces wonderful results, according to Hoffa, in lessening pain and exostosis, though the author has never observed any benefit. (*g*) *Local measures.* The *x*-rays, local applications of warm water and strong electric lights may afford temporary alleviation from pain but do not seem to affect the basic change; fibrous rheumatism is more benefited than arthritis deformans. *Hot air* causes hyperemia of the parts and is sometimes of real benefit; cages of wire, asbestos and felt are devised to enclose the joint, leaving around it an air space; the temperature is gradually raised by a lamp placed under a connecting elbow to 200° or 250° F. and then gradually lowered. *Bier's* method of *venous hyperemia*, used in tuberculous joints, is also advocated; the Esmarch constrictor above the joint should produce swelling but not pain. "Firing" the skin with the Paquelin is beneficial in lighter grades. *Operation:* resection may be considered in non-progressive, monarticular forms, but the general poor health and age of the patient rather contra-indicate it. (*h*) *Internal medication*, as cod-liver oil, arsenic, thyroid extract and syrup of iodide of iron may help a few cases. Luff uses guaiacol carbonate; the initial dose of five grains, t. i. d., is increased two grains every week till a single dose of twenty grains is given.

DISEASES OF THE BONES.

For hypertrophic pulmonary arthropathy, leontiasis ossea and osteitis deformans, see page 722.

OSTEOMALACIA.

Etiology.—Osteomalacia is a chronic nutritive disorder, characterized by decalcification and progressive softening of the bones, with bending, fracture or deformity. It occurs especially in adults and women (90 per cent.), particularly nursing or pregnant multiparæ.

Pathology and Symptoms.—The pelvis, vertebræ and ribs are oftenest affected, with pain which is considered "rheumatic," disturbances in walking, etc., and deformities. The osteomalacic pelvis, often first detected by a pelvic examination during pregnancy, exhibits an approach of the sacral promontory and the acetabula; scoliosis, lordosis or kyphosis develops; the bones of the leg curve and spontaneous fractures occur. The flaccid and atrophic muscles exhibit tremor or fibrillation. Radiographic and autopsy examination reveals decalcification in the spongy and compact bones, with replacement by vascular, gelatinous, perhaps cystic or hemorrhagic tissue. The urine contains an excess of calcium and phosphorus.

Prognosis and Treatment.—The chronic, usually progressive, course covers five to ten years; it is sometimes acute or at times arrested. Asthenia or intercurrent disease is the usual cause of death. Calcium and phosphorus are beneficial. That the disease may be related to the ductless glands, is shown by occasional cures by ovariotomy in advanced cases and by full doses of adrenalin (Bossi).

ACHONDROPLASIA.

Pathology.—From unknown causes, perhaps from pituitary or thyroid disease, there is a dystrophy of the epiphyseal cartilages (chondrodystrophia fetalis); connective-tissue invasion from the periosteum prematurely unites the diaphyses and epiphyses, thereby preventing development of the long bones. The intermediary cartilage is narrowed, irregular and zig-zag and stray nests of cartilage cells may develop into chondromata, exostoses or rapidly growing malignant osteocartilaginous tumors. The *x*-rays show the small or deformed epiphyses.

Symptoms.—In achondroplastic dwarfism, intelligence is normal, the head and trunk are nearly normal but the decreased stature (3–4 feet) is due to the short extremities. The humerus and femur may measure less than the tibia and ulna. The fingers are nearly equal in length and diverge. The hand is short and the fibula is longer than the tibia. The pelvis is contracted and lumbar lordosis follows tilting forward of the sacrum. The root of the nose is depressed.

FRAGILITAS OSSIUM.

Ostheim (1914) collected 193 cases, "including two types of cases, (1) the fetal type found either at birth or soon afterward, associated with imperfect bone formation and multiple fractures (imperfect osteogenesis or periosteal dysplasia), these cases commonly not surviving beyond the second year of life; and (2) the congenital type, found in infants and children, at birth or later, with fractures recurring often until puberty or even until adult life, associated with similar bone changes (idiopathic osteopsathyrosis), which children if well cared for, survive, but often in a badly crippled condition."

The bone trabeculæ are produced by calcification of the cartilage cells, whereas normally new bone is produced by osteoblasts on a cartilaginous matrix, the periosteum forming imperfectly laminated, dense bone with large spaces; the bones contain little calcium. Fractures occur in the diaphyses only. The fetus exhibits imperfect cranial ossification, apparently shortened, bowed extremities and many fractures.

OXYCEPHALY.

Steeplehead, thürmschädel, tête à la Thersite, caput turritum, is usually congenital and is due to early synostosis, especially of the coronal and sagittal sutures, whence the head grows sloping upward to compensate for the lack of growth laterally and anteroposteriorly. The *x*-rays show a dimpling of the inner table, considered characteristic by Fletcher, who collected under 90 cases (1910). Intelligence is normal, the eyes are prominent or actually exophthalmic and headache and optic neuritis or atrophy are usual. Decompressive operation is indicated.

SECTION XI.

INTOXICATIONS. SUNSTROKE.

ALCOHOLISM.

I. Acute Alcoholism.—(*a*) The *highest brain centres* are first affected, judgment (emotion predominating), motility next (disordered speech, gait and vision), then the lower centres (respiratory, circulatory and spinal). (*b*) The pulse becomes more rapid, though alcohol does not stimulate the heart. Full doses make the pulse slow and small. The face is flushed and the vascular tone is lax; for this reason alcoholics suffer little shock from traumatism. (*c*) *Respiration* is slow, deep, seldom stertorous. (*d*) The *pupils* are more often dilated than narrow and reactionless. (*e*) The *temperature* may fall markedly, even as low as 75°, from vasoparesis, usually with cyanosis and failing of the bulbar centres. Involuntary evacuations occur in the severest cases.

Dipsomania is intermittent, acute alcoholism, an hereditary psychosis in which, after intervals of abstinence and no craving for liquor, the subject becomes intoxicated.

Diagnosis.—The alcoholic breath is usually suggestive but epileptics and uremics while drunk may suffer skull fracture or apoplexy. Many mistaken diagnoses result from carelessness, but many are unavoidable; the apparently drunk patient should be treated for alcoholism, while other possibilities are carefully considered. As a rule the patient can be aroused, but in "dead drunks" this is not the case and incomplete coma may occur from other causes. The pupils are usually dilated in alcoholic, contracted in opium and uremic, and unequal in apoplectic, coma. Stertorous breathing is strongly indicative of apoplexy or uremia. Convulsions following delirium and muscular twitchings are less common than in other similar conditions, but at the time of writing the author observed uni- and then bilateral convulsions. The urine rarely contains alcohol, as at the most 5 per cent. of ingested alcohol is excreted by the lungs and kidneys. Sudden death may occur without adequate necropsy findings. One-third of all fatal poisonings are acute alcoholism.

Treatment.—Gastric lavage is sufficient in most cases. The convulsions, when severe, yield to a few whiffs of chloroform. Acute mania yields to a hypodermic of apomorphinæ hydrochloridum (gr. $\frac{1}{6}$). Collapse, cold extremities and cardiac and respiratory failure are relieved by hot coffee, strychnine, atropine, aromatic spirits of ammonia and local heat (*v. i.* Acute Opium Poisoning).

II. Chronic Alcoholism.—This is less an hereditary tendency than a lack of character, though Plutarch said that drunkards beget drunkards. Some people can take beer, wine, spirits or patent medicines throughout life without apparent injury; in others, even the mildest alcoholic drinks may induce visceral alterations, so that as in liver cirrhosis, it would seem that there is some other etiological factor. Alcohol in health is a bad food, an injurious narcotic or stimulant, a protoplasmic poison to the parenchymatous organs and arteries, and a check on metabolism.

Symptoms.—(*a*) *Nervous manifestations.* Functional changes include the alcoholic tremor, a most common symptom; mental dulness and weakness; nervousness, jumping from slight stimuli, irritability, impaired judgment and feeble will-power; neuralgias and muscular rheumatism. The alcoholic is suspicious, cruel, conceited and degenerate. Quinquad's sign is elicited by having the patient place his fingers vertically to the examiner's hand or body, when a series of jerking shock-like movements is noted; (i) if it is absent, alcoholism is absent; (ii) if it is moderate there is uncertainty and (iii) if it is marked, in 3 cases out of 5, the patient is an alcoholic. Of organic nervous manifestations, multiple neuritis is the most common; it may result from small amounts of alcohol, when there is a coincident metallic poisoning; epilepsy may result from alcoholism; hemorrhagic pachymeningitis, chronic leptomeningitis, acute polioencephalitis, degeneration in the anterior horns or Clark's columns, degeneration of the cerebral nervous tissue, slight optic neuritis and even immobile pupils may be more or less direct sequences. (*b*) *Circulation.* Arteriosclerosis often develops with its train of sequences, cardiac hypertrophy and dilatation, angina pectoris, cerebral softening or hemorrhage. (*c*) *Chronic bronchitis,* emphysema, chronic pharyngitis and laryngeal thickening are frequent collateral findings. (*d*) *Digestive organs.* Alcoholic gastritis (*q. v.*) is common; the quite characteristic vomitus matutinus is quieted only by another dose of its cause. Beer drinkers usually have large stomachs. Alcoholic cirrhosis has been discussed; fatty degeneration, arterial congestion and hypertrophy may antedate or accompany cirrhosis. Beer drinkers have lax bowels, while consumers of spirits are constipated. Hemorrhoids are common. (*e*) The *kidneys* undoubtedly suffer. Dickinson, Formad and Pitt think that renal disease is less common than is usually believed; the kidneys are hypertrophied without other change in 43 per cent.; several factors may be necessary to produce indurated kidneys. Sterility is common. (*f*) *Metabolic maladies,* as gout, obesity and some forms of diabetes may result from chronic alcoholism. (*g*) The *skin.* Acne rosacea is a very common but not, as Trousseau says, an "indelible stigma." The vessels of the nose, cheeks and other parts are suggestively dilated; the conjunctivæ are injected and the eyes water.

Prognosis.—The prognosis is unfavorable, both from the standpoint of curability and complications. English life insurance companies estimate that the risk is 33 per cent. less in abstainers than in alcoholics. The general resistance is lowered, thus favoring infections, as tuberculosis, and raising greatly the death-rate, most notably in pneumonia. Twenty per cent. of suicides are alcoholics. The offspring of inebriates are disposed to numerous nervous affections.

Treatment.—Treatment consists of correction of the habit and care of the complications, the latter of which have, in great part, already been considered. The cure of the habit is largely personal. Its prophylaxis is better than its cure. No person, however intellectual or strong-willed, can safely drink with any regularity. Institutional treatment is generally necessary. The vomiting should be treated as in acute and chronic gastritis; lavage is probably the most reasonable measure; a mixture of bismuth (page 55) and small doses (♏i–ij) of Fowler's solution and tr. nucis vom. (♏x) are valuable just before the administration of hot milk with tr. capsici, ♏x. Hyoscinæ hydrobromidum (gr. $\frac{1}{120}$) should be given two or three times at three-hour intervals, chloral hydrate (gr. x) and sod. bromide (ʒss) for three or four doses, before bedtime, or sulphonal (gr. xx) in hot milk before bedtime, in order to induce sleep. (See Lambert's treatment, page 964). Sipping ice-water, sucking lemons or drinking water with tr. quassiæ may satisfy the desire to "drink something." Cocaine and morphine are avoided, lest a substitution or succession of habits develop. Relapse is very common.

III. Delirium Tremens.—*Mania a potu* is an acute accident of chronic alcoholism and almost never follows an acute debauch in an otherwise temperate individual. It was noted among sailors by Sutton, of Greenwich, in 1813, but was probably best described by Ware, of Boston, in 1831. Sixty-six per cent. of cases occur in men, while multiple neuritis is more common in women. The patient, usually between thirty and forty years of age, has been, on the average, an alcoholic for eight years before delirium tremens develops; strong spirits, rather than beer or wine, are the cause, as they are of alcoholic convulsions. It may be precipitated by trauma to the chest, fractures, mental shock, sudden failure of the stomach to assimilate food, acute withdrawal of alcohol or, in 70 per cent. of cases, the shock of onset of an acute infection.

Symptoms.—It develops with restlessness and insomnia, which renewed drinking fails to allay; for a day or two the mind wanders, but can be brought back to realities. The patient may be facetious, but is soon disturbed by horrible hallucinations. There is "great versatility of ideas." The patient talks volubly, turns suspiciously to the head of his bed, drives his horses vigorously and brushes away the vermin from the coverlet; hallucinations of hearing are also common. He at first knows that the bugs and snakes are unreal, but they soon become actual torments, so that unless watched he will leap from the window after waiting for his opportunity. The pulse is rapid and soft, the skin is flushed or slightly cyanotic and covered with sweat, the tongue is tremulous and white, the urine scanty and often albuminous and sleep is constantly absent. There may be moderate fever, 101° to 103°.

Diagnosis and Prognosis.—The *diagnosis* is rarely uncertain. The chief danger of error is in overlooking coincident trauma, and infection. In heavy and constant drinkers, a delirium may be delirium tremens or an associated delirium, as from pneumonia.

The *outlook* is always uncertain. Early convulsions are of no great moment. The prognosis is determined by the pulse, general nutrition

and complicating alcoholic or other lesions. Rigidity of the muscles of the neck is ominous and indicates edema of the brain. The average mortality is 15 to 20 per cent., but varies as the affection is (*a*) uncomplicated (1 per cent. die, Lambert); (*b*) associated with other affections (grave outlook) or (*c*) occurs in the aged or debilitated (probably fatal outlook). Recurrence is frequent, 27 attacks being recorded. In fatal cases death usually results from exhaustion and heart failure; the tongue becomes dry, the delirium constant and the skin cold and clammy. The autopsy shows edema of the brain ("wet brain," Dana), degenerative changes in the brain and cord (Bonhöffer), arterial alteration (Collins) and hypostasis of the bases of the lungs.

Treatment.—(*a*) The patient should be *kept in bed* and watched constantly lest he escape. Strapping the ankles and wrists and confinement by a straight jacket is a harsh procedure and invites trauma to the hands and feet, but is inevitable when the family is indulgent or the nurses are negligent. (*b*) *Withdrawal of alcohol* is usually advocated but is a severe measure; the temporary continuance of moderate doses seems to steady the pulse and favor digestion. (*c*) *Sleep is induced* with great difficulty and, as Ware first pointed out, comes when the disease runs its course. Excessive medication should be avoided. Sod. bromide (℥ss), tr. capsici (♏v) and whisky (℥ij), in hot peptonized milk should be given every three hours, by mouth if possible, or by the nasal catheter, for nutrient enemata are difficult to give and are seldom retained. Chloral is always dangerous in inveterate alcoholism. Möller administers veronal, gr. xv, repeated once. Hyoscine with strychnine may be beneficial. Morphine should be given only by the hypodermic method, for its absorption can then be measured.[1] Protracted warm baths may quiet the patient but they are hard to administer. In 6 cases in which the violent delirium became ominous the writer etherized the patient, and in three instances with excellent results. (*d*) The *circulation* should be treated as in pneumonia. Large doses of digitalis are tolerated only

[1] In the treatment of 500 cases of delirium tremens, S. W. Ranson's conclusions are as follows: (1) In incipient cases the patients respond readily to treatment with chloral, ergot, bromides and whisky. (2) Delirious patients are very resistant to treatment. Sedatives increase the mortality (scopolamin, 13 per cent.); 15 to 30 grains of chloral in twenty-four hours may be given with good results, but larger doses increase the death-rate. (3) The only drug which reduced the mortality was ergot; the death-rate was reduced 21.6 per cent. (4) When whisky was given the mortality was increased 1.8 per cent. Ranson's cases were divided into two classes, incipient and delirious. The incipient cases showed only insomnia, restlessness and tremor. The fully developed cases were noisy, delirious, and had well-defined hallucinations. Of the sedative drugs the bromides were most extensively used and severe cases showed a mortality of 45.5 per cent.; of incipient cases treated with bromides, only 32.6 per cent. became delirious; while of similar cases not treated with bromides, 52.8 per cent. became delirious. Chloral is of no value after active delirium has set in; chloral is of great value in the treatment of incipient cases; chloral is superior to bromides because of the greater speed of its action. Morphine has no effect in delirious cases and is far inferior to small, repeated doses of the sedatives used in the incipient cases. Scopolamin, $\frac{1}{100}$–$\frac{1}{50}$ of a grain, in incipient cases has no controlling action, and proved extremely dangerous when given to actively delirious cases. In those cases in which ergot, fluidextract, in dram doses, repeated every four hours was used the mortality was only 30 per cent., while among those patients treated without ergot it was 51.6 per cent. Ergot is of value in the incipient cases, the percentage of patients developing delirium being reduced 23.3 per cent. by its use, possibly by decreasing the cerebral hyperemia. Whisky is of use in the treatment of incipient cases, lowering the percentage of patients becoming delirious by 20.2 per cent. In the delirious patients it is useless.

because they are not absorbed; the original treatment by digitalis is based on the fact that a nurse by mistake gave a toxic dose of digitalis; the patient improved and the "digitalis treatment" was thus instituted. (*e*) *Cold affusions* upon the neck and chest are recommended by Broadbent. (See Lambert's treatment, page 964.)

OPIUM POISONING.

I. Acute Poisoning.—This is important chiefly from a diagnostic standpoint. In the second stage it is strongly characterized by profound sleep, dry, flushed skin, narrow pupils, slow, strong "digitalis pulse" and slow, deep, stertorous breathing. The third or terminal stage is different; the skin becomes cyanotic and clammy, the narrowed pupils may dilate, the pulse becomes weaker (though the heart centres live after the other bulbar centres fail), respiration is very slow and halts. Mistakes in diagnosis are made by attributing undue importance to the "pin-point" condition of the pupils. Morphine may be detected in the stomach washings.

Treatment.—This consists of (*a*) *evacuation* by repeated gastric lavage. (*b*) *Chemical antidotes*, such as large amounts of tannic acid, should be given with the lavage and removed, as the tannates formed may dissolve; 1 to 1000 permanganate of potash (Moor) may be given by mouth or hypodermically. (*c*) *Respiration* must be sustained by walking the patient, but *fatigue must be guarded against;* phrenic faradization is indicated, but care is necessary lest too strong a current produce paralysis instead of stimulation; strychnine plus atropine may be given hypodermically, but the total dose of atropine should not exceed gr. $\frac{1}{30}$; pulling slowly and rhythmically on the tongue may help respiration; artificial respiration and oxygen inhalation may be used. (*d*) *The heart* is stimulated by coffee, strychnine, heat and digitalis.

II. Chronic Morphinism.—The use of morphine for pleasure only, so common in Asia, is rare with us; those taking morphine for pain and resisting its attractions are called morphinists; those using it for pleasure only are morphinomaniacs. The habit is contracted by its use for insomnia or for mental or physical pain. Forty-seven per cent. of morphine victims are said to be physicians; women, particularly prostitutes and nurses, druggists and—in Germany—officers rank next. It may be taken for years without mental or physical deterioration, but increase in the daily dose becomes necessary and the half-oxidized alkaloid produces symptoms which are only allayed by larger doses. Morphine acts as a nerve poison. The victim becomes restless and irritable without his dose. The mental and moral attributes are anesthetized and there is a "moral insanity." The patients lie by choice, neurotic symptoms are common, muscular and mental asthenia develop, the pupils are contracted, the sweat and saliva are decreased, the teeth decay, the appetite fails, the skin becomes sallow, itchy and dry, the hair coarse and gray, constipation is usual and slight ataxia may develop.

Prophylaxis.—Physicians should not give opiates freely. When administered for severe pain the habit is less often contracted than when given to neurotics. At least 20 per cent. of cases relapse after a "cure."

Treatment.—Treatment is almost impossible without (*a*) *institutional confinement,* which must of course be voluntary. The patient will obtain morphine if possible. (*b*) The *morphine* may be *withdrawn* abruptly, rather suddenly or very gradually; the second method is the best, stopping the morphine entirely in four to seven days. Its withdrawal is attended by awakening of dormant anesthetized functions; there is at first paroxysmal yawning and sneezing; roughening of the voice, paresthesia, neuralgias especially in the legs, clammy skin or rapid pulse, wide pupils, tremor, sexual excitement, extreme irritability, vomiting, colic and diarrhea also occur. The symptoms were attributed by Marme to di-oxymorphine, the antidote to which is morphine; Hitzig believes that they are largely due to hyperchlorhydria, for the gastric nerves under the influence of morphine secrete little hydrochloric acid; relief is obtained by washing out the stomach with a solution of Carlsbad salts. (*c*) The *patient should be kept in bed* for a week to obviate vasoparetic cardiac collapse. (*d*) *Symptomatic treatment* is indicated (i) by the *collapse* (aromatic spts. of ammonia, ♏xx, strychnine, gr. $\frac{1}{30}$, heat); (ii) by *nervousness* (warm baths, sodium bromide in doses of one dram every four hours); (iii) by the *insomnia* (hyoscine, gr. $\frac{1}{120}$ every two hours for two to four doses, sulphonal or trional, gr. xx); (iv) by the *gastralgia* (gastric lavage with alkaline salts); (v) by *diarrhea* and colic (as in enteritis, but vegetable astringents, large doses of bismuth, and no opiates should be used); (vi) by *neuralgia* (acetanilide, gr. v, fluidextr. gelsemii, ♏iij). Whatever treatment is instituted, alcoholism, chloralism, bromism or cocainism should not be substituted. Alexander Lambert's treatment aims to eradicate the craving for narcotics.[1]

[1] Lambert's prescription consists of:

℞—Tincturæ belladonnæ (15 per cent.)	20 c.c.		fl ʒv
Fluidextracti hyoscyami	10 c.c.	or	
Fluidextracti xanthoxyli	10 c.c.	āā	fl ʒiiss

M. et Sig.—Administer as directed.

He emphasizes the necessity of carrying out his treatment absolutely to the letter, and employing profuse catharsis. In *alcoholics* 6 to 8 drops are given every hour, day and night, until either the patient shows symptoms of belladonna excess, or from the cathartics the characteristic stool. The dose is increased by 2 drops given every six hours until 14 to 16 drops are taken. Usually an alcoholic can be given four compound cathartic pills at the same time that the specific is begun. After the drops have been given for fourteen hours, a further dose of compound cathartic pills is given, either two or four, depending upon the amount of action obtained from the previous pills. If the first pills have caused abundant action, only two are now necessary. At the twentieth hour of the drops, from two to four more compound cathartic pills are given, and after these have acted, the stools should begin to be green. An ounce of castor oil should now be given, and a few hours later the characteristic thick, green, mucous, putty-like stool will appear. Usually the drops must be continued, and at the thirty-second hour from two to four compound cathartic pills are again given, and a few hours later the castor oil. The drops can now be discontinued. In the first twenty-four hours with an older patient, or one in the midst of a spree, whisky should be given four or five times, in one- or two-teaspoonful doses, in milk. The whisky should not be continued after the first twenty-four hours, and in younger patients it is usually not necessary to give it at all. An alcoholic is less tolerant of belladonna than a morphine patient, but if they are sensitive to this drug they will show it in the first six or eight hours of the treatment. After the patient has been through with this treatment the desire for alcohol has ceased, and it is a question for the next few days of proper nutrition, and sometimes the administration of a drug for sleep. He finds the best hypnotics for these alcoholics are chloral and bromides. Whatever tonic is given of course must be non-alcoholic. In treating *morphinism*, patients do not suffer more than a bearable amount

LEAD POISONING.

Etiology.—The importance of plumbism or saturnism is increasing. (*a*) *Industrial plumbism* is very common. Workers in white- or red-lead factories usually acquire the disease. Miners of the metal generally escape, but smelters of lead ore and even animals and birds near the furnaces are frequently affected. Painters, plumbers, glaziers, less often tinners, printers, cameo polishers, cartridge makers, etc., may be poisoned. (*b*) *Accidental contamination of food or drink.* Poisoning may result from the action of acids in canned foods on the tin or solder, as in cans of sardines; wine or cider may become poisonous in the same way, as happened in Devonshire and Poitou (*colica pictonum*). Chromate foil, covering hams or candy, rubber nipples, bullets in pickled game, bread ground in lead vessels, chromate used to color buns (D. D. Stewart's report of the fatal Philadelphia epidemic), Seltzer or other charged waters, wine and *eau de vie* sweetened or cleared with litharge, are a few of the many subtle causes of saturnism. Drinking water conveyed through lead pipes is generally protected by the deposit of lime, and other salts, but poisoning is possible when the water is exceptionally pure or soft, thereby depositing little lime in the pipes, or when it is saturated with organic matter or collected from lead roofs. (*c*) Less frequent sources of plumbism are in the environment, and in hair-dyes, powders or cosmetics, linen, freshly painted walls, candles, sealing wax, brightly tinted toys, false teeth and thread. (*d*) *Therapeutic* and *criminal plumb-*

of discomfort in breaking away from the drug. First, the patient is made comfortable with his accustomed dose of morphine, and then given four or five compound cathartic pills. Then the drops should be given as prescribed for alcoholics. If there is a hypersensitiveness to belladonna the drops must be stopped and later begun again with a smaller dose. After the drops have been started and the time has come for the patient's usual dose of morphine, he receives from one-half to two-thirds of the kind of opium that he has been taking, and administered in the manner that he has been administering it. After fourteen hours of the drop treatment, the patient should be given four compound cathartic pills and five grains of blue mass, or some other form of vigorous cathartic such as the vegetable cathartic pills of the Pharmacopœia with ginger and capsicum and $\frac{1}{2\frac{1}{5}}$ of a drop of croton oil to each pill. Four or five of these last pills should be administered, if preferred to the compound cathartic pills and blue mass. After the twentieth hour of the drop treatment the patient should receive four or five more of the vegetable cathartic-ginger-capsicum-and-croton-oil pills and blue mass, and, if these do not quickly act, they should be followed by four or five compound cathartic pills and then an ounce of Epsom salt or Hunyadi water every half-hour for four or five doses. If in an hour or two these do not act, four or five more of the vegetable cathartic pills, followed by the salts, should be persisted in until the bowels act. Lambert states that this cathartic treatment will appear to be extraordinary, and it is surprising that the withdrawal of the morphine does not of itself produce a diarrhea. The inhibition can only be due to the atropine. If by the twentieth hour the cathartics do not act the symptoms of the reduction of the morphine, viz., sneezing, nervousness and pain, come on in full force, and the patient begins to suffer. If the cathartics do act well, a second dose of morphine or opium should be given of about one-third or one-sixth of what was given as the initial dose at the beginning of the treatment. Twelve hours after this second dose of morphine or opium the patient should again have four of the vegetable cathartic pills, each with ginger and capsicum and $\frac{1}{2\frac{1}{5}}$ of a drop of croton oil, or four compound cathartic pills with the 5 grains of blue mass, and following this the stools will begin to be green. After the liquid green stools have occurred following the thirty-second hour cathartic, an ounce of castor oil should be administered, and will cause the thick, green stool as described under alcoholic treatment. After this movement, the patient feels suddenly relaxed and comfortable and the nervousness ceases. Most patients then go to sleep. After the thirtieth hour of such treatment, and he well says earlier if the patient is in a weak condition, stimulation with strychnine, from $\frac{1}{60}$–$\frac{1}{30}$ of a grain, is advisable.

ism is rare; it may result from the acetate of lead when given internally or externally, and from impure subnitrate of bismuth; its criminal use is very uncommon. (*e*) *Predisposing factors* are alcoholism, age (from thirty to forty) and sex (75 per cent. are females, Olivier). Susceptibility varies greatly; plumbism may cause death within a week or may not develop until after forty-two years of work in the metal (Tanquerel des Planches, 1838). *Absorption of lead* usually occurs (i) by the digestive mucosa; most of the metal is precipitated by the albumin of the food and eliminated without absorption; (ii) by the respiratory mucosa, by inspiration, as dust in white-lead factories; or (iii) by the sound or diseased skin (cosmetics) or even the conjunctiva or vagina (lead douches). It is *eliminated* chiefly by the urine; Putnam found lead in 25 per cent. of 150 persons who had no evidence of plumbism; the liver and alimentary mucosa eliminate smaller quantities.

I. Acute Poisoning.—This is uncommon; its symptoms are (*a*) gastro-intestinal; a styptic, burning taste occurs in the mouth, thirst is present, there is a lead line on the gums in some cases, though less than in chronic poisoning, nausea and vomiting sometimes of white-lead chloride, abdominal colic, retraction of the abdomen, diarrhea with black (lead sulphide) or bloody movements or sometimes constipation. On post-mortem examination, the catarrhal gastro-enteritis may be wrongly considered a postmortem change; Orfila's white spots may be seen. (*b*) Under circulatory symptoms, the pulse may be slow (50 to 20) and irregular; anemia, cyanosis and collapse may occur; (*c*) nervous symptoms embrace vertigo, stupor, delirium, neuralgia, cramps, convulsions and even cerebral and peripheral paralyses; (*d*) nephritis with suppression of urine may develop.

Treatment.—Treatment consists of full doses of sodium and magnesium sulphate, in order to precipitate an insoluble lead sulphate, and to remove it, demulcent drinks (eggs and mucilages), opium for pain and cardiants.

II. Chronic Poisoning. — Symptoms. — (*a*) *Colic*, the most common symptom, occurred in 88 per cent. of Tanquerel's 1390 cases. It is due to local deposit of lead in the intestines, spasm of the bowel, changes in the nerves or angiospasm. It may develop early or late; it may be preceded by malaise, lumbago, pain in the legs or alcoholic excesses. It is accompanied by constipation (dry colic), rarely by diarrhea; first umbilical, it spreads over the abdomen, perhaps to the legs or scrotum; it is relieved by pressure, though sometimes the abdominal wall is hyperesthetic; vomiting and absolute constipation may simulate obstruction, and the author observed intussusception as a result of lead poisoning. There is no fever, the pulse is small and hard and the urine is scanty. Without treatment colic is protracted; its relief is usually marked by a bowel movement. Relapses are very frequent. (*b*) The *gingival lead line* (Burton, 1834), present in over 90 per cent., is a gray-black line, 2 to 3 mm. wide, seen most clearly on the lower gums and due to deposition from the blood of lead sulphide, precipitated by the sulphuretted hydrogen of the tartar. The coincident anemia brings it out very clearly. Similar deposits may occur under the buccal mucosa opposite the molars. It is

very frequently mistaken for tartar, which occurs on the teeth, sometimes for the black carbon line seen in miners, or rarely for the dark line in argyria or bismuth poisoning. (*c*) *Other digestive symptoms* are coated tongue, fetid breath, parotitis, saturnine dyspepsia and rarely icterus. Test meals show lactic acid and no HCl. (*d*) *Anemia* is very frequent in the "lead cachexia." The hemoglobin and erythrocytes decrease moderately; the red cells show basophile granules (Grawitz) and quite commonly nucleation, even in mild cases (Cadwalader, 1905). The leukocytes may be increased. (*e*) *Heart and vessels.* The heart is very often hypertrophied, as evidenced by a loud second aortic tone and aortic regurgitation of atheromatous origin; the arteries are often sclerosed, occasionally causing angina pectoris, frequently myocarditis, irregular heart action and perhaps some of the brain symptoms (*v. i.*). The asthma saturninum is cardiac or uremic. (*f*) The *kidneys* are often sclerotic, as a result of the elimination of the metal; the change does not begin around the vessels but in the tubules, which are the seat of a necrosing deposit of carbonate of lime. Nephritis, gout and plumbism may coexist. (*g*) *Nervous manifestations.* Peripheral degenerative *neuritis* occurs in 7 per cent., usually as a late symptom; one-tenth of these cases have not had colic. It is characterized by the paralysis of muscles with the same function, by muscular atrophy and cramps, tremor, cutaneous hyperesthesia, neuralgia, arthralgia (55 per cent.), myalgia and by varying degrees of reaction of degeneration. The musculospiral type, "*wrist-drop,*" is the most common and is bilateral; the supinator longus (a flexor) and the small extensor of the thumb escape involvement; tenderness and anesthesia are rare. Gubler's tumors are swellings of the extensor tendon sheaths, due to backward dislocation of the carpus or perhaps to a trophic change; they are hard, ovoid, indolent, seldom painful and regress with the paralysis. A second but rare form is the *brachial,* involving the deltoid, biceps, brachialis anticus and supinator longus, rarely the pectorals. A third type is the *Aran-Duchenne,* which is especially observed in tailors and involves the small muscles of the hand, the thenar and hypothenar eminences; it resembles that of chronic anterior poliomyelitis (which indeed has been found in a few cases at autopsy). A fourth type, the *peroneal,* involves the peronei and toe extensors; but 13 per cent. of lead palsies affect the leg. Lastly, rare types may involve the larynx, vagus and face; generalized paralyses, either slow or rapid like Landry's paralysis, are very rare; primary muscular atrophy may occur, most often of the Aran-Duchenne type.

Cerebral forms (*encephalopathia saturnina*) occur in 5 per cent.; they are often preceded by headache, colic and insomnia and are attended by amaurosis, limitation of the visual field, retinitis albuminurica or saturnina (with inflammation, engorgement and possibly ultimate atrophy) and paralysis of the eye muscles. The most frequent type is the *delirious* encephalopathy, which is variable or mobile in character, and sometimes resembles delirium tremens; the next type is the *convulsive* (epilepsia saturnina) which occurs without auræ, is usually generalized, often fatal and is always considered when epilepsy develops in an adult; the third type is the *comatose.* Hysteria frequently occurs with plumbic encephal-

opathy. Unrecognized cases may become dements (pseudodementia paralytica saturnina). Deposit of lead in the brain is the usual cause; sometimes the cerebral symptoms are arteriosclerotic (as hemiplegia) or uremic.

Lead poisoning is usually *diagnosticated* by the etiology, lead line, lead in the urine, colic and wrist-drop. In a case of long coma with choked disk, in the Cook County Hospital, no cause was found, until after a second examination, the lead line was seen. The man was a barber, who dyed the hair and probably absorbed the lead through his hands.

Prognosis.—The acute forms are more favorable. Vascular and renal changes are largely incurable. Atrophic paralyses are serious. The wrist-drop usually regresses, though it is sometimes permanent; 25 per cent. of cases with coma, convulsions and other cerebral symptoms die; after apparent recovery mental degeneration may develop.

Treatment.—(*a*) *Prophylaxis.* A mask should be worn in white-lead factories to avoid inhalation of lead dust; workers are careless and poisoning is frequent despite precautions. Painters and others may often avoid poisoning by carefully cleaning the hands and nails before eating. (*b*) *Lead colic* is treated by hypodermics of morphine, nitroglycerin and atropine to control pain and spasm. Spts. of chloroform (♏xx) with other aromatics may be given by mouth. (*c*) The *constipation* and removal of any lead salts unabsorbed in, or excreted by, the alimentary mucosa necessitate catharsis. Full doses of castor oil (ʒiv or more) with paregoric (ʒj) evacuate the bowels and relieve pain and spasm; sodium and magnesium sulphates purge and coincidently precipitate the lead as an insoluble sulphate. (*d*) *To eliminate the lead* from the tissues *potassium iodide* should be given. In acute or severe chronic cases it should be given *in small doses* (gr. iij–v), for it transforms the fixed tissue metal into free metal in the circulation; some writers obtain better effects from potassium bromide (gr. v–x). Warm baths and pilocarpine (gr. $\frac{1}{8}$) promote excretion by diaphoresis and plenty of water stimulates the renal elimination. (*e*) *Iron* (not arsenic) should be given for anemia. (*f*) For treatment of *neuritis*, see Multiple Neuritis.

ARSENICAL POISONING.

Acute poisoning has been considered under Pernicious Anemia.

Chronic Poisoning.—(*a*) *Criminal poisoning* is of great medicolegal importance, as in the unfortunate Maybrick case. (*b*) Poisoning by reason of occupation is uncommon. (*c*) *Therapeutic poisoning* is not common, but may occur in chorea, leukemia and pernicious anemia. (*d*) *Most cases are accidental.* The dyes in garments, toys, glazed paper used in kindergartens, wrappers, artificial flowers and wall paper are important sources of intoxication. The action of moulds (pencillium and mucor) may liberate volatile arsenical gases. Contaminated glucose, used in beer, caused the Manchester epidemic. Arsenic is absorbed by the lungs and alimentary tract and is chiefly eliminated by the kidneys, which may degenerate. Putnam found arsenic in the urine of 30 per cent.

of persons who showed no other symptoms of arsenical poisoning. Arsenic is widely found in plants, sea water and springs. Smaller amounts are eliminated by the bowel, milk and other secretions.

Symptoms.—The symptoms are (*a*) *gastro-intestinal,* as dry throat, vomiting, purging or colic; the "rice-water" stools contain flecks of mucus; and (*b*) *respiratory,* as dry cough or chronic bronchitis. (*c*) *Emaciation,* weakness, fever and anemia may occur. (*d*) *Cutaneous* symptoms, as erythema, keratosis, pemphigus, herpes and brown pigmentation and (*e*) *nervous* manifestations, as weak memory, vertigo, headache and multiple neuritis may develop. The neuritis has the same general features as the saturnine form, but sensory changes are more frequent and the legs are more often affected than the arms; paralysis of the peronei and foot extensors causes the "steppage" gait. The small muscles are less often affected than in plumbic or alcoholic neuritis; paralysis, ataxia, trophic disturbances and the reaction of degeneration occur as in other neuritides.

Treatment.—Treatment is that of lead poisoning or neuritis.

FOOD POISONING.

Bromatotoxismus (Vaughan) exists in several forms:

I. Meat Poisoning (*Kreatoxismus*).—Sausages and head cheese are more dangerous than beef or mutton. Sausage poisoning (botulism or allantiasis) was known in Germany a century ago. Van Ermingem isolated an anaërobic organism, the *Bacillus botulinus;* it contains spores, is flagellated and motile and grows only on alkaline media at a low temperature (18° to 35° C.); it is a saprophyte and causes symptoms by its toxins, which are not destroyed by the gastric juice. Paratyphoid organisms were found by Durham (1900). Meat may taste and smell normal yet contain ptomaines; their nature is still undetermined, though Cobert considers them promatropin. Canned meat has often caused severe symptoms. In instances botulism was caused by eating salad and beans.

Symptoms.—The symptoms of *botulism* appear in twelve to forty-eight hours, with nausea, vomiting and sometimes diarrhea. In the Wellback cases Ballard noted as early symptoms headache, chilliness or rigors and pains in the chest or back. Paralysis of the soft palate, larynx and esophagus rapidly develop and somewhat later paralysis of the bowel, or symptoms like atropine poisoning, mydriasis, ptosis, disordered vision, adynamia and weak heart action; the sensorium is clear and fever is unusual. The *mortality* ranges between 20 and 50 per cent.; death occurs in four to ten days and the autopsy shows parenchymatous degenerations and ecchymoses, but especially degeneration of the ganglionic cells, as those of the vagus or oculomotorius; if the patient survives, convalescence is established only after months.

The symptoms of other forms of meat poisoning are either *toxemic* (typhoid type) or *gastro-intestinal* (cholera type); the typhoidal symptoms, which appear after an incubation of four to six days, are fever, status typhosus and even roseolæ; the choleriform symptoms, which develop

in two to twelve hours, are vomiting, incessant purging, watery stools, clammy skin, collapse and cyanosis. Acute meningitis may be simulated exactly (A. D. Dunn). Mild forms occur in most epidemics.

Treatment.—*Early gastric lavage, colonic flushings* and *purgation* are indicated; calomel followed by castor oil is the best aperient. Otherwise symptomatic medication, as in cholera or typhoid, is indicated; *champagne, strychnine,* hypodermocylsis and opium may be given. The resemblance of botulism to the diphtheria toxins has suggested the use of antitoxine; its effects in animals have been remarkable, but to the author's knowledge it has not been used in man.

II. Poisoning by Milk.—Galactotoxismus produces symptoms analogous to those of the infantile affections of the bowel, as vomiting, bloody diarrhea, fever and collapse. In cheese poisoning Vaughan found tyrotoxicon and other more frequent toxins, probably albumoses. Similar ptomaines were found in refrozen ice-cream and custard. In Norway where tyrotoxismus is most prevalent, the colon bacillus has been repeatedly found. Home-made cheese is more dangerous than the factory product. Various forms of toxemia result; some are cholera-like and others resemble belladonna poisoning. Treatment is that of kreatoxismus.

III. Poisoning by Fish (*Ichthyotoxismus*) and *Shell-fish* (*Mytilotoxismus*).—(*a*) Ichthyotoxismus, described by Comby (1827), may be caused by poisonous fish or poisonous glands in certain fish, as the roe or testicles, the muscular parts being harmless; the barbel in Europe and the tetrodon, diodon and fugu in Asia are poisonous (*v.* BERIBERI). The poisoning due to putrefaction is more common. Paralytic symptoms, like curare poisoning, are most frequent. (*b*) Mussel poisoning (mytilotoxismus) may cause dyspeptic symptoms in mild infections and cholera-like symptoms or symptoms of belladonna poisoning in severe forms. Rashes are common. Brieger isolated a mytilotoxin, which is found largely in the liver. The danger lies in placing mussel- or oyster-beds near river mouths, so that they feed on poisonous excreta. In restaurants certain glands near the heads of lobsters, considered toxic, are carefully excised.

IV. Grain Poisoning (*Sitotoxismus*).—1. **Ergotism.**—Ergotism (Thuillier, 1830, and Tüczek, 1879) results from eating grain mixed with the ergot fungus (claviceps purpurea). Ergotized grain is found largely in bad harvests, resulting from wet springs and hot summers. Epidemics have occurred in Europe, and sporadic cases are not rare. *Acute ergotism,* with digestive symptoms, colic, cyanosis, weak heart and generally lethal outcome is less common than *chronic ergotism,* of which *two forms* exist; the first is the *convulsive* form, attended by paresthesia, lightning pains, headache, delirium, melancholy, dementia, muscular relaxation, generalized epileptiform attacks or local muscular contractures (flexion of the arms and extension of the toes and feet); it is said to result from the alkaloid cornutin. The *second form* is the gangrenous, due to sphacelinic acid and marked by localized gangrene, usually in the phalanges and less often in the tips of the ears and nose. Chronic ergotism is characterized by a number of *symptoms which suggest tabes,* as lightning pains, ataxia of station and movements, and lost knee-jerks; and in five

autopsies Tüczek and Siemens found degeneration with, later, sclerosis of the posterior columns; degeneration in the anterior cornua, and minute foci of softening or hemorrhage also occur. The clinical course is chronic and death is frequent from convulsions or cachexia.

TREATMENT.—Treatment includes avoidance of diseased grain and opium for convulsions and nitroglycerin for gangrene.

2. **Lathyrism** (*Lupinosis*).—Lathyrism is produced by meal made from vetches, chiefly the *lathyris sativus* and *cicera*. Irving described the disease in India and it has been seen also in Algiers and Italy. It probably produces a lateral and possibly some posterior sclerosis of the cord, though no autopsies are reported. Clinically there is a *spastic paraplegia* with increase of reflexes and some paresthesia.

V. Potato Poisoning.—Solanin is contained in potatoes in small amounts (0.06 per cent.) but larger amounts (0.4 per cent.) occur in potatoes which lie for some time in the ground or sprout in cellars (Bact. solaniferum). Fever, chills, general or cardiac weakness, vomiting, diarrhea and jaundice may develop.

PELLAGRA.

A disease with periodic alimentary disturbance, skin lesions and changes in the nervous system.

Etiology.—The older theory ran that maidism was a chronic nutritional or toxic affection due to the use of corn or maize, which was unripe, decayed or infected with the aspergillus or other moulds. The present tendency is to regard it as an infection. The disposing etiology is "peasant life, poverty and polenta" (corn). It is endemic in Italy, Roumania, France, Spain and Mexico and the United States where there were 30,000 to 50,000 cases in the six years before 1913. The first authentic description dates from Casal, of Spain (1735). The name is derived from pelle (skin) and agra (rough).

Prodromal symptoms develop in the spring and include lassitude, vertigo, headache, anorexia, coated tongue, vomiting, epigastric pain and diarrhea. There is seldom fever. The *vernal erythema* appears largely on the exposed parts, face, hands and neck, possibly initiated by the sun's rays. The circlet of eruption on the neck is very characteristic. The hands are most commonly and characteristically involved, the palms generally being exempt. The eruption is a dull red, like sunburn, fades on pressure and may become macular. The color then deepens to a livid blue, ending in desquamation and pigmentation. More rarely vesiculation, crustation, fissuring and suppuration develop. Recurrence occurs the next spring (in Italy) or the same fall (in the United States) and leaves the skin atrophic, parchment-like, perhaps ulcerated or ecchymotic.

Nervous Symptoms.—The prodromal symptoms (*v. s.*) intensify and the skin may become so hyperesthetic as to induce suicide. The patellars vary. Babinski's sign may be observed. Mental depression occurs in most cases; stupor, hallucinations, etc., may develop; 10 per cent. become insane. There are severe pains in the back but the most char-

acteristic symptom is the *ataxic paraplegia;* autopsies reveal a postero-lateral sclerosis, sometimes with atrophy of the anterior horns, lepto-meningitis and variable brain findings. In some cases mydriasis, cataract, paralysis of the eye muscles, optic neuritis and choroiditis develop.

Digestive symptoms commence in the prodromal stage. Stomatitis, the stipple-tongue (with black pigment), vomiting, pyrosis, meteorism and severe, even dysenteric, diarrhea increase the discomfort and lead to prostration and emaciation. There is slight anemia, with some increase of the large mononuclears, very few eosinophiles and no leukocytosis. The mortality is 35 per cent.

The rarer acute cases may resemble typhoid in symptoms and pathology (status typhosus and swelling with ulceration of the intestinal lymph structures), or meningitis (foci of acute meningomyelitis). Other findings at autopsy are atrophy of the digestive mucosa, parenchymatous degeneration and pigmentation, the latter of which is thought to occur from adrenal inflammation.

Treatment.—Treatment is prophylactic (proper storage of the grain); change of diet or of locality is indicated and arsenic should be given internally.

BERIBERI.

Definition.—Beriberi or *polyneuritis endemica* (Bälz, Scheube) is an endemic and epidemic affection of the tropics and subtropics, of unknown etiology, characterized anatomically by degenerative inflammation of the peripheral nerves, and clinically by motor and sensory disturbances, anasarca and cardiac disturbance. *Beri* signifies a sheep's gait, and *kakke* means disturbance of the gait.

History.—Beriberi is mentioned in the oldest Chinese writings and was observed in the Roman legions, 24 B.C., by Strabo and Cassius. In the seventeenth century, beriberi was noted in Brazil and the Malay archipelago; in recent times it has been described in India, Japan, the Dutch Oriental colonies and in this country.

Distribution.—The main foci of beriberi are (1) the Malay archipelago, Sumatra, Borneo and Java; also China, India, the Philippines, Japan and even Australia. The mortality was very high among the Chinese coolies in the Dutch possessions. (2) Africa, including the mainland and adjacent islands. (3) America and the West Indies, Brazil, Honduras and Cuba. Scheube doubts that the cases in the epidemic at Tuscaloosa, Alabama, were beriberi, but they apparently were Oriental beriberi. Cases appeared in Dublin (1894–1898, with forty-two deaths), in England and in Paris.

Etiology.—There are two distinct views: (1) That it is an acute infection (Bälz, Scheube); the same telluric conditions promote beriberi which promote malaria. In 80,000 cases among the Japanese troops in the Russo-Japanese War, Kokubo frequently found a coccus. (2) The second view is that beriberi results from *food.* Polished rice loses its pericarp, in which there is some vital substance the loss of which causes beriberi; polished or milled rice causes polyneuritis in fowl; and in the disease, the feeding of the pericarp is curative. Natives are more often

affected than Europeans, and males more frequently than females. In 1879 there were 8197 cases in Japan and in 1891, only 1. Poor food, overcrowding and lack of hygiene are important factors.

Symptoms.—The incubation is several months. The *symptoms* are threefold:

1. Neuritic Symptoms.—A more detailed description of these symptoms has been given under Multiple Neuritis. Anatomically, there is the same symmetrical degeneration or inflammation in the distal parts of the peripheral nerves, but also often in the phrenic and pneumogastric nerves and the cardiac, renal and solar plexuses; the nuclei of the sheath multiply; the axis-cylinders degenerate; there is a lumpy degeneration in the medullary sheath and later, a connective-tissue hypertrophy. In 8 cases there were degenerative changes in the cord, anterior horns and posterior ganglia. Clinically, the onset is gradual and the course chronic.

(*a*) *Motor Symptoms.*—The muscles of the calves, the knee extensors and the abductors and flexors of the thigh are weakened, flaccid, swollen and atrophied. The muscles are more frequently tender than the nerve trunks. In severe cases the trunk, arms and even the face are involved. Anatomically, the muscles are degenerated. Phrenic weakness and vagus symptoms, such as rapid heart, vomiting, epigastric oppression or digestive disorder are not infrequent; the lungs are sometimes acutely emphysematous, from involvement of the pulmonary plexus. Laryngeal, glossopharyngeal, hypoglossal or ocular participation is more rare. Cramps and fibrillary contractions are frequent, but ataxia is uncommon, although the name beriberi refers specifically to disturbed locomotion. The gait is described as like that of one walking in wet clothes or wading in water.

(*b*) *Sensory Symptoms.*—Paresthesia is common. Hypesthesia is most marked in the distal parts, though the soles are exempt; it is more frequent than anesthesia, which is rarely complete. Hyperesthesia may occasionally occur over the spine and abdomen. Pains over the joints and intercostal spaces are quite common.

(*c*) *Trophic Symptoms.*—These are uncommon. They include joint relaxation, herpes or skin atrophy.

(*d*) *Reflexes.*—The skin reflexes are seldom abolished. Reduction of the tendon reflexes develops equally with the paralysis.

(*e*) *Degeneration.*—Slight reaction of degeneration is common and may be marked in severe cases. It occurs particularly in the leg.

2. Dropsy.—Dropsy occurs in 97 per cent. of cases and is invariably in fatal cases; it may be marked in the legs and face. It is most severe in the pericardium, quite severe in the pleuræ, and least severe in the peritoneum. It is caused by cardiac and perhaps also by vasomotor involvement.

3. Cardiac Insufficiency.—In acute cases, death may result in a day, and 65 per cent. of acute cases terminate fatally within two weeks. There is palpitation, cardiac or epigastric oppression or pain; tachycardia, dyspnea and cyanosis develop; the right ventricle is hypertrophied and dilated; the first apical tone is weak; there is splitting of the heart

tones, gallop-rhythm, and systolic or even diastolic functional bruits; the urine is decreased in amount; the liver is turgid. Anemia is marked and the leukocytes may be increased. Acute pulmonary edema is usually fatal. The heart muscle is pale, friable, colloid or inflamed.

The *types* of beriberi only accentuate the above-named symptoms: (1) The *rudimentary form* may begin with fever and catarrh of the air passages and alimentary tract. There is a slight edema of the ankles, muscular weakness, especially in the legs, and muscular tenderness, paresthesia and palpitation. (2) The *atrophic form* is marked by atrophic paralysis. If edema does not develop, these types are called "dry forms." (3) The *dropsical form* is characterized by much transudation and pronounced insufficiency of the heart. (4) The *acute cardiac or pernicious form* (*v. s.*).

Diagnosis.—The leading points are the multiple neuritis, most marked in the legs; vagus neuritis and cardiac insufficiency; and dropsy.

Prognosis.—In Japan and the Dutch Indies the death-rate is 3 or 4 per cent.; in Brazil it formerly reached 50 to 75 per cent., and in the Malay archipelago, 40 to 50 per cent. Death results primarily from heart failure, secondarily from paralysis of respiration, less frequently from uremia, pulmonary embolism, aspiration pneumonia, dysentery, tuberculosis or marasmus. Even in recovery, permanent residua are common, such as weakness, or obstinate contractures of the legs, absence of knee-jerks and cardiac instability and hypertrophy. In 43 per cent. of the fatalities there is a sudden death. *Recurrences* are common.

Treatment.—In the early stages, purgation and salicylates are beneficial. In the later stages, electrotherapy and other measures employed in multiple neuritis (*q. v.*) are indicated. Venesection relieves the overladen right heart and dyspnea, and promotes action of the kidneys. Digitalis frequently fails when the vagus is degenerated; in dropsical forms it is helpful, as is caffeine. The ice-bag over the heart, bromides and belladonna may mitigate cardiac unrest and palpitation.

ILLUMINATING-GAS POISONING.

Illuminating gas contains H, hydrocarbons and CO. Steam is forced through the hot coke in the manufacture of the gas and methane, ethane, and benzene are added. Water-gas contains 45 per cent. CO. Alcoholism, the increasing difficulty in procuring morphine and other poisons, ignorance and carelessness cause many deaths yearly.

Symptoms are due to close union of the gas and the hemoglobin, abolition of gaseous interchange, a particular action of the poisons on the nervous system, and absence of oxidation of the waste substances of the body. *Respiration:* The breathing is rapid, loud, stertorous and often of the Cheyne-Stokes's type. W. G. Thompson gives the average number of respirations 30; they were 62 in one non-fatal and 80 in a fatal cases. The mouth is covered with froth which is often bloody. In fatal cases, there is found pulmonary congestion and edema; sometimes bronchopneumonia, atelectasis and emphysema. *Circulation:* The pulse is disproportionately rapid (120–140), considering the fever and respi-

ration, feeble and often irregular. The cherry-red blood is notorious. W. G. Thompson noted leukocytosis, even 50,000; in his fatalities, the white cells exceeded 18,000 but patients with higher counts may recover. The hemoglobin is decreased to 70 per cent. or lower (20 per cent.). G. J. Jones noted reduction of the reds to 1,000,000, often with polychromatophilia; contrary to Thompson's report, he finds no increase of the red cells.

Nervous Symptoms.—Coma ranks first. In most poisonings it is profound; enduring over forty-eight hours, the outlook is generally unfavorable, yet Kinnicutt reports recovery after two weeks of coma. Of Thompson's series, 80 per cent. of comatose cases recovered. The pupils are usually small and immobile but may vary or dilate. Muscular twitchings, convulsions, delirium, opisthotonos, rigidity, increased reflexes and incontinence are common. Postmortem findings include intense cerebral congestion, cerebral apoplexy, serous leptomeningitis, disseminated encephalitis, encephalomyelitis, multiple neuritis and cerebral thrombosis with softening in the internal capsule and lenticular nucleus (Kolisko). Acute mania and paralyses may regress after days or months. Gangrene of the legs may ensue.

Vomiting is occasional and dryness of the mouth the rule. The skin is generally cold, dry, cyanotic but may be clammy; in fatal cases Jones observed serous blebs. The temperature, at first subnormal, in nearly all cases rises after twelve to eighteen hours to 99° or 103°; high fever is usually fatal in one to three days but W. A. Steele records recovery with 110° and a pulse of 215. Glomerulonephritis is common. The urine is high in specific gravity and the nitrogen is increased.

Therapy.—Fresh air and oxygen inhalations seem rational but actually are seldom efficacious. The tongue is held out and occasionally gently pulled. Artificial respiration is indicated. Robin advises the sitting posture but it is difficult to see how it is practicable. The skin should be kept warm. Atropine, gr. $\frac{1}{100}$, and adrenalin, ♏xxx, are indicated but vasodilators, as nitroglycerin, are always injurious. Venesection followed by normal salt solution, given subcutaneously and repeated in urgent cases, is helpful.

SUNSTROKE.

The difference between the two forms, (1) sunstroke, and (2) heat exhaustion, was first recognized by Dowler, of New Orleans.

Sunstroke, also known as siriasis, insolation, *coup de soliel* and thermic fever, is due to exposure to the sun. A very frequent factor is alcoholism. There is thought to be a paralysis of the bulbar heat centres, leading to excessive production of heat and deficient elimination of it. The theory of infection has also been advanced.

Symptoms.—Symptoms may develop very abruptly and cause early or even immediate death from asphyxia and paralysis of the heart; this type was long confused with apoplexy. In most cases there is an interval lasting from a few minutes to an hour, in which there are headache, vertigo, visual disturbances, nausea, and perhaps vomiting before coma intervenes.

During the coma which develops rapidly, and in which the physician usually sees the patient, the following signs are observed: the face is first flushed, later rather cyanotic, and sometimes stained with petechiæ. The preliminary pupillary dilatation passes into myosis and the skin has a peculiar odor. The pulse usually exceeds 100, and is bounding. The rectal temperature ranges from 107° to 112° and respiration is deep, labored and stertorous. Save for the laboring chest, the patient is usually quiet; about 25 per cent. of the author's patients struggled during treatment by the cold bath and about 20 per cent. of the severe cases had epileptiform convulsions. Lewis and Packard (1901) in 92 cases found convulsions and unconsciousness in all severe cases; immobile pupils and absent knee-jerks were common. Involuntary evacuations are usual and the thin feces have a peculiar odor. There is a leukocytosis in about 75 per cent. of the cases. Recovery is frequent; consciousness returns and the fever falls. Despite treatment, the Cheyne-Stokes's breathing may develop, the pulse may grow smaller and more rapid, and death may follow, usually in a little more than a day. The autopsy shows early rigor mortis, fluidity of the blood, great venous hyperemia, particularly in the brain and lungs, and dilatation of the right and contraction of the left ventricle.

After sunstroke the patient becomes extremely susceptible to hot weather. Epilepsy, multiple neuritis and mental symptoms are among its after-effects. The author saw one case with a temperature of 109° in which the fever fell with hydrotherapy but rose to 104°; in a few days the trouble was recognized as typhoid.

Diagnosis.—The diagnosis is easily made. Practically, there is but one possible cause of confusion, pontine hemorrhage, but the latter never develops nearly as high a temperature and death is almost instantaneous; should the patient survive, paralysis, usually of the crossed type, is obvious.

Heat exhaustion may result from exposure to the sun or to high temperature with humidity; the cases seen by the writer came largely from laundries, basement stores or engine-rooms; heat exhaustion occurs also among stokers on steamers. The symptoms are those of a centric vasomotor paralysis. The skin is cool and livid, rather than red, and the rectal temperature is often 3° or 4° subnormal; the pulse is rapid and weak and the breathing is rapid but not labored. In short, the symptoms of collapse are present. Restlessness, anxiety and sometimes delirium are noted, though the sensorium is practically normal in most cases.

Treatment.—In *sunstroke* the chief indication is reduction of the retained heat, by immersing the patient in a *bath gradually cooled* by large pieces of ice. Shock is averted by this means; cerebral congestion is lessened by placing a block of ice under the neck, an ice-bag on the vertex and an ice-bladder over the anterior neck; active friction of the body and limbs with the open hands brings to the surface the blood, which is cooled by the bath and the rubbing with pieces of ice. With this treatment, few more than one-third of the cases should die. The patient should be taken from the bath when his rectal temperature is 102°, for the fall usually continues after cessation of treatment. Laxatives

are generally unnecessary, for the bowels move freely. Heart stimulants should be given *pro re nata*. For convulsions, a little chloroform by inhalation and a hypodermic of morphine are indicated. Asphyxia and distention of the right heart are relieved by phlebotomy.

In *heat prostration* the treatment is diametrically the opposite; a warm bath or hot bricks for subnormal registration, diffusive cardiants, as ammonia, camphor and strychnine, saline solutions under the skin and applications of mustard are indicated; cold and venesection are obviously contra-indicated.

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